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CONTENTS

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INTRODUCTION

Medicine is not merely a science but an art as well. It deals with the different processes of life. The ancient philosophers of siddha school defined the mode and methods of medicine.

A system of medicine without understanding and without a true knowledge of natural laws will remain forever a system of theories, mere opinion and of passive observation and inactivity.

It is nature that causes disease and it is again nature that effects their cures and therefore the physician should know the process of nature.

Siddhars who are considered to be super-human beings have defined age and other laws of nature to which all human beings are subject to.

In Thirumanthiram, which is the most important work of Thirumoolar, he defined medicine as follows.

"Medicine means the prevention of bodily illness
Medicine means the prevention of mental illness
Medicine's purpose is to avert disease
Medicine therefore is the prevention of death".

The original stanza in Tamil reads as follows

"மறுப்பதுடல் நோய் மருந்தென லாகும்
மறுப்பது ளநோய் மருந்தெனச் சாலும்
மறுப்பதினி நோய் வாரா திருக்க
மறுப்பது சாவையு மருந்தென லாமே".

- திருமூலர்

They seem to have realised most rationally that prevention is better than cure and that prevention should be the main purpose of medicine.

Life is man's most valuable possession and the next in order of value is health. Health is the chief basis for the development of ethical, economic, artistic and spiritual sides of man. Without health, life is deprived not only of much if not all, of its usefulness but also of its joys and pleasures.

Noi or disease denotes weakness and in which both mind and body are involved (or) the disease will cause suffering to the mind and body.

It is pointed by "Tholkaapiyar" that the disease means suffering and depression.

"பையுளும் சிறுமையும் நோயின் பொருள்".

(தொல் உரி 341)

When the disease is due to the persistent action of the adverse factors, it becomes chronic and complete cure at this stage is very difficult. The siddhars were aware of the fact of chronicity.

"வறிதால் இருளையென் யாக்கை யினியவர்

வரினும் நோய் மருந்தல்லர் வாராது".

The effect of treatment depends upon the prevention only. The disease has no effect if we adhere to the principles of prevention in our daily life. The Tamils of Sangam period were prompt in preventing diseases both internal and external to lead a healthy life.

SIDDHA PHYSIOLOGY

Siddhars believed that five elements are the basis of the universe and every human being. Our ancient literature Tholkaapiyam also accepts the concepts of siddhars. The universe is a composition of five elements viz earth, air, water, ether and fire which are known as “Panchabootham” in siddha system. So the human body is a composition of this pancha bootham.

“அண்டத்திலுள்ளதே பிண்டம்
பிண்டத்திலுள்ளதே அண்டம்
அண்டமும் பிண்டமும் ஒன்றே
அறிந்துதான் பார்க்கும் போதே”.

- சட்டமுனி ஞானம்

According to siddha physiology man is considered as the microcosm. Universe is considered as the macrocosm. It shows that the human body is the replica of the universe.

Vethas reveal that one of the five elements combined with the other four elements in different proportions to form the human body. The basic reason for the soul resting is uyirthathu or jeevathathu.

This uyirthathu divided into three thodas known as vatham, pitham, kabam and acquires three characters (mukkunam – sathuva, rajo, thamo) thereby it protects and develops the soul and the body.

Each and every atom consists of 96 thathuvas. These 96 thathuvas are invisible to our naked eye until it is present in a single atom. Since it

mingles or joins to form a multi cellular body and it gets larger size according to the shape and merges to act respectively.

Due to the combination of 96 thathuvas, soul originates, acquires shape and multiplies to grow larger and finally gets a body to live and then performs its duties, multiplies its generations, gets its old ages and dies. Finally it reaches its initial stage where it was in primitive.

This 96 thathuvas are limited to all human beings in normal condition. This not only consists of the physical components of the human body but also the mental intellectual components like passions, qualities, knowledge, the functions of the sense organs and motor organs and their co-ordination.

The physiology of siddha system involves 96 basic factors, seven constituent elements, 14 reflexes, aru suvaigal, four udal thee and three udal vanmaigal.

"உறுதியாம் பூதாதி யோரைந்தாம்....."

- வேதாந்த தத்துவக் கட்டளை

Panchabootham 5 - Five elements

Earth	-	all organic living bodies and organic substances are created
Water	-	It combines all the things
Air	-	It spreads all over the space
Fire	-	It gives colour and brightness to the things
Space	-	It gives space to all other boothams

Gnanenthirium or Pori 5 - Five sense organs

1. Ear
2. Skin
3. Eye
4. Tongue
5. Nose

Pulan 5 – Functions of five sense organs

1. Hearing
2. Touch sense
3. Vision
4. Taste
5. Smell

Kanmaenthiriam 5 – Five motor organs

1. Mouth - stands as space
2. Hand - stands as air
3. Leg - stands as fire
4. Anus - stands as water
5. Sex organs- stands as earth

Kanmavidayangal 5 – Functions of five motor organs

1. Speech
2. Flexion and extension of upper limbs and lower limbs
3. Walking
4. Defecation
5. Ejaculation of semen and propulsion of ova

Anthakaranas 4 – Four intellectual faculties

1. Manam - Mind or the reasoning faculty
2. Puththi - Knowledge
3. Siddham - Determination or firm conviction
4. Agangaram - Achievement

Arivu 1 - Intellect or wisdom

Naadies 10:

There are ten channels

1. Idakalai
2. Pinkalai
3. Suzhumunai
4. Siguvai
5. Purudan.
6. Kanthari
7. Aththi
8. Alampudai
9. Sankini – Present in external genitalia
10. Gugu – Present in anus

Vayu 10 – Ten vital airforces

1. Abanan
2. Pranan
3. Udhanan
4. Samanan

5. Viyanan
6. Koorman
7. Naagan
8. Kirukaran
9. Devathathan
10. Dhananjeyan

Aasayam 5 - Five visceral cavities

Amarvasayam	-	Stomach
Pahirvasayam	-	Liver, small intestine
Malavasayam	-	Rectum, large intestine
Salavasayam	-	Urinary bladder
Sukkilavasayam	-	Seminal vesicle and ovary

Kosam 5 - Five vestures of the soul

1. Annamaya kosam
2. Piranamaya kosam
3. Manomaya kosam
4. Vinganamaya kosam
5. Anandhamaya kosam

Aatharam 6:

1. Moolatharam
2. Swathitanam
3. Manipooragam
4. Anagatham

5. Vishuthi
6. Aakkinai

Malam 3 - Three mental binders

1. Aanavam
2. Maayai
3. Kanmam

Mandalam 3 – Regions

1.Gnayiru (sun) - Solar plexus

2. Thingal (moon) - Lunar plexus

3.Thee (fire)

Thodam 3 - Three humours

Three humours are the fundamental principles and essential factors in the composition and constitution of the human body. The three humours vatham, pitham and kabam represent wind, bile and phlegm respectively.

Relation between boothas and mukkutram

Vatham - Air

Pitham - Fire

Kabam - Water

Formation of the three humours

“வந்த கலை மூன்றில் வாயும பானனுடன்

தந்த பிராணன் சமானனும் - சந்தமுறக்

கூட்டுறவு ரேசித்தால் கூறும் வாதம் பித்தம்

நாட்டுங் கபமேயாம் நாடு”.

Vatham	-	Idakalai	+	Abanan
Pitham	-	Pinkalai	+	Piranan
Kabam	-	Suzhumunai	+	Samanan

"வாதமாய்ப் படைத்துப் பித்த வன்னியாய்க் காத்துச் சேட்ப

சீதமாய்த் துடைத்து....."

- தேரையர் மருத்துப் பாரதம்.

Vatham	-	Creation
PItham	-	Protection
Kabam	-	Destruction

"மெய்யளவு வாதமொன்று

மேல்பித்த மோரரையாம்

ஐயங்காலென்றே அறி".

- கண்ணுசாமியம்

Vatham	-	1 maathirai
Pitham	-	½ maathirai
Kabam	-	¼ maathirai

Qualities:-

Vatham	**Pitham**	**Kabam**
Dry	Hot	Cold
Cold	Acid	Heavy
Subtle	Mobile	Sweet
Rough	Liquid	Soft
Unstable	Acute	Unctuous
Light	Pungent	Viscid

Functions:-

Vatham	Pitham	Kabam
Respiration	Acidity	Strengthens bones and joints
Giving briskness	Burning sensation	Gives lustre to the body
Functions of the body	Yellowish discolouration	Makes strength
Regulation of 14 Vegangal	of eye, skin and urine etc	Fearlessness
Functions of 7 udal kattugal	Digestion	Patience
Protects and strengthens five sensory organs	Profuse sweating Dizziness	Immunity

Types:-

Vatham	Pitham	Kabam
Pranan	Anarpitham	Avalambagam
Abanan	Ranjagam	Kilaedhagam
Viyanan	Saadhagam	Bothagam
Udhanan	Aalosagam	Tharpagam
Samanan	Pirasagam	Sandhigam
Naagan		
Koorman		
Kirukaran		
Devathathan		
Dhananjayan		

Edanai 3 - Three physical bindings

1) Porul patru - Material bindings

2) Puthalvar patru - Off springs bindings

3) Ulaga patru - World bindings

Vinai 2 – Two deeds

1) Nalvinai - Good deeds

2) Thee vinai - Bad deeds

Gunam 3 – Three cosmic qualities

Sathuva gunam

Rasathuva gunam

Thamasa gunam

Raagam 8 – Eight passions

1. Kaamam - Desire
2. Krotham - Hatred
3. Lopam - Stinginess
4. Moham - Lust
5. Matham - Pride
6. Marcharyam - Internal conflict
7. Idumbai - Mockery
8. Agankaaram - Ego or self love.

Avaththai 5 – Five states of consciousness

1. Ninaivu - Wakefulness
2. Kanavu - Dream
3. Urakkam - Sleep
4. Parurakkam - Stuper
5. Uyirppadakkam - Stage of samaathy

Udal thathukkal 7 - Seven constituent elements

Seven udal thathukkal are responsible for the entire structure of the human body.

“இரசம் உதிரம் இறைச்சி தோல் மேதை
மருவிய வத்தி வாழும் பொரு மச்சை
பரவிய சுக்கிலம் பாழாம் உபாதி
உருபம லானுடல் ஒன்றெனலாமே”.

- திருமந்திரம் 2080

1. Saaram - Chyle
2. Chenneer - Blood
3. Oon - Muscle
4. Kozhuppu - Fat
5. Enbu - Bone
6. Moolai - Bone marrow
7. Sukkilam / suronitham - Sperm / ovum

Udal thathus maintain the function of different organs, systems and vital parts of the body. They play a very important role in the development and nourishment of the body. Each thathu receives its nourishment from the previous thathu. When one thathu is defective it affects the successive thathu.

Vegangal 14 – Fourteen Urges / Reflexes

1. Vatham
2. Thummal
3. Siruneer
4. Malam
5. Kottaavi
6. Pasi
7. Neer Vetkai
8. Kasam
9. Elaippu
10. Nithirai.
11. Vaanthi
12. Kanneer
13. Sukkilam / Suronitham
14. Suvaasam.

Udal Vanmai 3 – Strength and vitality constitute udal vanmai.

1) Eyarkai vanmai (Innate immunity)	-	Inherited vitality.
2) Kaala vanmai (Seasonal immunity)	-	Vitality that is generally found in different age periods as well as different seasons.
3) Seyarkai vanmai	-	Improvement of vitality obtained by good habits and physical exercises

Udal akkini 4 – Four body fires

- Samaakkini
- Mandhaakkini
- Deekshanaakkini
- Vishamaakkini

Suvaigal 6 – Six tastes

Suvai is a peculiar sensation caused by the contact of soluble substances with the tongue. The sense is effected by the tongue.

Combination of two boothas constitute a suvai (taste)

"மண்ணுடனே புனல்தீக்கால் முறையாகச் சேர்ந்திட்டால் வருமே இனிப்பு
திண்ணமிலம் துவர்ப்பிரசம் சதாகதியோ டார்தீயின் திடமா முறைப்பும்
எண்ணரிய கசப்புமுண்டாந் தண்ணீரில் கனலிணைப்பாலெழுமா - முவர்ப்பு
உண்ணரிய அறுசுவையின் பிறப்பிதெனும் குருசித்தருரைத்த மறையே".

- மருத்துவத் தனிப்பாடல்கள்

Arusuvai

Inippu	-	Mann + Neer	-	Kabam
Pulippu	-	Mann +Thee	-	Pitham
Uppu	-	Neer +Thee	-	Pitham
Kaippu	-	Vali + Vinn	-	Vatham
Kaarppu	-	Vali + Thee	-	Pitham
Thuvarppu	-	Mann + Vali	-	Vatham

SIDDHA PATHOLOGY

Pathology is the scientific study of structure and function of the body in a disease. It deals with causes, effects, mechanism and nature of diseases.

Siddha pathology deals with the diseased condition of the human, which is due to food alterations, seasonal and environmental variations, helding the 14 reflexes and by the behaviours. The disease is reflected through the pulse formed by the three humours.

The whole siddha system of medicine rests on the maintenance or restoration of the equilibrium between the three thathus, which coincides with the following kural.

"மிகினும் குறையினும் நோய் செய்யும் நூலோர்

வளி முதலா எண்ணிய மூன்று."

- திருக்குறள்

Siddha system approaches and analyses the disease on the basis of the 'Thridosha theory' . The doshas or humours are vatham, pitham and kabam. Disease is due to the disturbance in the equilibrium between the three humours. When these are in perfect balance and harmony a person is said to be healthy. Imbalance and derangement in thridoshas causes disease. So the diseases are studied on the basis of thridosha theory.

"மதித்திடற்கருமை வாய்ந்த

மாண்பரிகாரரமெல்லாந்

துதித்திட வுணர்ந்தானேனுந்

துகளறப் பிணியின்றன்மை,

பதித்திடவுணரானாகிற்

பயனுறானாகாலானே

விதித்திடு பிணித்திறத்தை

விளம்புது முதற்கண்மன்னோ”

- சிகிச்சாரத்ன தீபம்

“முப்பிணி மருவி முனிவு கொள் குறிப்பைத் தப்பா

தறியும் தன்மையும் வாத பித்த வையம் பிரிவையு

மவைதாம் ஏறியிறங்கி இணைந்து கலந்து மாறி

மாறி வரும் செயற்கையாற் பிணி நேர்மையறிந்து

நீட்டு மருந்தே சீரியதாமெனச் செப்புவர் சித்தரே”.

Food variations

Diet plays a vital role in preserving the human body. The food is formed on the basis of 6 tastes.

“புளி துவர் விஞ்சுங்கறி யாற்பூரிக் கும்வாதம்

ஒளி யுவர்கைப் பேறில் பித்துச் சீறும் - கிளிமொழியே

கார்ப்பினிப்பு விஞ்சிற் கபம்விஞ்சு ஞ்சட்டிரதச்

சேரப் புணர் நோயணுகாதே”.

- கண்ணுசாமியம்

Sour and astringent cause an increase in vatham. Salt and bitter cause an increase in pitham. While pungent and sweet cause an increase in kabam.

Environmental changes

The environmental factors (Thinai) may also pave the way for the development of diseases. Thinai has been classified into 5 types.

A. Kurinji : Kabam pertains here. Anemia, fever and abdominal mass may develop.
B. Mullai : The dwellers of this land suffer from vatha and pithakaba diseases.
C. Marutham : The inhabitants of this region have controlled vatham, pitham and kabam . This is the prompt place for a healthy inhabitation.
D. Neithal: The people of this land suffer from vatha diseases, further it leads to increased body mass, enlargement of liver and flatulence.
E. Palai : The inhabitants of this land suffer from vatha , pitha and kaba diseases.

Seasonal variations

Due to seasonal variations, changes in three humours (thridosha) occur and this lead to a disease.

1) Kar kalam (Avani and Puratasi) - Disturbances in three humours

	is most prominent. Pitham is increased from its normal state. Vatham is also increased from its normal level and spreads continuously to other areas of the body.
2) Koothir kalam (Iypasi & Karthigai)-	Altered pitham spreads to other areas of the body.
3) Munpani kalam (Markazhi & Thai)-	Three thodas are in equilibrium.
4) Pinpani kalam (Maasi and Pankuni)-	Kabam increases from its normal state.
5) Elavenil kalam (Chithirai and Vaigasi)-	Altered kabam spreads to other parts of the body.
6) Muthuvenil kalam (Aani & Aadi) -	Kabam comes to equilibrium and vatham gets aggrevated.

Variations in 3 humours

The disease is mainly caused because of the inequilibrium in one or more among the 3 humours that exist in human as ‘Uyir Thathukkal’.

Three humours	Characteristic features of increasing	Characteristic features of decreasing
Vatham	Weakness and occasionally blackening of the body. Desire to take hot diet, shivering, abdominal distension , constipation, diminished immunity, insomnia, giddiness, laziness, blabbering and generalised weakness.	Stiffness, diminished voice, impaired intellectual function, disturbance in general activities, semi-consciousness, fatigue, excessive salivation, paleness and cooling of the body, breathlessness, cough and excessive sleep.
Pitham	Yellowish discolouration of eyes, faeces, urine and skin. Polyphagia, polydypsia, burning sensation all over the body and decreased sleep.	Loss of appetite, cooling of the body, impaired pigmentation of the skin, symptoms related with decreased kabam.
Kabam	Abdominal distension, salivation, fatigue, paleness and cooling of the body, heaviness of the body, breathlessness, cough and excessive sleep.	Giddiness, sublaxation of joints, prominence of bones. Kabam present in the lungs gets decreased. Excessive sweating in the hair follicles and palpitation.

Udal thathukkal

When the three humours of the human body are affected by various factors they immediately change the nature of the 7 physical constituents, i.e. udal thathukkal.

Udal thathukkal (Physical constituents)	Features of increasing	Features of decreasing
Saaram (Chyle)	Features related with decrease in kabam, loss of appetite	Dryness of skin, loss of weight, tiredness, the functions of sense organs are diminished
Senneer (Blood)	Boils and tumours in different parts of the body, splenomegaly, soolai (pain), hypertension, haematuria, redness of the eyes, leprosy, jaundice	Desire for cold things, dryness, discolouration and paleness of the skin
Oon	Tumours or extra growth around the neck, face, abdomen, thigh and genitalia	Lethargy of 5 sensory organs, pain in the joints, loss of subcutaneous fat
Kozhuppu	Identical to increasing features of oon, tiredness, dyspnea on exertion	Splenomegaly, loin pain, emaciation

Enbu	Excessive ossification and dentition	Weak bone pain in the joints, splitting of hair and nails
Moolai	Sense of heaviness of the body and eyes, swelling of smaller joints of hand and feet, oliguria, non – healing ulcers	Osteoporosis , blur vision
Sukkilam / Suronitham	Sexual activity increases, urinary calculi	Pain in the genitalia and accompanied inability to reproduce

URGES (14 VEGANGAL)

There are 14 natural reflexes involved in the physiology of normal human beings and if willfully suppressed, the following are resulted.

1. Vatham (Flatus)

This urge should not be suppressed. If it is suppressed it leads to chest pain, epigastric pain. Abdominal pain, body ache, constipation, dysuria and indigestion predominates.

2. Thummal (Sneezing)

If arrested it leads to headache, facial pain, low back pain and neuritic pain in the sense organs.

3. Siruneer (Urine)

If arrested it leads to urinary retention, urethral ulcer, joint pain, pain in the penis, gas formation in abdomen.

4. Malam (Faeces)

If arrested it leads to pain in the knee joints, headache, general weakness, flatulence and other diseases may also originate.

5. Kottavi (Yawning)

If arrested it leads to indigestion, leucorrhoea, abdominal disorders and urinary disorders.

6. Pasi (Hunger)

If arrested it leads to the tiredness of all organs, emaciation, syncope, apathetic face and joint pain.

7. Neer vetkai (Thirst)

If arrested it leads to the affection of all organs and pain may supervene.

8. Kaasam (Cough)

If it is suppressed severe cough, bad breath and heart diseases will be resulted.

9. Ilaippu (Exhaustiveness)

If suppressed it will lead to fainting, urinary disorders and rigor.

10. Nithirai (Sleep)

All organs will get rest only during sleep. So it should not be avoided. If disturbed it will lead to headache, pain in the eyes, deafness and slurred speech.

11. Vaanthi (Vomiting)

If arrested it leads to itching and symptoms of increased pitham.

12. Kanneer (Tears)

If it is suppressed it will lead to sinusitis, headache, eye diseases and chest pain.

13. Sukkilam (Semen)

If it is suppressed there will be joint pain, difficulty in urination, fever and chest pain.

14. Swaasam (Breathing)

If it is suppressed there will be cough, abdominal discomfort and anorexia.

Udal Vanmai:

The disease affecting an individual is also based on the udal vanmai. The udal vanmai is classified into 3 types.

1. Eyarkai Vanmai

This is based on sathuva, rajo and thamo gunas and it is the strength which is present naturally.

2. Seyarkai Vanmai

The mukkuna based body is maintained by food habits. Seyarkai vanmai means improving the strength by diet and medicine.

3) Kaala Vanmai:

It is based on kaalangal. The strength that is gained by seasonal variations as well as the age of a person.

Investigations in siddha system

The methods adopted in siddha system of medicine are poriyaalarithal, pulanaalarithal and vinaathal. Poriyaalarithal means diagnosing through the five organs of perception namely nose, tongue, eyes, ears and skin. Pulanaalarithal means diagnosing through the five objects of senses namely smell, taste, vision, audio and sensation of skin. Vinaathal is a method of interrogating the problem of the patient from his own words or from attendars.

Envagai Thervugal

"நாடி ஸ்பரிசம் நா நிறம் மொழி விழி

மலம் மூத்திரமிவை மருத்துவ ராயுதம்".

1. Naadi (Pulse)
2. Sparisam (Sense of touch)
3. Naa (Tongue)
4. Niram (Colour)
5. Mozhi (Speech)
6. Vizhi (Eyes)

7. Malam (Stool)
8. Moothiram (Urine)

"மெய்க்குறி நிறந்தொனி விழி நாவிருமலம் கைக்குறி".

- தேரையர்

Naadi (Pulse)

This is a unique diagnostic method in siddha system of medicine. It is responsible for existence of life. It is felt one inch below the wrist on the radial side by palpating with the top of the index finger, middle finger and ring finger which denotes vatham, pitham and kabam.

Suitable places to feel pulses

"தாது முறைகேள் தனிக் குதிச் சந்தோடு
ஓதுறு காமிய முந்தி நெடு மார்பு
காது நெடுமூக்குக் கண்டம் கரம் புருவம்
போதுறு முச்சு புகழ் பத்தும் பார்த்திடே".

In siddha system of medicine changes of urine is studied under two peculiar headings. They are "Neerkuri and Neikuri".

"வந்த நீர்க்கரி எடை மணம் எஞ்சலென்
றைந்தியலுளவவை யறைகுது முறையே."

- தேரர் நீர்க்குறி - நெய்க்குறி

Neikuri

1) அரவென நீண்டின.்.தே வாதம்

2) ஆழி போற்பரவின் அ.்.தே பித்தம்

3) முத்தொத்து நிற்கின் மொழிவதென்கபமே.

- தேரர் நீர்க்குறி - நெய்க்குறி

This procedure is an important one in siddha system of medicine to find out the diagnosis as well as in prognosis aspect of the disease.

So diseases in man do not originate itself. It is developed from the alteration of three doshas or humours and are classified in to 4448 as per siddha physician Yugi's theory.

AIM AND OBJECTIVES

The Tamils of Sangam period were used to follow healthy food habits and their life style saved them from many diseases and disease causing factors. But now a days because of unhealthy, unwanted food habits and modified life style of our people, many diseases gained entry among them.

One of them is cardiovascular disease, which is the leading cause of death in all continents. Despite of all the advances in the medical field, which has reached its sky as a limit, the incidence of heart diseases is rising in many developed countries.

In evaluating patients with heart failure, it is important to identify not only the underlying cause of the heart disease but also the precipitating cause of heart failure. Identification of such precipitating causes is of critical importance because their prompt alleviation may be life saving.

The aim of this dissertation topic is

- To collect the evidences found in siddha literature regarding Oorthuva Vatham.
- To review the altered thridosha (or) mukkuttram and pathology in siddha aspect.
- To depict the unique diagnostic procedures (ie) Envagai thervugal, mentioned in siddha literatures for Oorthuva Vatham.
- To use modern parameters in the investigation of Oorthuva Vatham.

- To know the pathogenesis (origin and development) of Oorthuva Vatham.
- To find out the aggravating factors which cause the condition to worsen and to find out the preventive methods.

READING LINES BETWEEN YUGI'S POEM

In Yugi vaithiya sinthamani under “Vatha Roga Nithanam”, “Oorthuva Vatham” was broadly dealt in the 298th stanza. It is as follows.

ஊர்த்துவ வாதம்

“நூலான சுவாசந்தான் மேலே நோக்கி
நுணுக்கமா மிடையுடனே வயிறு விம்மி
வாலான வாய் தனிலே நுரையுமுண்டாய்
வாய் கழுத்துமார்புமே மிகவு நொந்து
ஆலான வங்கமெல்லா மழன்று காணும்
அடித்தொடையு மடிக்கையு மிகவே வற்றும்
பாலான மேனியுமே பசுமை காணும்
பருவூர்த்வ வாதமென்றே பகர லாமே”.

- யூகி வைத்திய சிந்தாமணி

ஊர்த்துவ வாதம்

The author Yugi mentioned the symptoms of the above disease under the title “Oorthuva Vatham”.

Oorthuvam means rising or tending upwards. It denotes the erect position of the patient due to laboured breathing. The sensation of breathlessness usually is relieved by sitting upright since this position reduces venous return and pulmonary capillary pressure. In far advanced heart failure orthopnea (dyspnea in the recumbent position) may become so severe that patients cannot lie down at all and must spend the entire night in sitting position.

"நூலான சுவாசந்தான் மேலே நோக்கி"

Dyspnea in the recumbent position which is characterised by extreme shortness of breath and rapid, shallow breathing.

"நுணுக்கமா மிடையுடனே வயிறு விம்மி"

Distension of the abdomen.

"வாலான வாய்தனிலே நுரையு முண்டாய்"

Frothy bronchial secretions pour out of the mouth.

"வாய் கழுத்துமார்புமே மிகவு நொந்து"

Pain in the mouth, neck and chest due to breathlessness.

"ஆலான வங்கமெல்லா மழன்று காணும்"

It denotes fatigue, poor effort tolerance, weakness and low grade fever.

"அடித்தொடையு மடிக்கையு மிகவே வற்றும்"

It denotes serious weight loss, cardiac cachexia and skeletal muscle atrophy.

"பாலான மேனியுமே பசுமை காணும்"

Generalised anasarca.

The summary of Oorthuva Vatham is as follows.

In congestive cardiac failure, pulmonary congestion reduces lung compliance and can obstruct the small airways. This causes dyspnea which means laboured breathing. Due to inability of the heart to empty properly, congestion of tissues occurs (Hepatic congestion and ascites). Pulmonary congestion results in escaping of bronchial secretions through

the mouth. Pain in the mouth, neck and chest occurs due to dyspnea. Low cardiac output causes fatigue, listlessness, poor effort tolerance. Chronic heart failure is associated with cardiac cachexia and skeletal muscle atrophy. Generalised anasarca results due to congestion of tissues and protein loss.

PATHOLOGICAL VIEW OF READING LINES IN MODERN ASPECT

"நூலான சுவாசந்தான் மேலே நோக்கி"

The clinical manifestations of left-sided heart failure result from accumulation of fluid upstream in the lungs and from decreased left ventricular output. Among them one of the major clinical manifestations is pulmonary congestion and oedema which causes dyspnea and orthopnea.

A frequent cause of death in heart failure is acute pulmonary oedema occurring in patients who have had chronic heart failure for a long time.

Pulmonary oedema can result from either the elevation of pulmonary hydrostatic pressure or the increased capillary permeability.

The hydrostatic pressure in the pulmonary capillaries is much lower (average 10mm Hg). Normally the plasma oncotic pressure (25 mmHg) is adequate to prevent the escape of fluid into the interstitial space and hence lungs are normally free of oedema.

Elevation in pulmonary hydrostatic pressure (Haemodynamic oedema).

In heart failure, there is increase in the pressure in pulmonary veins which is transmitted to pulmonary capillaries. This results in imbalance between pulmonary hydrostatic pressure and the plasma oncotic pressure so that excess fluid moved out of pulmonary capillaries into the interstitium of the lungs. Simultaneously, the endothelium of the pulmonary capillaries

develops fenestrations, permitting passage of plasma proteins and fluid into the interstitium.

The interstitial fluid so collected is cleared by the lymphatics present around the bronchioles, small muscular arteries and veins. As the capacity of the lymphatics to drain the fluid is exceeded (about tenfold increase in fluid), the excess fluid starts accumulating in the interstitium (interstitial oedema) i.e, in the loose tissues around bronchioles, arteries and in the lobular septa.

Next follows the thickening of the alveolar walls because of the interstitial oedema. Upto this stage, no significant impairment of gaseous exchange occurs. However prolonged elevation of hydrostatic pressure and due to high pressure of interstitial oedema, the alveolar lining cells break and the alveolar air spaces are flooded with fluid (alveolar oedema) driving the air out of alveolus, thus seriously hampering the lung function.

This increases the work of the respiratory muscles required to inflate the lungs. The activation of receptors in the lungs results in the rapid, shallow breathing which is the characteristic feature of cardiac dyspnea. The oxygen cost of breathing is increased by the excessive work of the respiratory muscles. This is coupled with the diminished delivery of oxygen to these muscles which occurs as a consequence of the reduced cardiac output and this may contribute to fatigue of the respiratory muscles and the sensation of shortness of breath.

"நுணுக்கமா மிடையுடனே வயிறு விம்மி"

Fullness of abdomen, anorexia, nausea associated with abdominal pain are frequent complaints and may be related to the congested liver and portal venous system. An enlarged, tender, pulsating liver and ascites present.

"வாலான வாய் தனிலே நுரையுமுண்டாய்"

This is due to pulmonary congestion with pulmonary oedema. The patient brings out lot of sputum which is characteristically frothy as the bronchial secretion is mixed up with air.

The fluid which enters the lungs is beaten up into a frothy by the pulmonary ventilation.

In grave diseases both lungs are entirely filled with oedema fluid and frothy fluid may pour out of the mouth.

"வாய் கழுத்துமார்புமே மிகவு நொந்து"

Pulmonary oedema reduces the compliance of the lungs and thereby increases the work of the respiratory muscles required to inflate the lungs. The cost of breathing is increased by the excessive work of the respiratory muscles. This is coupled with the diminished delivery of oxygen to these muscles, which occurs as a consequence of the reduced cardiac output and which may contribute to fatigue of the respiratory muscles.

"ஆலான வங்கமெல்லா மழன்று காணும்"

Due to inadequate blood supply to the muscle fatigue, poor effort tolerance and weakness occur.

Low grade fever is because of reduction of cutaneous flow and lung infection due to pulmonary oedema.

"அடித்தொடையு மடிக்கையு மிகவே வற்றும்"

With severe chronic heart failure there may be serious weight loss and skeletal muscle atrophy.

The serious weight loss and cachexia are because of

1. Elevation of circulating concentrations of cachectin or tumour necrosis factors. (TNF-∝) and interleukin–1 derived from macrophages.
2. Elevation of the metabolic rate, which results in part from the extra work performed by the respiratory muscles, the increased oxygen needs of the hypertrophied heart and / or the discomfort associated with severe heart failure.
3. Anorexia, nausea and vomiting due to congestive hepatomegaly and abdominal fullness.
4. Impairment of intestinal absorption due to congestion of the intestinal veins.
5. Protein losing enteropathy.

Skeletal muscle atrophy is due to prolonged diminished functional activity i.e, wasting of muscles of limb immobilised in cast (disuse atrophy). Reduction of the number and size of parenchymal cells of an organ or its parts which was once normal is called atrophy. Irrespective of the underlying cause for atrophy, the pathological changes are similar.

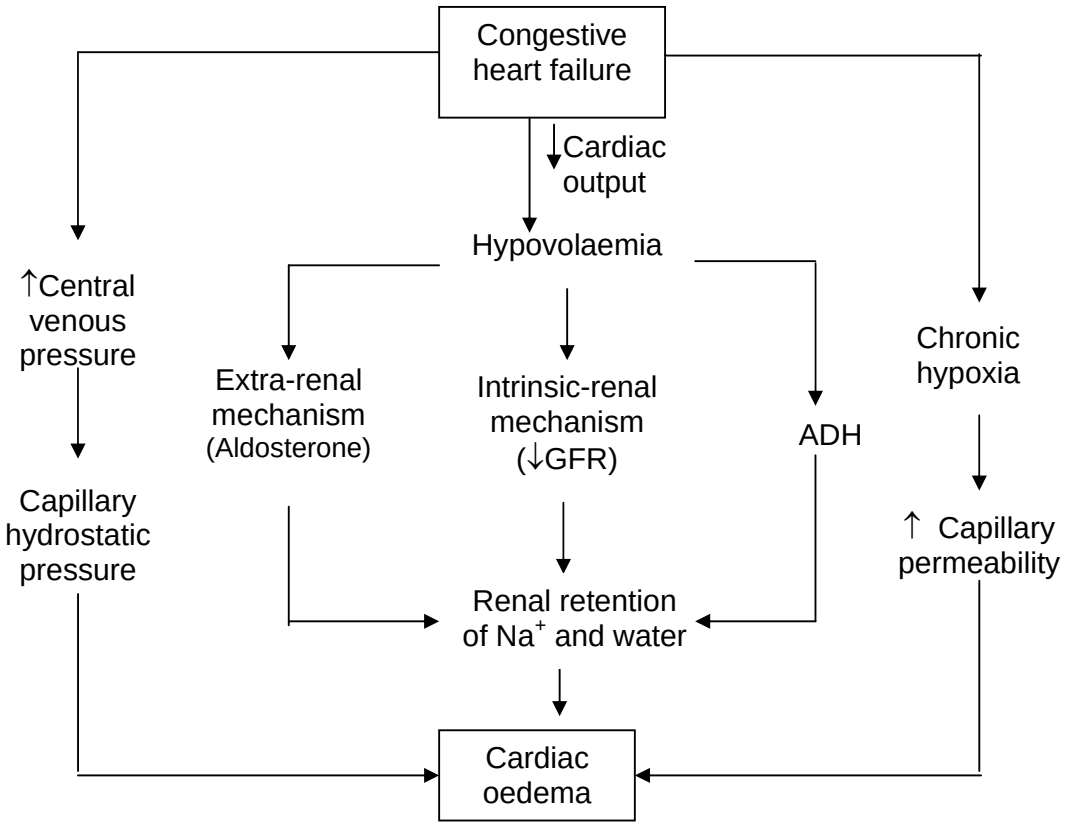
The organ is small, often shrunken. The cells become smaller in size but are not dead cells.

Shrinkage of cell size is due to reduction in cell organells, chiefly mitochondria, myofilaments and endoplasmic reticulum. There is often increase in the number of autophagic vacuoles containing cell debris. These autophagic vacuoles may persist to form residual bodies in cell cytoplasm.

"பாலான மேனியுமே பசுமை காணும்"

Generalised oedema develops in congestive cardiac failure. Pathogenesis of cardiac oedema is explained on the basis of the following hypothesis.

MECHANISMS INVOLVED IN THE PATHOGENESIS OF CARDIAC OEDEMA



1. Reduced cardiac output causes hypovolaemia which stimulates intrinsic–renal and extra–renal hormonal (renin–angiotensin– aldosterone) mechanisms as well as ADH secretion resulting in sodium and water retention and consequent oedema.

2. Due to heart failure, there is elevated central venous pressure which is transmitted backward to the venous end of the capillaries, raising the capillary hydrostatic pressure and consequent transudation, this is known as back pressure hypothesis.

3. Chronic hypoxia may injure the capillary wall causing increased capillary permeability and results in oedema, this is called forward pressure hypothesis. However this theory lacks support since the oedema by this mechanism is exudate whereas the cardiac oedema is typically transudate.

Cardiac oedema is influenced by gravity and is thus characteristically dependent oedema i.e, in an ambulatory patient it is on the lower extremities, while in a bed–ridden patient oedema appears on the sacral and genital areas. The accumulation of fluid may also occur in serous cavities.

PATHOLOGICAL VIEW OF DISSERTATION TOPIC IN SIDDHA ASPECT

“பிணியினுற் பத்தியைப் பேசுவன் பிணிமுதல்
வாத பித் தங்கப மன் மந்திரி தந்திரி
வீதமா யுடலரண் மெய்ம்புர வரசு செய்
முறை செயுமாதலான்.....”

- தேரையர் காப்பியம்

According to siddha aspect, vatham is said to be the initiator of all activities of our body. So the author mentioned vatham as ‘ Arasan’ in the above lines.

“வாதபித்தமைய மூன்றும்
வன்பலத்துடனே தத்தம்
பேதமொன்றில்லா வண்ணம்
பேசியதானந் தன்னில்
நீதியாய் நிலைத்து நிற்கில்
நெடும்பிணி சிக்கவில்லை
தாதுவுமொன்றோ டொன்று
தாவிடிற் பிணிகள்தானே”.

- கண்ணுசாமியம்

Imbalance and derangement in thridosha cause diseases. During the acute stage of diseases any of the three thathus (3 humours) or seven udal kattugal (7 constituent elements) may be affected. If the condition is not treated promptly, it will lead to the affection of other thathus and the condition will become chronic.

By knowing the distribution of vatham, one can understand that vatham is capable of affecting many of the systems of the body. If we don't treat the derangement of vatham, the condition will lead to 'Thontha Vinaigal' (secondary affections) and causes suffering to the body.

ALTERED THRIDOSHA IN OORTHUVA VATHAM

I. Vatham: Ten forms of vatham are described in our siddha literatures.

1. Pranan: 'Pranan' is considered as heart centre. It controls heart, circulation, inspiration and expiration.

In 'Oorthuva Vatham' the circulation of blood to other parts of the body is affected i.e., pranan is affected.

2. Abanan

The functions of abanan is micturation, defecation etc.

In 'Oorthuva Vatham', sodium and water retention, oliguria and steatorrhoea occur because of the derangement of abanan.

3. Viyanan

Viyanan is distributed throughout the body. It is said to be the part of the sky. Its place in our body is heart. The functions of viyanan is walking, movements of the body etc.

In 'Oorthuva Vatham", viyanan is affected and exhibits the symptoms like dyspnea on exertion.

4. Samanan

The equilibrium between the ten forms of vatham is maintained by samanan. It prevents the increase of other vayus and facilitates digestion and absorption.

In ‘Oorthuva Vatham’, samanan is affected and results in indigestion and poor tissue perfusion.

5. Kirukaran

Kirukaran produces appetite and its affection causes anorexia in ‘Oorthuva Vatham’.

6. Thananjayan

In ‘Oorthuva Vatham’, thananjayan is affected and it causes generalised anasarca.

II.Pitham

"போமென்ற பித்தத்துக்குக்கிருப்பிடமே கேளாய்,

பேரான கண்டத்தின் கீழதாகும்.

- யூகி

From the above lines, we understand that heart is one of the dwelling places of pitham.

There are five forms of pitha.

1. **Anarpitham**

Anarpitham is responsible for digestion. Due to the derangement of anarpitham, indigestion results in ‘Oorthuva Vatham”.

2. Sathaga Pitham

Ability to do our work is due to the role of sathaga pitham.

Sathaga pitham is affected in ‘Oorthuva Vatham and exhibits in the form of poor effort tolerance.

III. Kabam

Thorax is one of the places in which kabam dwells.

According to siddha physiology, there are five forms of kabam.

1. Avalambagam

Its living place is said to be the lungs and it is responsible for the elastic property of pulmonary tissues (surfactant). It maintains the normal physiological function of the heart.

In ‘Oorthuva Vatham’, avalambagam is affected and results in impairment of the function of the heart.

IV. Udal Kattugal (7 Constituent Elements)

1 Increase of Saaram leads to loss of appetite in ‘Oorthuva Vatham’.

2 Decrease of Oon causes fatigue and atrophy of pelvic girdle muscles.

In ‘Oorthuva Vatham’ cardiac cachexia and skeletal muscle atrophy are due to decrease of oon.

3 Decrease of Kozhuppu causes emaciation which is one of the features of ‘Oorthuva Vatham’ in chronic stage.

So, Oorthuva Vatham is a disease affecting all the three thathus and most of the udal kattugal.

Eventhough heart is said to be the dwelling places of vatham, pitham and kabam, the normal physiological function of the heart and circulation are maintained by vatham and kabam. So primarily, there is affection of vatham and kabam in Oorthuva Vatham. If this condition is not treated promptly it leads to the affection of pitham and udal kattugal also.

Siddha pathology deals with the diseased condition of the human, which is due to food alteration, seasonal and environmental variations, helding the 14 reflexes and by the behaviour. The disease is reflected through the pulse formed by the three humours.

The underlying cause of 'Oorthuva Vatham' may be due to altered food habits and life style modification. Modern medicine also stresses the same concept.

EVALUATION OF THE DISSERTATION TOPIC

MATERIALS AND METHODS

The clinical study on Oorthuva Vatham was carried out at the post graduate department of Noi Naadal Branch in Government Siddha Medical College, Palayamkottai.

Case selection and supervision

Cases were selected with the allied symptoms of Oorthuva Vatham as mentioned in Yugi vaithiya sinthamani.

The detailed history of the past and present illness, personal and family history were observed.

The author had selected 20 cases to evaluate the typical picture by using siddha as well as modern parameters.

Evaluation of clinical parameters

A detailed history and clinical features of the patients were taken carefully. The clinical history contains

1. **History of cardinal symptoms**
    a) Dyspnea on exertion or breathlessness including paroxysmal nocturnal dyspnea, orthopnea
    b) Chest pain
    c) Cough
    d) Expectoration

e) Haemoptysis

f) Palpitation

g) Syncopal attacks

2. Evidences of congestion

a) Exertional breathlessness

b) Oedema of feet, puffiness of face, anasarca

c) Distension of abdomen and pain in right hypochondrium, anorexia, nausea, vomiting

3. Detailed history of past illness

a) Hypertension

b) Diabetes

c) Coronary artery disease

d) Hyperlipidemia

e) Obesity

f) Recurrent lower respiratory tract infection

g) Tuberculosis

h) Syphilis

i) STD

j) HIV infection

4. History of hospitalization

a) Number of admissions

b) Duration of admission

c) Investigations done e.g. ECG, X-ray, Echo–cardiography, cardiac catheterization

d) Diagnosis reached, if known

e) Treatment history

f) Relief obtained or not

g) Advised surgery / intervention or not

5. History of cardiac surgery, angioplasty or valvuloplasty

6. Family history

a) Hypertension

b) Diabetes

c) Coronary artery disease

d) Hyperlipidemia

e) Congenital heart disease

f) Cardiomyopathies

Study of siddha clinical diagnosis

Modes of investigating the cases are poriyaal arithal, pulanaal arithal and vinaathal which are adopted to assess the humoral pathology. These modes were carried out on the fundamental of udal kattugal and envagai thervugal.

The clinical investigation

For further detailed study about the disease, modern investigatory parameters were used and the following laboratory investigations were done in these cases.

Blood

1. Total count (TC)
2. Differential count (DC)
3. Haemoglobin (Hb)
4. Erythrocyte sedimentation rate (ESR)

Bio-chemical

1. Blood sugar
2. Blood urea
3. Serum cholesterol

Urine

1. Albumin
2. Sugar
3. Deposits

Other tests

1. X- ray chest PA view
2. ECG
3. Echo-cardiogram
4. USG abdomen & pelvis

Results were observed with the respect of the following aspects.

1. Medical history
2. History of socio- economic status
3. Food habits
4. Family history
5. History of smoking in CAHD patients
6. History of alcoholism in Dilated Cardiomyopathy patients
7. Obstetric history (recent pregnancy)
8. Aetiological factors
9. Clinical features
10. Physical examination
11. Laboratory findings
12. X-ray chest findings
13. ECG findings
14. Echo–cardiogram findings
15. USG abdomen & pelvis findings
16. Mukkutra nilaigal
17. Udal thathukkal
18. Envagai thervugal

1. Medical history:

S.No	Previous history	No. of. cases
1	Rheumatism	2
2	Angina	8

2. History of socio-economic status:

S.No	Socio-economic status	No. of cases
1	Poor	5
2	Middle class	6
3	Well-to-do	9

3. Food habits:

S.No	Food habits	No. of cases
1	Vegetarian	5
2	Mixed food habits	15

4. Family history in CAHD patients:

S.No	Family history	No. of cases
1	Positive family history	9
2	Negative family history	5

5. History of smoking in CAHD patients: (Males)

S.No	History of smoking	Pack years	No. of cases
1	Present	20 x 20 = 400	2
		30 x 10 = 300	1
		20 x 10 = 200	5
2	Absent	-	3

Pack years : Duration of smoking in years X number of cigarettes smoked / day.

6. History of alcoholism in Dilated Cardiomyopathy patients: (Males)

S.No	History of alcoholism	Duration	No. of cases
1	Present	30 yrs	1
		20 yrs	1
		15 yrs	1

7. Sex:

S.No	Sex	No. of cases
1	Male	15
2	Female	5

8. Aetiological factors:

S.No	Aetiological factors	No. of cases
1	Coronary artery heart disease	14
2	Cardiomyopathy	4
3	Mitral stenosis	2

9. Clinical features:

S.No	Clinical features	No. of cases
1	Exertional dyspnea	20
2	Orthopnea	15
3	Paroxysmal nocturnal dyspnea (PND)	6
4	Abdominal distension	12
5	Bronchial secretions pouring out of the mouth	1
6	Fatigue	20
7	Low grade fever	5
8	Cardiac cachexia	1
9	Generalised anasarca	1

10. Physical Examination:

(i) General Examination:

S.No	Build and nutrition	No. of cases
1	Obese	5
2	Moderately built	14
3	Cachectic	1

S.No	Nails and conjunctiva	No. of cases
1	Pallor	7
2	Icterus	2

S.No	Oedema	No. of cases
1	Bilateral pitting oedema - legs	20
2	Generalised anasarca	1

(ii) Cardiovascular Examination

(i) Peripheral

S.No	Peripheral	No. of cases
1	↑JVP	20
2	Pulse-irregularly irregular	2
3	Hypertension	
	(i) Grade 1 (mild)	4
	(ii) Grade 2 (Moderate)	3

(ii) Central – Auscultation

S.No	Central - Auscultation	No. of cases
1	Heart sounds	
	S_3 +	12
	Loud P_2	5
2	Murmurs	
	Mid diastolic murmur	2

RELEVANT EXAMINATION OF OTHER SYSTEMS:

(i) Abdomen:

S.No	Abdomen examination	No. of cases
1	Liver	
	- Tenderness	12
	- Hepatomegaly	12
2	Ascites	5

(ii) Respiratory system:

S.No	Respiratory system	No. of cases
1	Basal rales	14

DISTRIBUTION OF MUKKUTTRAM AND UDAL KATTUGAL

Distribution of mukkuttram

a) Derangement of vatham

S.No	Types of vatham	No. of cases affected	Changes
1.	Pranan	20	Circulation to other parts of the body is affected
2.	Abanan	20	Sodium and water retention, oliguria
3.	Viyanan	20	Dyspnea on exertion
4.	Uthanan	-	-
5.	Samanan	20	Increase of other vayus, indigestion and poor tissue perfusion
6.	Naagan	-	-
7.	Koorman	-	
8.	Kirukaran	20	Anorexia
9.	Devathathan	-	-
10.	Thananjayan	1	Generalised anasarca

B) Derangement of pitham

S.No	Types of pitham	No. of cases affected	Changes
1.	Anar pitham	20	Indigestion

2.	Ranjaga pitham	7	Pallor
3.	Sathaga pitham	20	Poor effort tolerance, difficulty in doing normal works
4.	Alosaga pitham	-	-
5.	Prasaga pitham	-	-

C. Derangement of kabam

S.No	Types of kabam	No. of cases affected	Changes
1.	Avalambagam	20	Impairment of the function of the heart
2.	Kilethagam	-	-
3.	Bothagam	-	-
4.	Tharpagam	-	-
5.	Santhigam	-	-

UDAL THATHUKKAL

S.No	Udal thathukkal	No. of cases affected	Changes
1.	Saaram	20	Loss of appetite
2.	Senneer	7	Pallor

3.	Oon	1	Fatigue, atrophy of pelvic girdle muscles
4.	Kozhuppu	1	Emaciation
5.	Enbu	-	-
6.	Moolai	-	-
7.	Sukkilam / suronitham	-	-

THE PICTURE OF ENVAGAI THERVUGAL

1.Naadi:

The Naadi observed in 20 cases was 'Vathakabam'.

S.No	Envagai Thervugal	No. of cases affected	Changes in affected cases	No. of cases not affected
2	Sparisam	5	Pyrexia	15
3	Naa	7	Paleness	13
4	Niram	20	Abnormal shining of the skin over the oedema	-
5	Mozhi	-	-	20
6	Vizhi	7	Paleness	13
7	Malam	5	Loose stools	15
8	Moothiram	4	Oliguria	16

Moothiram

a) Neerkuri

S.No	Characters of urine	Changes	No. of cases
1	Niram – Specific changes in colour	Dark yellow colour	2
2	Edai – Changes in specific gravity	–	–
3	Manam – changes in smell	–	–
4	Nurai - abnormal froth	–	–
5	Enjal – quantity and deposits	Oliguria Increased number of puscells, epithelial cells	4 4

b) Neikuri

S.No	Neikuri	No. of cases	Result
1	Lengthens like a snake	8	Vatha neer
2	Spreading quickly	12	Indicates asaathiyam

ALLIED PARAMETERS

1. ECG findings

The ECG may show evidence of previous myocardial infarction. The most convincing ECG evidence of myocardial ischaemia is obtained by demonstrating reversible ST segment depression or elevation with or without T wave inversion at the time the patient is experiencing symptoms.

In my clinical study on Oorthuva Vatham 14 CAHD patients were selected. The ECG changes observed in them were as follows.

S.No	ECG findings	No. of cases
1.	T wave inversion alone	4
2.	ST elevation alone	1
3.	ST elevation with QS pattern	3
4.	ST depression alone	2
5.	ST depression with T wave inversion	2
6.	Qs pattern in V_1-V_6	1
7.	Atrial fibrillation with rapid ventricular response	1

The ECG changes observed in dilated cardiomyopathy patients are

ST depression with T wave inversion – 3 patients

In mitral stenosis patients, evidence of left atrial hypertrophy is seen and P wave is absent (feature of atrial fibrillation).

2. Echo-cardiogram findings

The Echo findings observed in 14 CAHD patients were

S.No	Echo findings	No. of cases
1.	Severe LV systolic dysfunction	5
2.	Severe LV systolic dysfunction with impaired LV relaxation	6
3.	Moderate LV systolic dysfunction with impaired LV relaxation	3

The Echo findings observed in 4 dilated cardiomyopathy patients were

S.No	Echo findings	No.of cases
1.	Severe LV systolic dysfunction	1
2.	Moderate LV systolic dysfunction	3
3.	Dilatation of all the four chambers	2
4.	Global hypokinesia	2

The Echo findings observed in 2 mitral stenosis patients were

S.No	Echo findings	No.of cases
1.	LA dilatation 0.8 sq cm of M.V.O (mitral valve orifice) Ef 21%	1
2.	LA dilatation 1 sq cm of M.V.O Ef 35%	1

3. Chest x- ray findings

The chest x-ray demonstrated the following findings.

S.No	Chest X-ray findings	No. of cases
1.	Cardiomegaly	20
2.	Pleural effusion (which is demonstrated by peripheral haziness of lung fields)	5
3.	Congested lung fields	5
4.	Lt. atrial enlargement Straightening of the left heart border	2

4. USG abdomen & pelvis

An enlarged, tender, pulsating liver also accompanies systemic venous hypertension and is observed in heart failure from any cause.

Among 20 patients, 12 patients demonstrated congestive hepatomegaly in USG abdomen findings.

MODERN ASPECTS

ANATOMY OF THE HEART

Introduction

The heart is a conical hollow muscular organ situated in the middle mediastinum. It is enclosed within the pericardium. It pumps blood to various parts of the body to meet their nutritive requirements. The Greek name for the heart is cardia from which we have the adjective *cardia.* The Latin name for the heart is cor from which we have the adjective *coronary.*

The heart is placed obliquely behind the body of the sternum and adjoining parts of the costal cartilages, so that one-third of it lies to the right and two-thirds to the left of the median plane. The direction of blood flow, from atria to the ventricles is downwards forwards and to the left. The heart measures about 12 x 9 cm and weighs about 300 g in males and 250 g in females.

External Features

The human heart has four chambers. These are the right and left atria and the right and left ventricles. The atria lie above and behind the ventricles. On the surface of the heart they are separated from the ventricles by an atrioventricular groove. The atria are separated from each other by an interatrial groove.

The atria are separated from the ventricles by a circular *atrioventricular or coronary sulcus.* The *anterior inter-ventricular groove* is

nearer to the left margin of the heart. *The posterior interventricular groove* is situated on the diaphragmatic or inferior surface of the heart. The two interventricular grooves meet at the inferior border near the apex.

Apex of the heart

Apex of the heart is formed entirely by the left ventricle.It is situated in the left fifth intercostal space 9 cm lateral to the midsternal line just medial to the midclavicular line. In the living subject, pulsations may be seen and felt over this region.

Base of the heart

The base of the heart is also called its posterior surface. It is formed mainly by the left atrium and by a small part of the right atrium.

Borders of the heart

The *upper* border is slightly oblique, and is formed by the two atria, chiefly the left atrium. The *right border* is more or less vertical and is formed by the right atrium. The *inferior border is* nearly horizontal and is formed mainly by the right ventricle. The *left border* is oblique and curved. It is formed mainly by the left ventricle, and partly by the left auricle.

Surfaces of the heart

The *anterior or sternocostal surface* is formed mainly by the right atrium and right ventricle: and partly by the left ventricle and left auricle. The *inferior or diaphragmatic surface* rests on the central tendon of the diaphragm. It is formed in its left two-thirds by the left ventricle, and in

its right one-third by the right ventricle. The *left surface* is formed mostly by the left ventricle, and at the upper end by the left auricle.

Musculature of the heart

Cardiac muscle fibres form long loops which are attached to the fibrous skeleton. The atrial fibers are arranged in a superfical transverse layer and a deep antero posterior layer. The ventricular fibres are arranged in superficial, middle and deep layers. The middle layer of fibres of heart are thickest.

The Right Atrium

The right atrium is the right upper chamber of the heart.

External Features

1. The chamber is elongated vertically, receiving the superior vena cava at the upper end and the inferior vena cava at the lower end.
2. The upper end is prolonged to the left to form the right *auricle.*
3. Along the right border of the atrium there is a shallow vertical groove which passes from the superior vena cava above to the inferior vena cava below. This groove is called the sulcus terminalis. The upper part of the sulcus contains the sinuatrial or SA node which acts as the pacemaker of the heart.
4. The right atrioventricular groove separates the right atrium from the right ventricle.

Tributaries or Inlets of the Right Atrium

(i) Superior vena cava, (ii) inferior vena cava, iii) coronary sinus, (iv) anterior cardiac veins,(v) venae cordis minimi (Thebesian veins), (vi) and sometimes the right marginal vein.

Right Atrioventricular Orifice

Blood passes out of the right atrium through the right atrioventricular or tricuspid orifice and goes to the right ventricle.

The Right Ventricle

The right ventricle is a triangular chamber which receives blood from the right atrium and pumps it to the lungs through the pulmonary trunk and pulmonary arteries.

Features

1. Externally, the right ventricle has two surfaces anterior or sternocostal and inferior diaphragmatic.
2. The two parts are separated by a muscular ridge called the supraventricular crest or infundibuloventricular crest situated between the tricuspid and pulmonary orifices.
3. The interior shows two orifices.
    a. the right atrioventricular or tricuspid orifice, guarded by the tricuspid valve, and
    b. the pulmonary orifice guarded by the pulmonary valve.
4. The septomarginal trabecula or moderator band is a muscular ridge. It contains the right branch of the AV bundle.

5. The wall of the right ventricle is thinner than that of the left ventricle in a ratio of 1:3

The Left Atrium

The left atrium is a quadrangular chamber situated posteriorly.

Features

1. The posterior surface of the atrium forms the anterior wall of the oblique of pericardium.
2. The anterior wall of the atrium is formed by the interatrial septum.
3. Two pulmonary veins open into the atrium on each side of the posterior wall.

Arteries supplying the heart

The heart is supplied by two coronary arteries, arising from the ascending aorta. Both arteries run in the coronary sulcus.

Right coronary artery

Right coronary artery is smaller than the left coronary artery. It arises from the anterior aortic sinus.

Branches

(A) Large branches: (1) Marginal, and
(2) posterior interventricular.

(B) Small branches: (1) Nodal in 60% cases, (2) right atrial,
(3) infundibular, and (4) terminal

Area of distribution

1. Right atrium
2. Ventricles

(i) Greater part of the right ventricle, except the area adjoining the anterior interventricular groove.

(ii) A small part of the left ventricle adjoining the posterior interventricular groove.

3. Posterior part of the interventricular septum.
4. Whole of the conducting system of the heart except a part of the left branch of the AV bundle. The SA node is supplied by the left coronary artery in about 40% of cases.

Left Coronary Artery

Left coronary artery is larger than the right coronary artery. It arises from the left posterior aortic sinus.

Branches

A. Large branches: (1) Anterior interventricular, (2) branches to the diaphragmatic surface of the left ventricle, including a large diagonal branch.

B. Small branches: (1) Left atrial, (2) pulmonary, and (3) terminal.

Area of distribution

1. Left atrium
2. Ventricles

(i) Greater part of the left ventricle, except the area adjoining the posterior interventricular groove.

(ii) A small part of the right ventricle adjoining the anterior interventricular groove.

3. Anterior part of the interventricular septum

4. A part of the left branch of the AV bundle

The veins of the heart

These are the great cardiac vein, the middle cardiac vein, the right marginal vein, the posterior vein of the left ventricle, the oblique vein of the left atrium, the right marginal vein, the anterior cardiac veins, and the venae cordis minimi. All veins except the last two drain into the coronary sinus which opens into the right atrium. The anterior cardiac veins and the venae cordis minimae open directly into the right atrium.

A. *Coronary sinus*

The coronary sinus is the largest vein of the heart. It is situated in the left posterior coronary sulcus. It is about 3 cm long. It receives the following tributaries.

1. The great cardiac vein
2. The middle cardiac vein
3. The small cardiac vein
4. The posterior vein of the left ventricle
5. The oblique vein of the left atrium
6. The right marginal vein

B. Anterior cardiac veins

C. Venae cordis minimi

Lymphatics of the heart

Lymphatics of the heart accompany the coronary arteries and form two trunks. The right trunk ends in the brachiocephalic nodes, and the left trunk ends in the tracheobronchial lymph nodes at the bifurcation of the trachea.

Nerve supply of the heart

Parasympathetic nerves reach the heart via the vagus. These are cardioinhibitory; on stimulation they slow down the heart rate. Sympathetic nerves are derived from the upper two to five thoracic segments of the spinal cord. These are cardio-acceleratory, and on stimulation they increase the heart rate, and also dilate the coronary arteries. Both parasympathetic and sympathetic nerves form the superficial and deep cardiac plexuses, the branches of which run along the coronary arteries to reach the myocardium.

PHYSIOLOGY OF THE HEART

The function of cardiovascular system is to supply oxygen, nutrients and other essential substances to the tissues of the body and to remove carbon dioxide and other metabolic end products from the tissues.

Actions of the heart

The activities of the heart are classified into four types:

1. Chronotropic action
2. Inotropic action
3. Dromotropic action
4. Bathmotropic actions.

All these actions of the heart are continuously regulated. It is essential for the heart to cope up with the needs of the body. All the actions are altered by the stimulation of nerves supplying the heart or some hormones or hormonal substances secreted in the body.

1. Chronotropic action

Chronotropic action is the frequency of heartbeat or heart rate. It is of two types:

i. Tachycardia or increase in heart rate

ii. Bradycardia or decrease in the heart rate.

2. Inotropic action

Force of contraction of heart is called inotropic action. It is of two types:

i. Positive inotropic action or increase in the force of contraction

ii. Negative inotropic action or decrease in the force of contraction.

3. Dromotropic action

Dromotropic action is the conduction of impulse through heart. It is of two types:

i. Positive dromotropic action or increase in the velocity of conduction

ii. Negative dromotropic action or decrease in the velocity of conduction.

4. Bathmotropic action

Bathmotropic action is the excitability of cardiac muscle. It is also of two types:

i. Positive bathmotropic action or increase in the excitability of cardiac muscle

ii. Negative bathmotropic action or the decrease in the excitability of cardiac muscle.

Divisions of circulation

The blood flows through two divisions of circulatory system:

1. Systemic circulation
2. Pulmonary circulation.

1. Systemic circulation

It is otherwise known as greater circulation. The blood pumped from left ventricle passes through a series of blood vessels of arterial tree or arterial system and reaches the tissues. The blood vessels of the arterial system are the aorta, larger arteries, smaller arteries and arterioles. The arterioles branch into the capillaries. The capillaries are responsible for exchange of various substances between blood and the tissues. It is because the wall of the capillaries is permeable to various substances.

After exchange of materials at the capillaries, the blood enters the venous system and returns to right atrium of the heart. The blood vessels of the venous tree or venous system are the venules, smaller veins, larger veins and vena cava. From right atrium, blood enters the right ventricle. Thus, through the systemic circulation, the oxygenated blood or arterial blood is supplied from heart to the tissues and the venous blood returns to the heart from the tissues.

2. Pulmonary circulation

It is otherwise called lesser circulation. Blood is pumped from right ventricle to lungs through pulmonary artery. The exchange of gases occurs between blood and alveoli of the lungs through pulmonary capillary membrane. The oxygenated blood returns to left atrium through the pulmonary veins.

Thus, the left side of the heart contains oxygenated or arterial blood and the right side of the heart contains the venous blood.

Properties of cardiac muscle

1. Excitability

Definition

The ability of a tissue to give response to a stimulus is called excitability. In all the tissues, the initial response to a stimulus is the development of action potential. It is followed by the physiological action in the form of contraction, secretion etc.

Action Potential

Action potential in a single cardiac muscle fiber occurs in 4 phases:

1. A rapid depolarization
2. Initial repolarization
3. A plateau
4. Final repolarization.

The approximate duration of the action potential in cardiac muscle is 250 to 350 m sec (0.25 to 0.35 sec).

2. Rhythmicity

Definition

Rhythmicity is the ability of a tissue to produce its own impulses regularly. It is more appropriately named as autorhythmicity. It is also called self-excitation. The property of rhythmicity is present in all the tissues of the heart. However, heart has a specialized excitatory structure from which the discharge of impulses is rapid. This specialized structure is

called pacemaker. From this, the impulses spread to other parts through the specialized conductive system.

3.Conductivity

Human heart has a specialized conductive system through which the impulses from SA node are transmitted to all other parts of the heart.

Conductive system in human heart

The conductive system in human heart comprises:

1.AV node

2.Bundle of His

3.Right and left bundle branches

4.Purkinje fibers.

SA node is situated in right atrium just below the opening of superior vena cava. AV node is situated in the right posterior portion of intra-atrial septum. The impulses from SA node are conducted to AV node by three types of internodal fibers.

1.Anterior internodal fibers of Bachman

2.Middle internodal fibers of Wenckebach

3.Posterior internodal fibers of Thorel.

All these fibers from SA node converge on AV node and interdigitate with fibers of AV node. From AV node, the bundle of His arises. It divides into right and left branches which run on either side of the interventricular septum. From each branch of Bundle of His, many Purkinje fibers arise and spread all over the ventricular myocardium.

4. Contractility

Contractility is ability of the tissue to shorten in length (contraction) after receiving a stimulus. Various factors affect the contractile properties of the cardiac muscle. The different contractile properties are:

I) All or none law

When a stimulus is applied, whatever may be the strength, the whole cardiac muscle responds to the maximum or it does not give response at all. It is called all or none law. Below the threshold level, i.e. if the strength of stimulus is not adequate, the muscle does not give response.

ii) Staircase phenomenon

The staircase phenomenon occurs because of quick succession of stimuli with a time interval of only two seconds in between the stimuli. During this period, the beneficial effect is produced which facilitates the force of successive contraction. So there is a gradual increase in force of contraction.

iii) Summation of subliminal stimuli

When a stimulus with a subliminal strength is applied, the quiescent heart does not show any response. When few stimuli with same subliminal strength are applied in succession, the heart shows response by contraction. It is due to the summation of the stimuli.

iv) Refractory period

It is the period in which the muscle does not show any response to a stimulus. Refractory period is of two types

1.Absolute refractory period

2.Relative refractory period.

Cardiac Cycle

Definition

Cardiac cycle is defined as the sequence of coordinated events which take place during heart beat. Each heart beat consists of two major periods called systole and diastole. During systole, there is contraction of the cardiac muscle and pumping of blood from the heart through arteries. During diastole, there is relaxation of cardiac muscle and filling of blood. Various changes occur in different chambers of the heart during each heart beat. These changes are repeated during every heart beat in a cyclic manner.

Divisions of cardiac cycle

The contraction and relaxation of atria are called atrial systole and atrial diastole respectively. The contraction and relaxation of ventricles are called ventricular systole and ventricular diastole respectively. However, in clinical practice, the term 'systole' refers to ventricular systole and 'diastole' refers to ventricular diastole. Thus events of cardiac cycle are classified into two divisions.

1. Systole
2. Diastole.

Subdivisions and duration of cardiac cycle

When the heart beats at the normal rate of 72/minute, the duration of each cardiac cycle is about 0.8 second. The duration of systole is 0.27 second and that of diastole is 0.53 second. Generally, systole is divided into two subdivisions and diastole is divided into five subdivisions. The subdivisions and the duration systole and diastole are:

Systole		Time (second)
1. Isometric contraction	=	0.05
2. Ejection period	=	0.22
		0.27

Diastole		
1. Protodiastole	=	0.04
2. Isometric relaxation	=	0.08
3. Rapid filling	=	0.11
4. Slow filling	=	0.19
5. Atrial systole	=	0.11
		0.53

The total duration of cardiac cycle is 0.27 + 0.53 = 0.8 second.

Regulation of heart pumping

When a person is at rest, the heart pumps only 4 to 6 liters of blood each minute. During severe exercise, the heart may be required to pump

four to seven times this amount. This section discusses the means by which the heart can adapt to such extreme increases in cardiac output.

The basic means by which the volume pumped by the heart is regulated are

1. Intrinsic cardiac regulation of pumping in response to changes in volume of blood flowing into the heart and
2. Control of the heart by the autonomic nervous system.

Intrinsic regulation of heart pumping – The Frank-Starling Mechanism

The amount of blood pumped by the heart each minute is determined by the rate of blood flow into the heart from the veins, which is called venous return. That is, each peripheral tissue of the body controls its own blood flow, and the total of all the local blood flow through all the peripheral tissues returns by way of the veins to the right atrium. The heart in turn automatically pumps this incoming blood into the systemic arteries, so that it can flow around the circuit again.

This intrinsic ability of the heart to adapt to changing volumes of inflowing blood is called the Frank - Starling mechanism of the heart, in honor of Frank and Starling, two great physiologists of nearly a century ago. Basically, the Frank - Starling mechanism means that the greater the heart muscle is stretched during filling, the greater will be the force of contraction and the greater will be the quantity of the blood pumped into the aorta. Or another way to express this is: within physiological limits, the

heart pumps all the blood that comes to it without allowing excessive damming of blood in the veins.

Explanation of the Frank-Starling Mechanism

When an extra amount of blood flows into the ventricles, the cardiac muscle itself is stretched to a greater length. This in turn causes the muscle to contract with increased force because the actin and myosin filaments are then brought to a more nearly optimal degree of interdigitation for force generation. Therefore, the ventricle, because of its increased pumping, automatically pumps the extra blood into the arteries. This ability of stretched muscle, up to an optimal length, to contract with increased force is characteristic of all striated muscle, not simply of cardiac muscle.

In addition to the important effect of stretching the heart muscle, still another factor increases heart pumping when its volume is increased. Stretch of the right atrial wall directly increases the heart rate by 10 to 20 percent ; this, too, helps increase the amount of blood pumped each minute, although its contribution is much less than that of the Frank - Starling mechanism.

Heart Sounds

Introduction

The mechanical activities of the heart during each cardiac cycle produce some sounds, which are called heart sounds. Generally, heart sounds are produced by movements of:

1. Blood through the chambers of the heart
2. Cardiac muscle
3. Valves of the heart.

The heart sounds are heard by placing the ear over the chest or by using a stethoscope or microphone. These sounds are also recorded graphically.

Different heart sounds

Four heart sounds are produced during each cardiac cycle. The first and second heart sounds are more prominent and resemble the spoken words 'LUBB' and ‘DUBB' respectively. These two heart sounds are heard by using the stethoscope.

Third heart sound is a mild sound and it cannot be heard by using stethoscope. It is heard by using a microphone. The fourth heart sound is an inaudible sound. This sound is studied only by graphic registration, i.e. the phonocardiogram.

Importance of heart sounds

The study of heart sounds has important diagnostic value in clinical practice because; the alteration in the heart sounds indicates the cardiac diseases involving the valves of the heart.

HEART SOUNDS

Features	First heart sound	Second heart sound	Third heart sound	Fourth heart sound
Occurs during	Isometric contraction period and part of ejection period.	Protodiastole and part of isometric relaxation	Rapid filling phase	Atrial systole
Cause	Closure of atrioventricular valves	Closure of semilunar valves	Rushing of blood into ventricle	Contraction of atrial musculature
Characteristics	Long, soft and low pitched. Resembles the word 'LUBB'	Short, sharp and high pitched. Resembles the word 'DUBB'	Low pitched	Inaudible sound
Duration (sec)	0.10 to 0.17	0.10 to 0.14	0.07 to 0.10	0.02 to 0.04
Frequency (cycles per sec)	25 to 45	50	1 to 6	1 to 4
Relation with ECG	Coincides with peak of ‘R' wave	Precedes or appears 0.09 second after peak of ‘T’ wave	Between ‘T’ wave and 'P' wave	Between 'P' wave and 'Q' wave
Vibrations in phonocardiogram	9 to 13	4 to 6	1 to 4	1 to 2

CONGESTIVE HEART FAILURE

Heart failure is defined as the pathophysiologic state in which impaired cardiac function is unable to maintain an adequate circulation for the metabolic needs of the tissues of the body.

As blood flow out of the heart slows blood returning to the heart through the veins backs up, causing congestion in the tissues.

The term Congestive Heart Failure (CHF) is used for the chronic form of heart failure in which the patient has evidence of congestion of peripheral circulation and of lungs. CHF is the end–result of various forms of serious heart diseases.

Etiology

Heart failure may be caused by one of the following factors, either singly or in combination.

1. Intrinsic pump failure

The most common and most important cause of heart failure is weakening of the ventricular muscle due to disease so that the heart fails to act as an efficient pump. The various diseases which may culminate in pump failure by this mechanisms are

i) Ischaemic heart disease

ii) Myocarditis

iii) Cardiomyopathies

iv) Metabolic disorders , e.g. beriberi.

v) Disorders of the rhythm, e.g. atrial fibrillation and flutter.

2. Increased workload on the heart

Increased mechanical load on the heart results in increased myocardial demand resulting in myocardial failure. Increased load on the heart may be in the form of pressure load or volume load.

i) Increased pressure load may occur in the following states.

a) Systemic and pulmonary arterial hypertension.

b) Valvular disease. eg: mitral stenosis, aortic stenosis, pulmonary stenosis

c) Chronic lung diseases.

ii) Increased volume load occurs when a ventricle is required to eject more than normal volume of the blood resulting in cardiac failure. This is found in the following conditions.

a) Valvular insufficiency

b) Severe anemia

c) Thyrotoxicosis

d) Arteriovenous shunts

e) Hypoxia due to lung diseases.

3. Impaired filling of cardiac chambers

Decreased cardiac output and cardiac failure may result from extra cardiac causes or defect in the filling of the heart.

a) Cardiac tamponade. eg haemopericardium, hydropericardium.

b) Constrictive pericarditis.

Chronic heart failure

More often, heart failure develops slowly as observed in the following states.

i) Myocardial ischaemia from atherosclerotic coronary artery disease.

ii) Multivalvular heart disease.

iii) Systemic arterial hypertension.

iv) Chronic lung diseases resulting in tension hypoxia and pulmonary arterial hypertension.

v) Progression of acute into chronic failure.

Left sided and right sided heart failure

Though heart as an organ eventually fails as a whole, but functionally, the left and right heart act as independent units.

Left-sided heart failure

Left–sided heart failure is initiated by stress to the left heart. The major causes are:

i) Systemic hypertension

ii) Mitral or aortic valve disease (stenosis)

iii) Ischaemic heart disease.

iv) Myocardial diseases. eg. Cardiomyopathies , myocarditis.

v) Restrictive pericarditis.

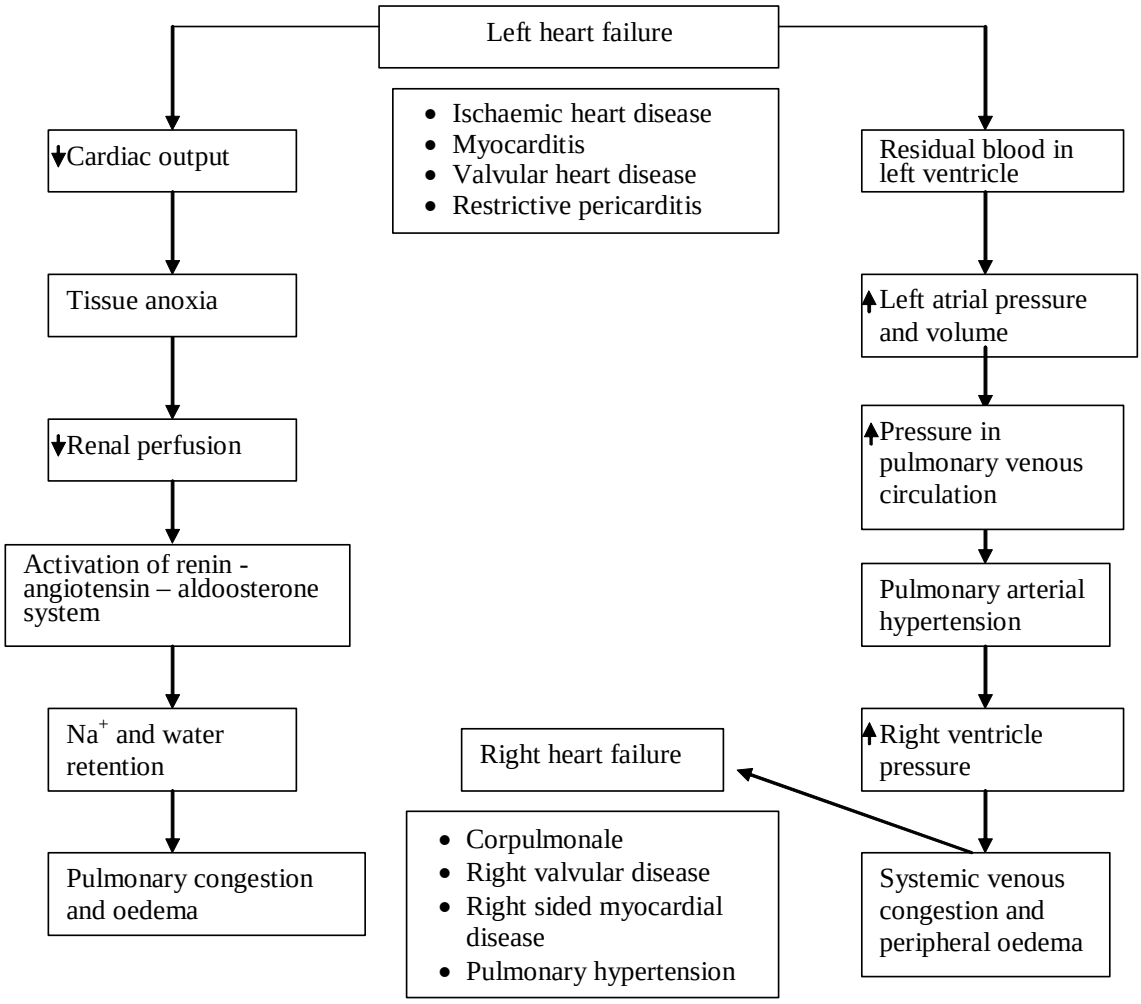
Right–sided heart failure

Right sided heart failure occurs more often as a consequence of left sided heart failure. However, some conditions affect the right ventricle primarily producing right sided heart failure.

These are as follows:

i) As a consequence of left ventricular failure.
ii) Cor pulmonale in which right heart failure occurs due to intrinsic lung diseases.
iii) Pulmonary or tricuspid valvular disease.
iv) Pulmonary hypertension secondary to pulmonary thromboembolism.
v) Myocardial disease affecting right side.
vi) Congenital heart disease with left-to-right shunt.

SCHEMATIC EVOLUTION OF CONGESTIVE HEART FAILURE AND ITS EFFECTS.



In evaluating patients with heart failure, it is important to identify not only the underlying cause of the heart disease but also the precipitating cause of heart failure. Identification of such precipitating causes is of critical importance because their prompt alleviation may be life saving.

Precipitating causes

1. Infection

Patients with pulmonary vascular congestion are also more susceptible to pulmonary infections; any infection may precipitate heart failure. The resulting fever, tachycardia and hypoxemia and the increased metabolic demands may place a further burden on the overloaded, but compensated myocardium of a patient with chronic heart disease.

2. Anemia

In the presence of anemia, the oxygen needs of the metabolizing tissues can be met only by an increase in the cardiac output. In this manner, the combination of anemia and previously compensated heart disease can lead to inadequate oxygen delivery to the periphery and precipitate heart failure.

3. Thyrotoxicosis and pregnancy

As in anemia and fever in thyrotoxicosis and pregnancy, adequate tissue perfusion requires an increased cardiac output.

Similarly heart failure not frequently occurs for the first time during pregnancy in women with rheumatic valvular disease in whom cardiac compensation may return following delivery.

4. Arrhythmias

In patients with compensated heart disease, arrhythmias are among the most frequent precipitating cause of heart failure.

5. Rheumatic and other forms of myocarditis

Acute rheumatic fever and a variety of other inflammatory or infectious processes affecting the myocardium may impair myocardial function in patients with or without pre-existing heart disease.

6. Infective endocarditis

The additional valvular damage, anemia, fever and myocarditis that often occur as a consequence of infective endocarditis may singly or in concert, precipitate heart failure.

7. Physical, dietary, fluid, environmental and emotional excesses

The augmentation of sodium intake, the inappropriate discontinuation of medications to treat heart failure, blood transfusions, physical overexertion, excessive environmental heat or humidity and emotional crises all may precipitate heart failure in patients with heart disease who were previously compensated.

8. Systemic hypertension

Rapid elevation of arterial pressure, as may occur in some instances of hypertension of renal origin or upon discontinuation of anti-hypertensive medication may result in cardiac decompensation.

9. Myocardial infarction

In patients with chronic but compensated ischemic heart disease, a fresh infarct, sometimes otherwise silent clinically, may further impair ventricular function and precipitate heart failure.

10. Pulmonary embolism

Physically inactive patients with low cardiac output are at increased risk of developing thrombi in the veins of the lower extremities of the pelvis. Pulmonary emboli may result in further elevation of pulmonary arterial pressure which in turn may produce or intensify ventricular failure. In the presence of pulmonary vascular congestion, such emboli also may cause pulmonary infarction.

Clinical Manifestations

Congestive heart failure combines the features of the both right and left heart failure.

The clinical manifestations of left-sided heart failure result from accumulation of fluid upstream in the lungs and from decreased left ventricular output. Accordingly the major pathologic changes are as under:

i) Pulmonary congestion and oedema causing dyspnea and orthopnea.

ii) Decreased left ventricular output causing hypoperfusion and diminished oxygenation of tissues. eg

1) In kidneys causing ischemic acute tubular necrosis.
2) In brain causing hypoxic encephalopathy.
3) And in skeletal muscles causing muscular weakness and fatigue.

The clinical manifestations of right-sided heart failure are upstream of the right heart such as systemic and portal venous congestion and reduced cardiac output. Accordingly, the pathologic changes are as under:

i) Systemic venous congestion in different tissues and organs. e.g. subcutaneous oedema on dependent parts, passive congestion of the liver, spleen and kidneys, ascites, hydrothorax, congestion of leg veins and neck veins.

ii) Reduced cardiac output resulting in circulatory stagnation causing anoxia, cyanosis and coldness of extremities.

In summary, in early stage the left heart failure manifests with features of pulmonary congestion and decreased left ventricular output, while the right heart failure presents with systemic venous congestion and involvement of the liver and spleen.

Signs

1. Raised JVP – Positive hepato-jugular reflux

2. Enlarged and tender liver – systolic pulsation of liver if tricuspid incompetence.

3. Oedema – The erect position favours collection of fluid in feet, ankles and recumbent position favours accumulation in sacral region. If oedema is severe it may be associated with hydrothorax (more often on right side) and hydropericardium and occasionally ascites.

4. Evidence of heart disease

- Signs associated with underlying disease

- Cardiomegaly with evidence of right ventricular or combined ventricular enlargement.

- Rt. ventricular gallop murmur of functional TR common.

5. Peripheral cyanosis – may occur due to slow peripheral circulation

6. Cardiac cachexia – loss of subcutaneous fat and muscle tissue.

Framingham criteria for diagnosis of CCF

Major criteria

- Paroxysmal nocturnal dyspnea
- Neck vein distension
- Rales
- Cardiomegaly
- Acute pulmonary oedema
- S_3 gallop
- Increased venous pressure (>16cm H_2O)
- Positive hepatojugular reflux

Minor criteria

- Extremity oedema
- Night cough
- Dyspnea on exertion
- Hepatomegaly
- Pleural effusion

- Vital capacity reduced by 1/3rd from normal
- Tachycardia ($\geq$ 120 bpm)

Major minor

Weight loss $\geq$ 4.5 kg over 5 days treatment

To establish a clinical diagnosis of CCF by these criteria at least one major and 2 minor criteria are required.

Pathophysiology

Central to any consideration of CHF is a discussion of cardiac hypertrophy, the compensatory response of the myocardium to increased work. Myocardial hyperfunction induces increased myocyte size (cellular hypertrophy through addition of sarcomeres, the contractile elements) that causes an increase in the overall mass and size of the heart. The diameters of cardiac myocytes can increase from the normal 15 μm to 25μm or more in hypertrophy.

The structural / biochemical / molecular basis for myocardial contractile failure is obscure in many cases. In some instances (e.g., myocardial infarction), there is obvious death of myocytes and loss of vital elements of the 'pump'; the remaining, non infarcted regions of cardiac muscle are over worked. In contrast, in valvular heart disease, increased pressure or volume work affects the chamber wall globally. The increased myocyte size that occurs in cardiac hypertrophy is usually accompanied by decreased capillary density, increased intercapillary distance and deposition of fibrous tissue.

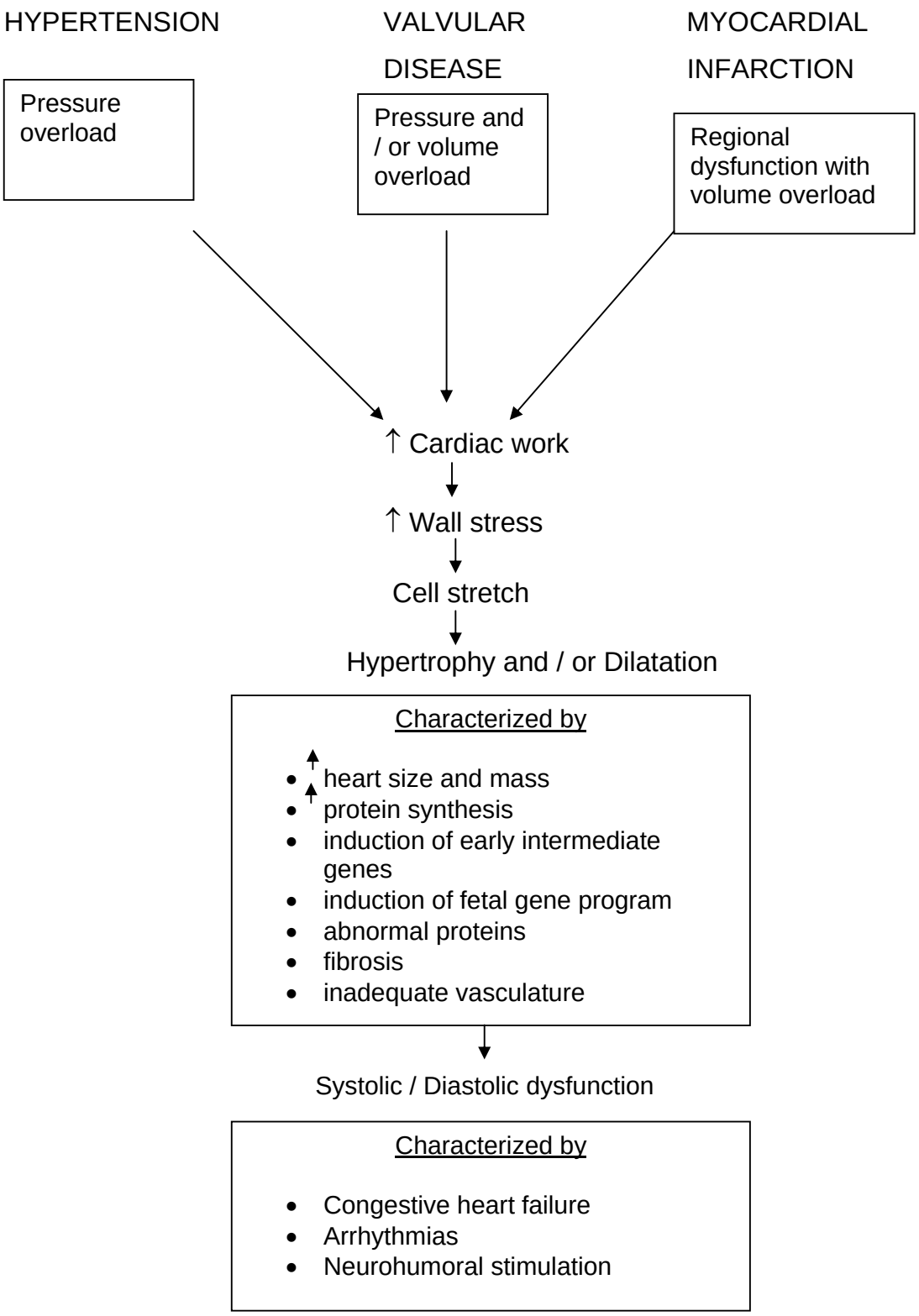
Moreover, the molecular changes in hypertrophied hearts that initially mediate enhanced function may contribute to the development of heart failure. With prolonged hemodynamic overload, gene expression is altered, leading to re-expression of a pattern of protein synthesis analogous to that seen in fetal cardiac development; other changes are analogous to events that occur during mitosis of normally proliferating cells. Thus proteins related to contractile elements, excitation–contraction coupling, and energy utilization my be significantly altered through production of different isoforms that either may be less functional than normal or may be reduced or increased in amount.

Whatever, the underlying basis for CHF, a variety of compensatory mechanisms come into play when the hypertrophied heart can no longer accommodate the increased demand. The heart begins to dilate, as discussed earlier, thereby stretching the sarcomeres and increasing the force of contraction and secondarily the stroke volume. Increased stretching of myocytes, however, leads to further hypertrophy. Simultaneously there is expansion of the blood volume, further augmenting stroke volume. Eventually, however, the compensatory mechanisms themselves constitute an added burden. Myocardial hypertrophy may become increasingly detrimental because of the increased metabolic requirements of the enlarged muscle mass. Indeed, muscle mass and wall tension are major determinants of the oxygen consumption of the heart; the other major factors are heart rate and contractility (inotropic state, or force

of contraction). Increased blood volume, which supports the cardiac output in the short term, also imposes an additional load on the failing heart.

Ultimately, the primary cardiac disease and the superimposed compensatory burdens further encroach on the myocardial reserve until cardiac dilatation progresses beyond the point at which adequate myocardial tension can be generated. Then begins the downward slide of stroke volume and cardiac output that often ends in death.

SCHEMATIC REPRESENTATION OF THE SEQUENCE OF EVENTS IN HYPERTROPHY AND HEART FAILURE, EMPHASIZING CARDIAC CELLULAR AND EXTRA CELLULAR CHANGES



General measures for the management of heart failure

Education

Explanation of nature of disease, treatment and self - help strategies.

Diet

- ❖ Good general nutrition and weight reduction for the obese
- ❖ Avoidance of high-salt foods and added salt, especially for patients with severe congestive heart failure.

Alcohol

- ❖ Moderate or eliminate alcohol consumption
- ❖ Alcohol induced cardiomyopathy requires abstinence

Smoking

Stopping

Exercise

Regular moderate aerobic exercise within limits of symptoms

Vaccination

Influenza and pneumococcal vaccination should be considered

Complications

In advanced heart failure a number of non-specific complications may occur.

- Renal failure
- Hypokalaemia
- Hyperkalaemia
- Hyponatraemia

- Impaired liver function
- Atrial and ventricular arrhythmias

Diagnosis and investigations

When symptoms suggest congestive heart failure, the diagnosis usually is confirmed by physical examination, patient history and various tests to detect abnormal function of the left ventricle and / or heart valves.

During physical examination the physician looks for an underlying cause and assesses heart function.

A stethoscope is used to detect abnormal heart sounds (murmurs) that may indicate a leaky or narrowed (stenotic valve) and to detect fluid accumulation in the lungs.

The physician also looks for enlarged (distended) veins in the neck and for swelling in the legs (oedema particularly in the ankles and feet) and / or the abdomen.

A patient history may include gathering information about the following

- Alcohol and drug use
- History of hypertension including treatment
- Prior chest pains or heart attack
- Recent viral illness
- Recent pregnancy

Simple tests (eg. urea, electrolytes, hemoglobin, thyroid function, ECG, chest X-ray) may help to establish the nature and severity of the underlying heart disease and detect any complication.

Echocardiography is a very useful investigation and should be considered in all patients with significant heart disease in order to

- assess ejection fraction

(Normally approximately 60% of the blood in the left ventricle is ejected each time the heart beats (contracts)

Pts with 40 $\hat{A}$ – 45 % EF – Mildly depressed ejection fraction

Pts with 35 $\hat{A}$ – 40 % EF – Moderately depressed ejection fraction

Pts with 10 $\hat{A}$ – 25 % EF – Severely depressed ejection fraction

- determine the aetiology (ie, coronary artery disease, heart attack and valve dysfunction)
- detect hitherto unsuspected valvular heart disease (eg.occult mitral stenosis) and other conditions that may be amenable to specific remedies
- identify patients who will benefit from long term therapy with drugs.

Brain natriuretic peptide (BNP) is elevated in heart failure and can be used as a screening test in breathless patients and those with oedema.

The chest X-ray in left heart failure

A rise in pulmonary venous pressure from left sided cardiac failure first shows on the chest X-ray as an abnormal distension of the upper lobe

pulmonary veins (with the patient in the erect position). The vascularity of the lung fields becomes more prominent and the right and left pulmonary arteries dilate. Subsequently interstitial oedema cause thickened interlobular septa and dilated lymphatics. These are evident as horizontal lines in the costophrenic angles (septal or ‘Kerley B’ lines). More advanced changes due to alveolar oedema cause a hazy opacification spreading from the hilar regions and pleural effusions.

- Cardiac catheterization may be performed in patients with angina and patients with a history of heart attack to determine if coronary heart disease (CHD) is causing heart failure.

- In some cases, a less invasive procedure (eg stress test) is used to assess the possibility of coronary heart disease.

DISCUSSION

20 patients were selected for the clinical study on “Oorthuva Vatham” and all patients were undergone investigations by both siddha as well as modern parameters. The results observed on various headings were discussed for further diagnosis.

INTERPRETATION OF CLINICAL HISTORY

1) Age

In women beyond the menopausal age group, the incidence of problems like ischaemic heart disease increases in equal propotion as that in their male counterparts. Similarly all the 3 female patients of CAHD are beyond the menopausal age group.

2) Sex

Males are more prone to develop conditions like CAHD, as they are habituated to smoking and consumption of alcohol in larger numbers than their female counterparts. In clinical study on Oorthuva Vatham 15 patients out of 20 patients are males.

3) Address

People hailing from the urban region are prone to develop problems related to urbanisation like exposure to constant stress and problems developing consequent to this, eg CAHD. The study revealed that 10 out of 14 CAHD patients are hailing from urban regions.

4) History of previous illness

Mitral stenosis is almost always rheumatic in origin. The study revealed that 2 mitral stenosis patients had past history of rheumatic fever at the age of 10 years. 8 out of 14 CAHD patients had past history of angina.

5) Family history

Presence of CAHD in the patient's close relatives may make the patient more prone to develop a similar problem. Similarly among 14 patients who had developed CAHD, 9 patients had positive family history.

6) Social history

i) Smoking

Since smoking is the most important single risk factor causing CAHD, 8 out of 11 male patients had the history of smoking.

ii) Alcoholism

Alcohol is an important aetiological factor in a significant propotion of cardiomyopathy patients. The clinical history revealed that all the 3 male patients of dilated cardiomyopathy had the history of alcoholism.

INTERPRETATION OF ALLIED PARAMETERS

1.ECG

ECG of CAHD patients showed evidence of previous MI. The ECG changes in dilated cardiomyopathy were non–specific. In mitral stenosis there was evidence of left atrial hypertrophy, pulmonary hypertension and features of atrial fibrillation.

2. Echo–cardiogram

Normally approximately 60% of blood in the left ventricle is ejected each time the heart beats (contracts). Ventricular function was affected in all patients and all had depressed Ef (Ejection fraction). There was dilatation of all the four chambers in dilated cardiomyopathy.

In Echo findings of mitral stenosis patients, there was left atrial dilatation and reduced M.V.O (mitral valve orifice). In rheumatic mitral stenosis, the mitral valve orifice is slowly diminished by progressive fibrosis and calcification of the valve leaflets. So the flow of blood from left atrium to left ventricle is restricted and left atrial pressure rises. There is dilatation and hypertrophy of the left atrium. Normally the M.V.O is about $5cm^2$. The patients usually remain asymptomatic until the stenosis is approximately $2cm^2$ or less.

3. Chest X–ray

The size of the heart was enlarged in all patients. Pleural effusion was seen because of pulmonary venous congestion. Left atrial

enlargement was seen because of restricted blood flow from left atrium to left ventricle in mitral stenosis patients.

4. USG abdomen & pelvis

Due to inability of the heart to empty properly, there is increased venous pressure and congestion of tissues in portal venous system in congestive heart failure. The USG abdomen report revealed congested hepatomegaly.

Routine blood tests and urine tests were done in all the patients.

5. Blood test:

In total count of WBC and differential count of WBC, there was no significant changes.

ESR was elevated in most of the patients and this may be due to lung infection .

Haemoglobin level was reduced in all the patients and this may be due to indigestion and loss of appetite etc.

7 patients were diabetic and their reports revealed increased blood sugar levels.

The level of urea was elevated in 4 patients and the level of creatinine was elevated in 3 patients.

10 patients had elevated cholesterol levels. Among them 8 patients had moderately increased cholesterol levels (200 – 250mg/dl) and 2 patients had severely increased cholesterol levels (> 300mg/dl).

Among 20 patients, 2 patients suffered from jaundice and they had elevated bilirubin levels in blood.

6. Urine test

2 patients showed albuminuria and 6 patients showed presence of sugar in the urine.

The urine of 4 patients revealed increased number of puscells and epithelial cells.

The urine of 2 jaundice patients showed presence of bile salts and bile pigments.

INTERPRETATION OF ENVAGAI THERVUGAL

1. Naadi

The naadi observed in all patients was “Vathakabam”

2. Sparisam

Five patients had mild pyrexia and this may be due to lung infection.

3. Naa

On examination of the tongue, paleness was present in 7 patients because of reduced Hb levels.

4. Niram

In the observation of the colour of the body, all the patients had abnormal shining of the skin over the oedema.

5. Vizhi

The conjunctiva was pale in 7 patients because of reduced Hb levels.

6. Malam

5 patients had loose stools because of congestion of tissues in the abdomen.

7. Moothiram

1. Niram 2 jaundice patients had dark yellow coloured urine due to the presence of bile salts and bile pigments in the urine.

2. Enjal : The quantity was decreased in 4 patients and they had increased pus cells and epithelial cells in the urine.

Neikuri

The neikuri lengthened like a snake in 8 patients and it denotes ‘Vatha Neer’.

The Neikuri spreaded quickly in 12 patients and it denotes ‘Asaathiyam’.

MUKKUTRA NILAIGAL AND UDAL KATTUGAL

1. Vatham

Pranan, Viyanan, Abanan, Samanan, Kirukaran and Thananjayan are affected since of characters such as impaired circulation of the heart, dyspnea on exertion, oliguria, steatorrhoea, generalised anasarca and indigestion.

2. Pitham

Anarpitham, Saathagapitham and Prachaga pitham are affected since of characters such as indigestion, poor effort tolerance and generalised anasarca.

3. Kabam

Avalambagam is affected since of character such as impairment of cardiac function.

UDAL KATTUGAL

Increase in Saaram causes loss of appetite in Oorthuva Vatham.

Decrease in Oon and Kozhuppu cause cardiac cachexia.

HIGHLIGHTS OF THE DISSERTATION

'Oorthuva Vatham' comes under 'Vatha Roga Nithanam' in Yugi vaithiya sinthamani 800 which is characterized by orthopnea, abdominal distension, pulmonary oedema, pain in the mouth, neck and chest, low grade fever, fatigue, cardiac cachexia, generalised oedema (anasarca).

Since pranan is said to be the centre of the heart, it controls heart and circulation. In Oorthuva Vatham, pranan is primarily affected and results in impaired circulation to other parts of the body. If this acute condition is not treated it leads to the affection of other thathus and udal kattugal also.

In siddha text books heart diseases are classified under the headings like Thamaraga noi, Maarbu noi and Iruthaya rogam. In Yugi vaithiya sinthamani, Yugi discussed the symptoms of congestive heart failure under the heading 'Oorthuva Vatham' which is unknown to us.

Heart is the first organ through which all the blood of the body passes during circulation. So heart diseases have serious effects on other systems of the body also. If properly recognized earlier, the disease can be treated more effectively and the complications can be prevented.

PREVENTIVE MEASURES

Our food habits and life style may predispose to heart diseases. For e.g., diets largely derived from fats than protein-rich diet cause hyperlipidemia. Dietary imbalance, overnutrition, inactivity and sedentary life style lead to obesity.

Ancient people followed healthy and restricted food habits. So they were able to prevent many of the disease causing factors.

Suvai in the food have effects in the health of the human body. Their proper proportion in food causes well being and their excessive or less intake causes deleterious effects. For e.g. excessive intake of astringent causes heart diseases, fatigue , constipation, abdominal pain etc.

Yogic physical culture tone up all the involuntary organs of the body which are mainly concerned with such processes as circulation, respiration, secretion, digestion, evacuation etc., Without doubt yogic exercises assure a normal individual of his physical well being; but they are also both curative and recuperative in action.

According to siddhars, the 14 natural reflexes (14 vegangal) involved in the physiology of normal human beings and if willfully suppressed, it leads to the affection of normal function of the body.

CONCLUSION

The lines that were said by Yugi in Yugi vaithiya sinthamani 800 under the heading “Oorthuva Vatham” well explained the clinical condition similar to “Congestive Cardiac Failure”.

The lines of the version were well analysed on the siddha and modern parameters and the patients were thoroughly examined with clinical and bio-chemical analysis.

“Poriyaal arithal”, “Pulanaal arithal”, “Vinaathal”, “Envagai Thervugal”, “Neerkuri” and “Neikuri” helped in the proper diagnosis of the disease.

Routine biochemical examination were carried out to know the general condition of all patients. Echo, ECG, X-ray chest and USG abdomen & pelvis were carried out to confirm the diagnosis.

As a siddha physician we know the importance of our “Noi Illa Neri” principles which can only prevent our people from many of these ailments. These principles were said by learned Yogis for the sake of our people. We should guide the people in the proper way and make them to follow these principles to achieve the WHO target (Health for all by 2000 AD).

P.G.RESEARCH CENTRE

GOVT.SIDDHA MEDICAL COLLEGE, PALAYAMKOTTAI

NOI NADAL BRANH – V

ANNEXURE – 1

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

Complaints & Duration	:
H/O Present illness	:
H/O Previous illness	:
Personal History	:
Family History	:

Clinical Examination – Siddha aspect

General Examination

Yakkai	:
Gunam	:
Irukkai nilai	:
Padukkai nilai	:

Suvasa enn :

Kuruthi azhutham :

Special Examination

Pori / Pulan

Mei - Sensation :

Vaai - Taste :

Kan - Sight :

Mooku - Smell :

Sevi - Hearing :

Kanmendriyam / Vidayam

Vaai - Vasanam :

Kai - Dhanam :

Kaal - Kamanam :

Eruvai - Visarkam :

Karuvai - Anantham :

Paruvakalam

Karkalam :

Koothirkalam :

Munpanikalam :

Pinpanikalam :

Elavernirkalam :

Mudhuvenirkalam :

Utkayam / Athakayam

Puyam - Foreran :

Sayam - Arm :

Kaal - Leg :

Paatham - Feet :

Uyir thathukkal

1)Vatham

Pranan :

Abanan :

Viyanan :

Uthanan :

Samanan :

Nagan :

Koorman :

Kiruharan :

Deathathan :

Dhananjayan :

2) Pitham

Anilam :

Ranjagam :

Pirasagam :

Aalosagam :

Sathagam :

3) Kabham

Avalambagam :

Kilethagam :

Pothagam :

Tharpagam :

Santhigam :

Ezhu Udal Thathukkal

Saaram :

Senneer :

Oon :

Kozhuppu :

Enbu :

Majjai :

Sukkilam / Suronitham :

EN VAGAI THERVUGAL

MEI KURI (SPARISM)

Examination of the Skin

Inspection

Colour of the Skin

Eruptions

Haemorrhages

Ulcers, excoriations, fissures etc.

Boils, carbuncles, scars, trophic changes etc.

Eruption

Types of rashes

Maccular

Roseolar

Erythematous

Papular

Pustular

Lenticular

Nodular

Vesicular

Bullous

Wheals

Burrows

Blackheads

Plaques

Scales

Ulcers

Duration

Mode of onset

Associated pain

Size and pain

Nature of the floor

Character of the edge

Discharge

Tenderness

Surrounding skin

Lymphnodes

Pruritis

Infestation

Skin diseases

Metabolic & endocrine

Hepatic disorders

Renal diseases

Blood diseases

Examination of the hair

Falling of the hair

Patchy loss of hair

Loss of hair in temporal region

Characteristic features of the hair

Sweat

Physiological / Pathological

Lymphglands

Site

Shape

Size

Consistency

Mobility

Tenderness

Examination of the nails

Examination of the Head, neck, Face

Skull

Size

Shape

Face

Eyebrows

Eye lids & Eye lashes

Nose

Lips

Ears

Neck

Examination of the Chest

Shape and Size

Movements

Rate of respiration

Breath Sounds : Normal / Abnormal

Heart Rate & Sounds

Examination of the Breast

Examination of the Abdomen

Shape

Size

Examination of the Genital Organs

Examination of the Extermitis

Upper & Lower Limb **:** General Examinations

Special Examinations

Tests for Tone, Power & reflex

NIRAM

Colour of the skin, Hair, Nail, Teeth, Tongue, Gums

Sputum – Normal / Abnormal

MOZHI

Larynx

Congenital

Acquired

Traumatic

Tongue

Congential Abnormalities

Ear **:** Deafness

Palate **:** Cleft palate

VIZHI

Examination of Eye

Visual acuity

Visual field

Colour sense

Pupil

Size

Equality

Regularity

Reaction of light accommodation

NAA

Colour

Size

Shape

IRU MALAM

Malam

I. Macroscopic Examination

Amount

Colour

Odour

Consistency

Abnormal Constituents

II. Microscopic Examination

III. Chemical Examination

Siruneer

Quantity

Colour & Transparency

Specific Gravity

Deposit

NAADI

The state of vatha, pitha and kabha naadi.

Examination of Pulse & its Indication

Rate

Rhythm

Volume

Force &

Character

Noi kanippu

MODERN ASPECTS

ANNEXURE – II

General Examination

Consciousness	:	General Appearance	:
State	:	Nourishment	:
Weight	:	Facies	:
Height	:	Jaundice	:
Skin Changes	:	Engorged venis	:
Anaemia	:	Clubbing	:
Cyanosis	:	JVP	:
Pedal Odema	:	Koilonychia	:
Abdominial distension	:	Brittle Nail	:
Congential anomally	:		
Lymphadenopathy	:		

Pluse Rate	Rhythm	Volume (Rt)	Volume (Lt)	Character
Blood Presure : mm/Hg	Upper limb	-------	-------	
	Lower limb	-------	-------	

Respiratory Rate:

Systematic Examination

Cardiovascular System :

Respiratory System :

Gastro intestinal System :

Central nervous System :

Laboratory Investigations

Blood

TC	:	MCV	:
DC: P, L, E, B, M	:	MCH	:
Hb%	:	MCHC	:
ESR		Serum Protein	:
1/2hr	:	Serum Cholestrol	:
1hr	:	Blood Urea	:
RBC Count	:	Serum Iron	:
Platelet Count	:	Serum Ferritin	:
Reticulocyte Count	:	Serum TIBC	:
PCV	:	Peripheral Blood Smear	:

Motion

Ova :

Cyst :

Occult blood :

Urine

Albumin :

Sugar :

Deposits :

Bile Salt :

Bile Pigment :

Special Investigation

Barium meal and endoscopy :

Bone marrow examination :

Skiagram :

Sputum for AFB :

Radiological investigation :

Ophthalmoscopic examination :

E.C.G.

Etc. ***:***

Case Summary ***:***

Fate of the Disease ***:***

Line of treatment ***:***

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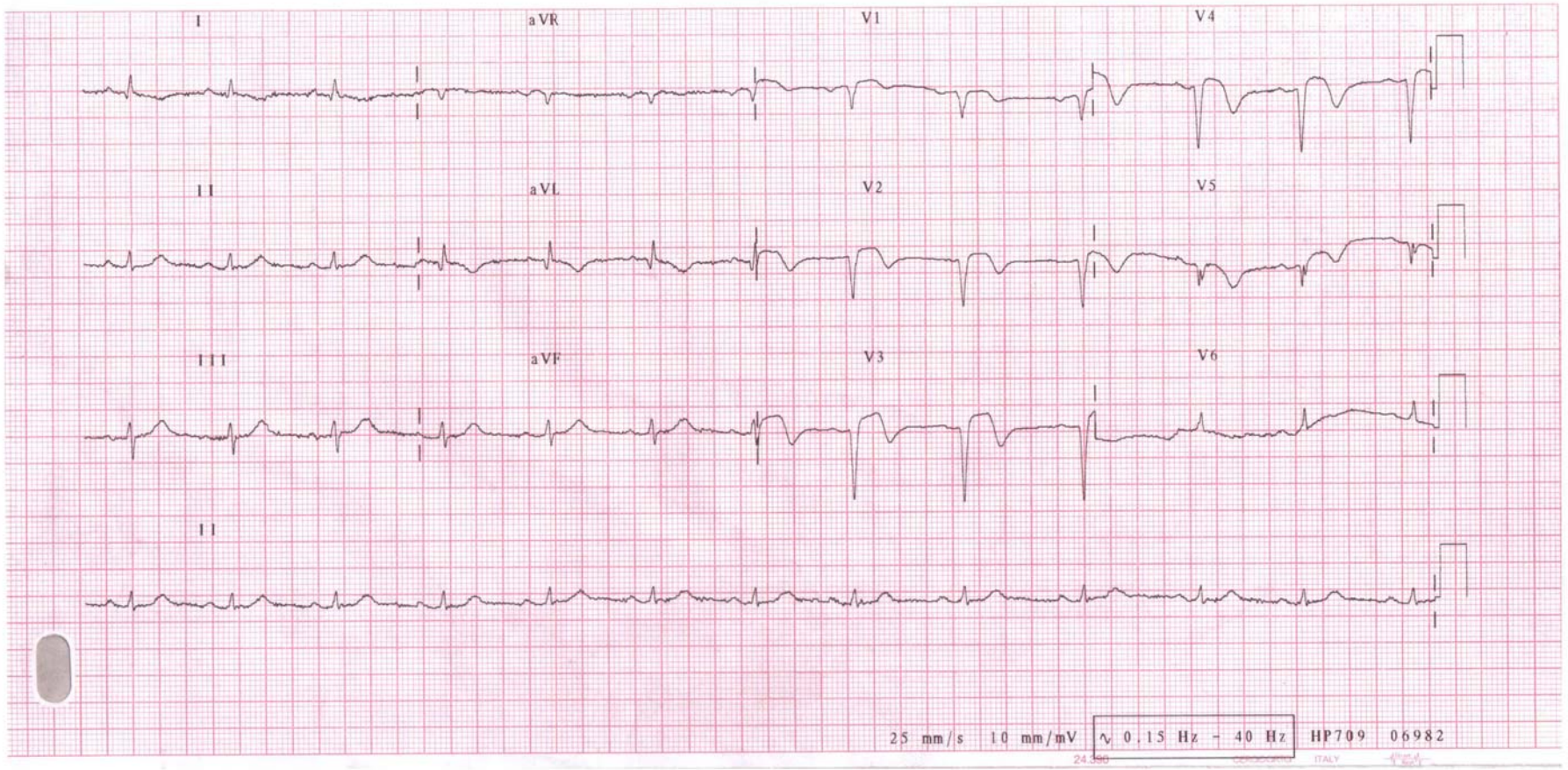
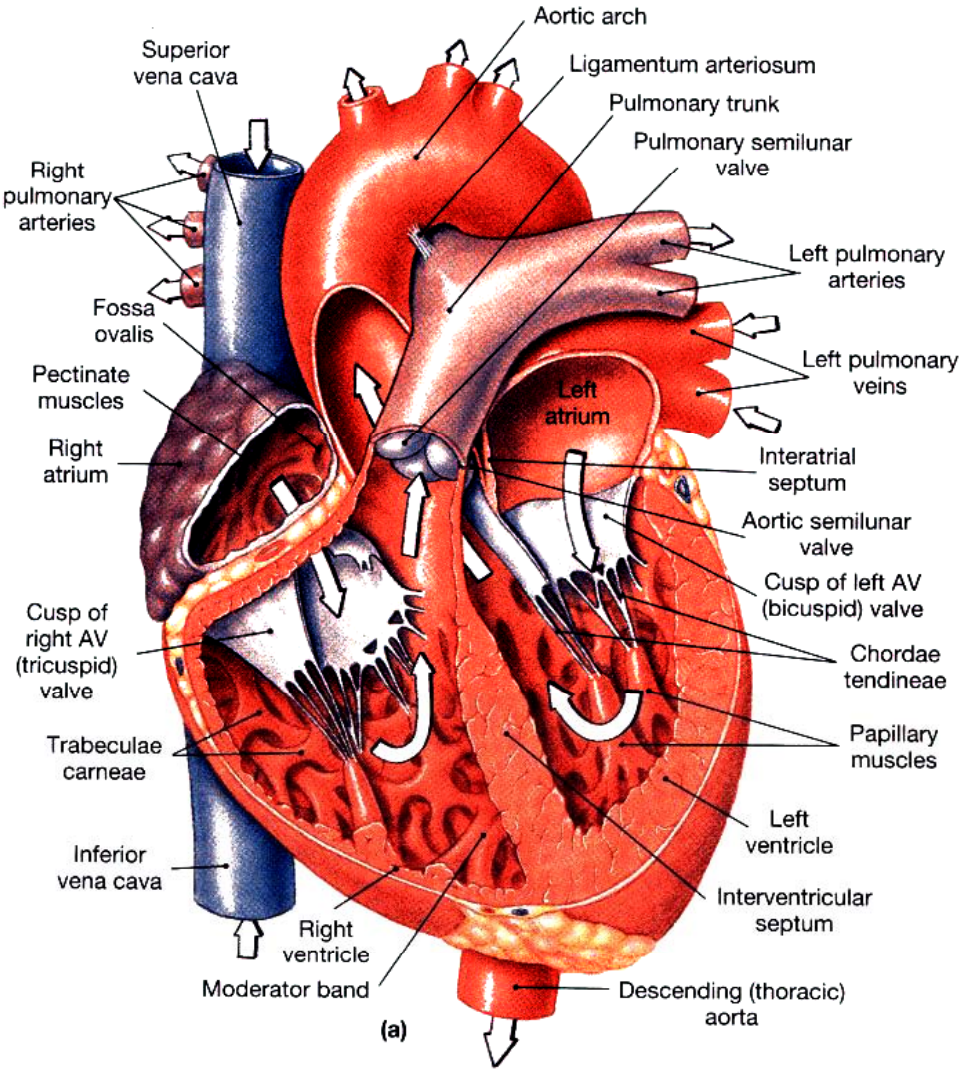
CASE NO : 20



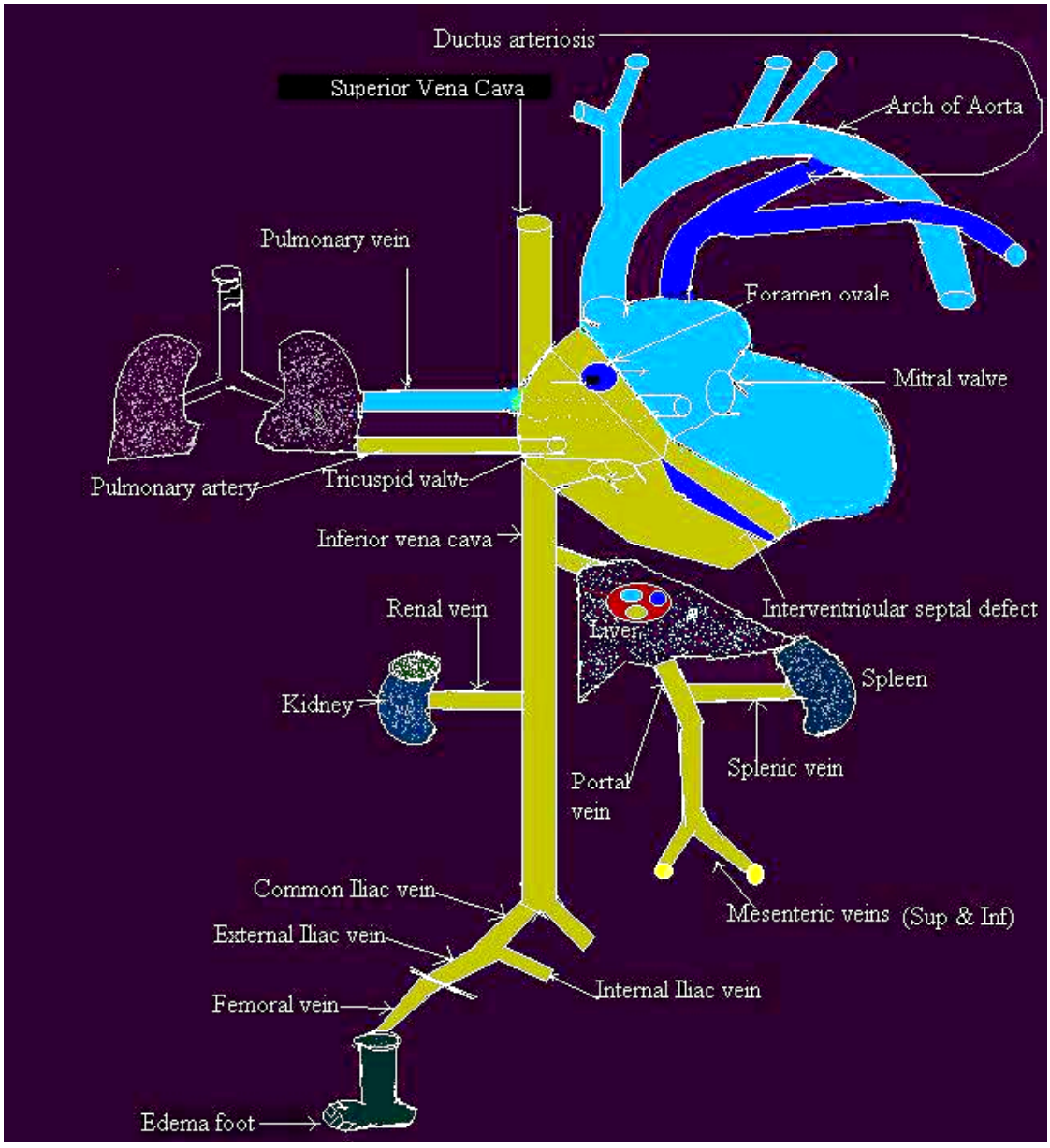
A case of CAHD with

- QS pattern in V_1 - V_5
- Mild ST elevation with primary T inversion in V_1 - V_5
- Normal sinus rhythm
- Recent antero septal infarction

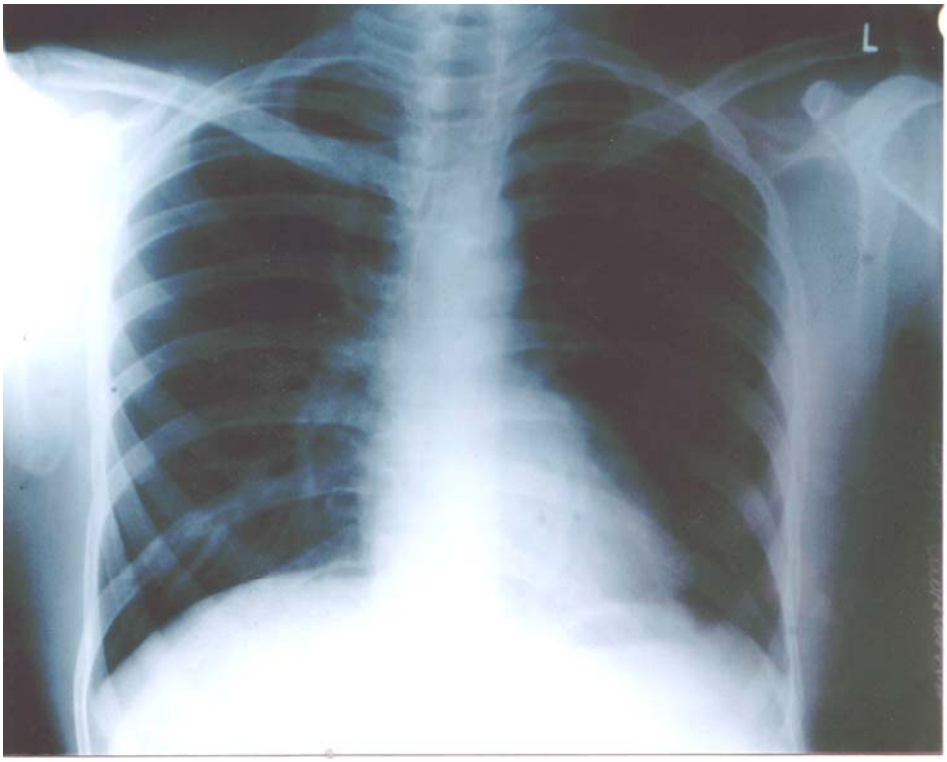
HEART - ANATOMY



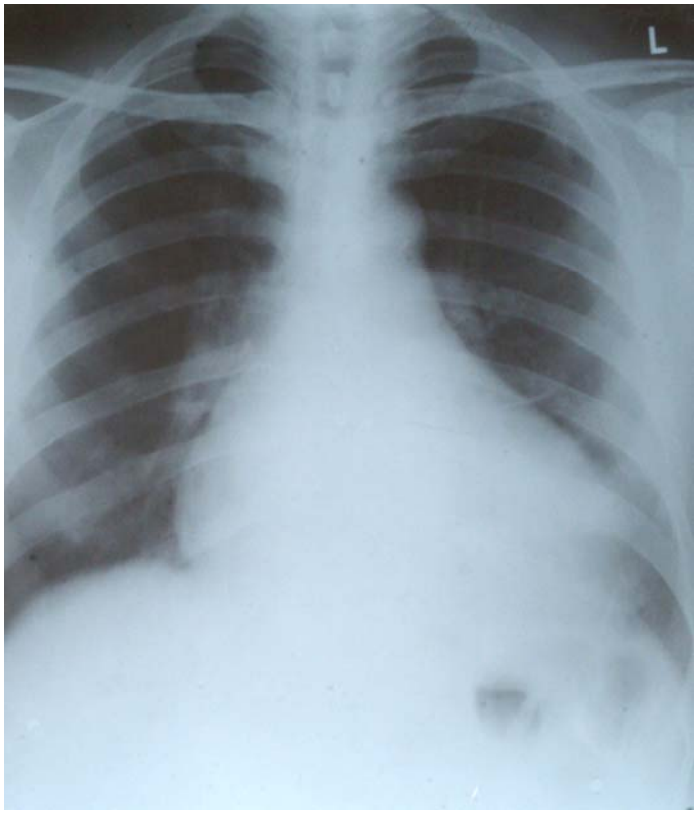
CONGESTIVE HEART FAILURE



CHEST X-RAY PA VIEW- NORMAL



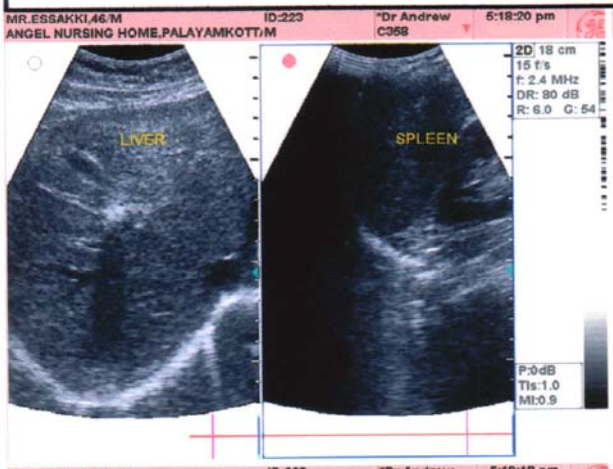
CASE NO : 8 – CARDIOMEGALY WITH PERICARDIAL EFFUSION

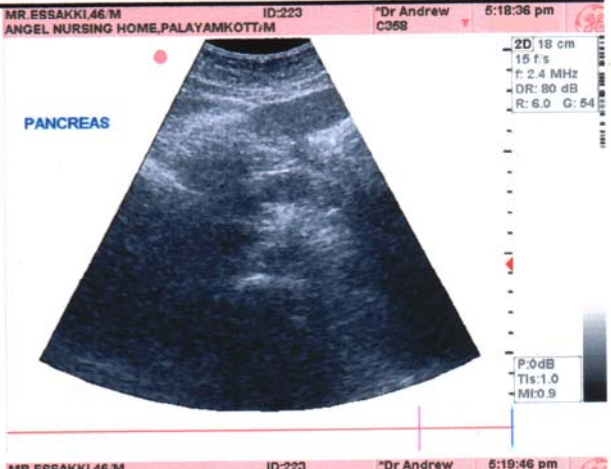


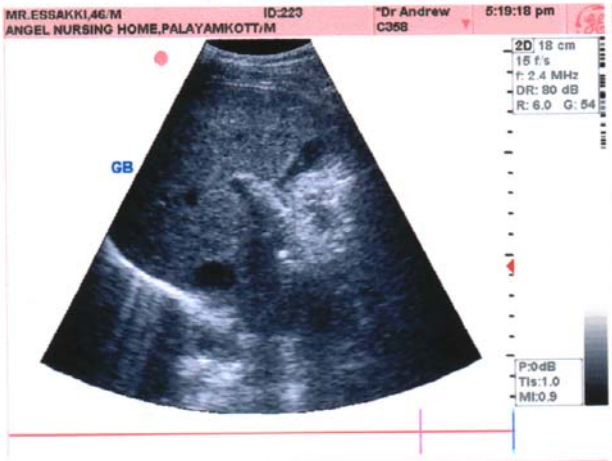
ANGEL NURSING HOME

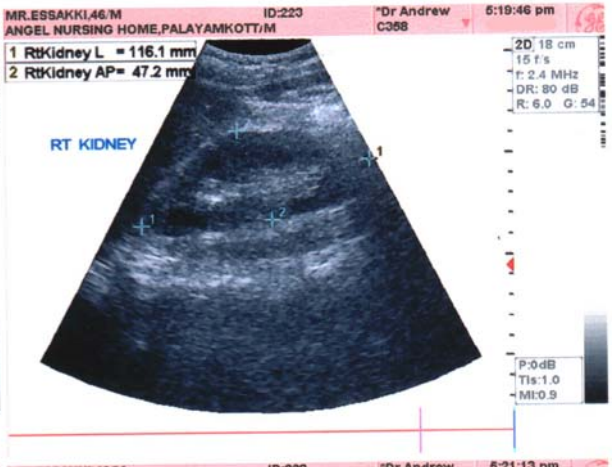
1,Shanthi Nagar, Palayamkottai, Tirunelveli
Tel : 2540783

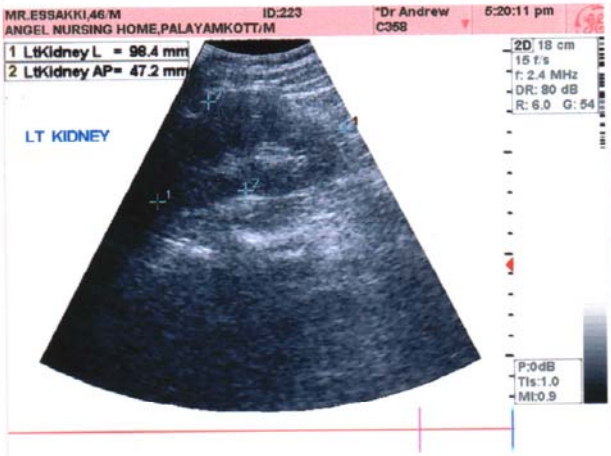
Name: MR.ESSAKKI,46/M
ID: 223 Sex: M Exam Date: 04 Mar 2007
Age: Referring Doctor:

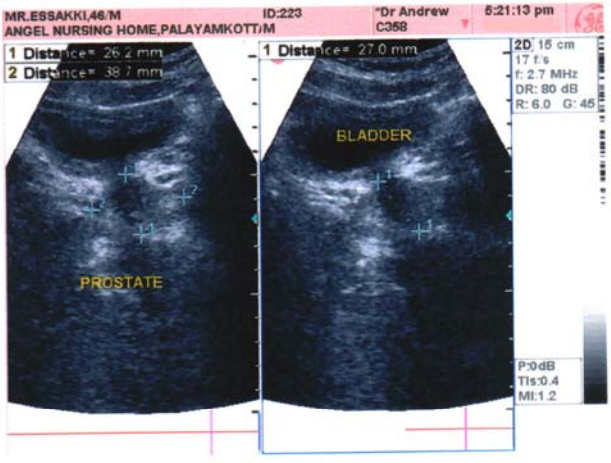












ANGEL HEART CENTRE

ANGEL NURSING HOME
1,Shanthi Nagar, Palayamkottai, Tirunelveli
Tel : 2540783

ANGEL NURSING HOME,PALAYAMKOTTAI

Vascular Report

Patient Information

Name: MR.ESSAKKI,46/M
ID: 223
BP:

Age:
Birth Date:

Exam Date: 04 Mar 2007
Referring M.D.:

2D		Doppler
LtKidney AP	47.2 mm	
LtKidney L	98.4 mm	
RtKidney AP	47.2 mm	
RtKidney L	116.1 mm	

Diagnosis:

Comments:

LIVER APPEARS ENLARGED.IVC & HEPATIC VEINS APPEAR DILATED. CONGESTIVE HEPATOMEGALY.

GALL BLADDER,SPLEEN,PANCREAS,KIDNEYS & BLADDER APPEAR NORMAL.

PROSTATE 3.9 X 2.7 X 2.6 CM.NORMAL.

Referral Reason:

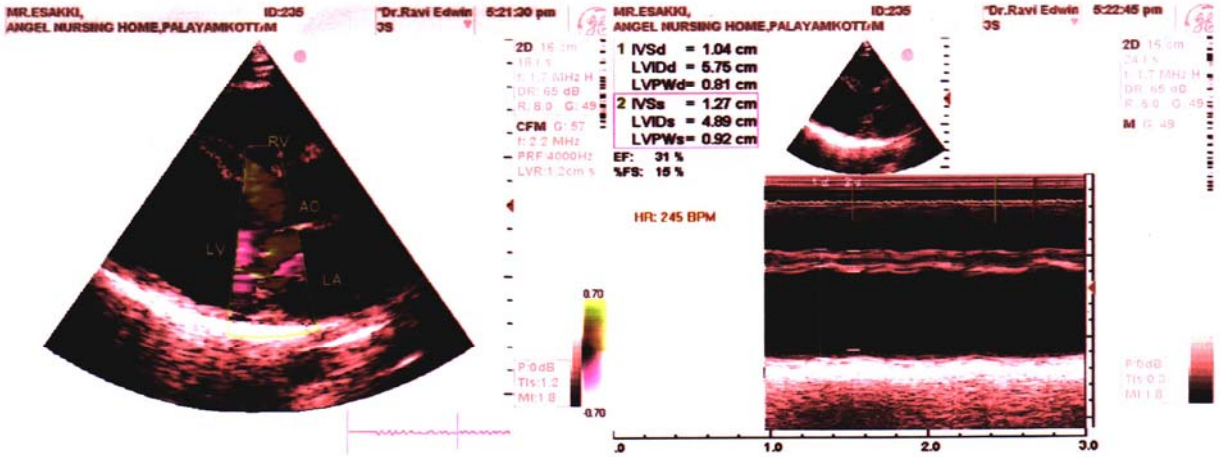
Sonographer:	Physician:

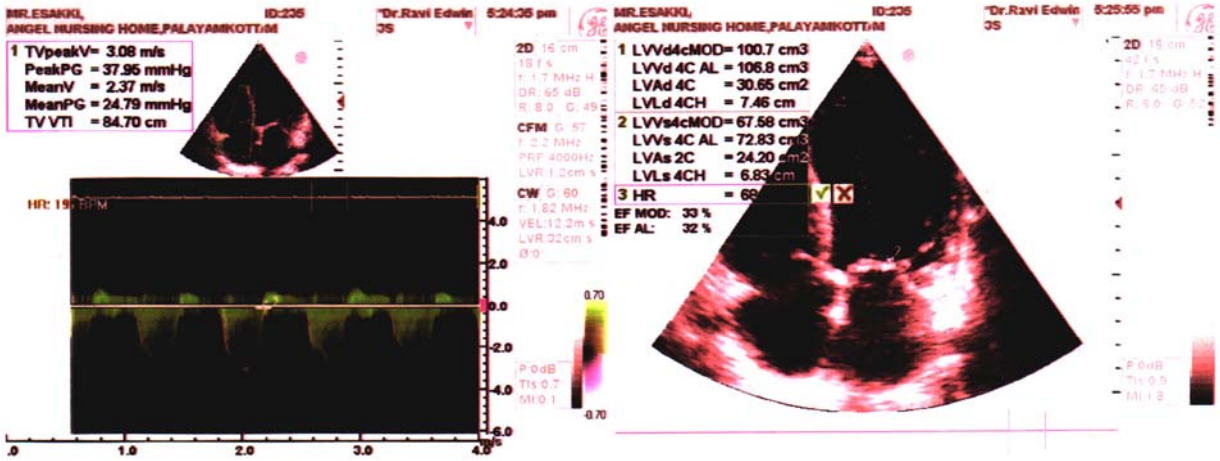
Dr. Andrew Jebakumar,
M.B.B.S., DMRD

ANGEL NURSING HOME

1,Shanthi Nagar, Palayamkottai, Tirunelveli
Tel : 2540783

Name: MR.ESAKKI,46/M
ID: 235 Sex: M Exam Date: 04 Mar 2007
Age: Referring Doctor:





ANGEL HEART CENTRE

ANGEL NURSING HOME

1,Shanthi Nagar, Palayamkottai, Tirunelveli
Tel : 2540783

Echo-Cardiography Report

Patient Name: MR.ESAKKI,46/M
ID: 235
Sex: M
BSA: 0.00 m²

Exam Date: 04 Mar 2007
Referring Doctor: DR.RE
Height:
Weight:

Referral Reason:
DCM - CARDIAC EVALAUTION.

2D- Measurements

LVVd 4C MOD	100.7 cm3	LVAd 4CH	30.65 cm2	LVVd 4C AL	106.8 cm3	EF 4C AL	31.82 %
LVVs 4C MOD	67.58 cm3	LVAs 4CH	24.20 cm2	LVVs 4C AL	72.83 cm3	EF 4C MOD	32.92 %

M- Mode - Others

AVCS	2.70 cm
AoRoot	3.28 cm
LA D	3.80 cm
LA/Ao	1.16

M- Mode - LV Study

IVSd	1.04 cm
IVSs	1.27 cm
LVIDd	5.75 cm
%FS	15.00 %
EF Teich	31.32 %
LVIDs	4.89 cm
LVPWd	0.81 cm
LVPWs	0.92 cm

M- Mode - LV Study

LVVd Teich	163.3 cm3
LVVs Teich	112.2 cm3
SV Teich	51.16 cm3
LVd MassASE	206.3 g
LVs MassASE	197.9 g

Mitral valve Doppler

Aortic valve Doppler

Tricuspid valve Doppler

TVpeakV	3.08 m/s
TVmeanV	2.37 m/s
TVpeakPG	37.95 mmHg
TVmeanPG	24.79 mmHg

Pulmonic valve Doppler

Comments:
ALL CHAMBERRS DILATED.

MITRAL REGURGITATION - MILD.

TRICUSPID REGURGITATION - MILD.

PULMONARY HYPERTENSION - MODERATE.

GLOBAL HYPOKINSIA OF LV.

LV DYSFUNCTION - SEVERE. EF- 31 % (M-MODE)

NO CLOT OR PERICARDIAL EFFUSION.
DILATED CARDIOMYOPATHY - SEVERE LV DYSFUNCTION.

Dr. J.M. RAVICHANDRAN EDWIN
M.D., D.M., (Cardio)

ECG - NORMAL