ASSESSMENT OF
ANNAVAHA SROTAS DUSHTI
IN MOOTRAKRUCCHRA VYADHI

A thesis submitted to

TILAK MAHARASHTRA VIDYAPEETH, PUNE

For the Degree of

DOCTOR OF PHILOSOPHY (Ph. D.)

Subject	**:**	**Rognidan & Vikrutividnyan**
Under Faculty of	**:**	**Ayurved**
Name of Candidate	**:**	**Vaidya Mrs. Jai Kiran Kini**
Under the Guidance of Name of the Guide	**:**	**Dr. Vidya Hirlekar**
Name of the Department	**:**	**Ayurved**
Month & Year	**:**	**October 2015**

DECLARATION

I hereby declare that the thesis entitled "ASSESSMENT OF ANNAVAHA SROTAS DUSHTI IN MOOTRAKRUCCHRA VYADHI" completed and written by me has not previously formed the basis for the award of any degree or other similar title or any other university or examining body.

Place - Pune

Date – 26/10/2015

Vd. Mrs. Jai Kiran Kini

CERTIFICATE

This is to certify that the thesis entitled,

ASSESSMENT OF ANNAVAHA SROTAS DUSHTI IN MOOTRAKRUCCHRA VYADHI" which is being submitted herewith for the award of the Degree of Vidyavachaspati (Ph. D.) in Ayurveda of Tilak Maharashtra Vidyapeeth, Pune is the result of original research work completed by **Vd . Mrs. Jai Kiran Kini** under my supervision and guidance. To the best of my knowledge and belief the work incorporated in this thesis has not formed the basis for the award of any degree or similar title of this or any other University or examining body.

Place - Pune

Date – 26/10/2015

Prof. Dr.Vidya Hirlekar

PH. D. (Ayurveda)

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Date

Dr. Jai Kiran Kini

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I N D E X

CHAPTER

1

INTRODUCTION

Introduction

In present scenario, to control all global diseases, early diagnosis and elimination of frequency increasing etiopathological causative factors of the disease are needed to be studied well. Chronic kidney diseases carry the noticeable importance in the global burden disease report 2010 as well 2013[1].CKD is the 12th cause of death and the 17th cause of disability.

'Preventive nephrology' emphasizes early detection of kidney disease and the institution of measures to slow down its progression. The Indian CKD care program will have a major component of early detection and prevention to be conducted at the primary care level.[2]

As per *ayurveda*, kidney diseases and its symptoms correlate with *vyadhi* of Mootravaha *srotas dushti. (*[3]

In present scenario patients of *Mootravaha srotas Vyadhi* are increasing rapidly. During my course of post graduation study in 2002, I observed many patients suffering from *mootrakrucchra* and various diseases related to Urinary Tract with symptoms like painful micturition, incontinence of urine, dribbling micturition etc. The patients from different categories like age groups, casts, living status, atmosphere & food habits etc. were observed during that period. Amongst them though the symptom 'Dysuria' was common but the associated symptoms were different

Now days in 2012 onwards, the number of patients of *mootravaha srotasdushti* is increasing rapidly. And it is observed that, the frequency of this *srotas dushti* in per patient is also increased and with the awareness of life style management also this particular diseases showing more complicate stage.

An Ayurvedic view behind this disease & the relation with pathological investigation is extremely important for early diagnosis & treatment as well as relapse point of view of the diseases which leads to the CKD which is a major cause of global morbidity and mortality even in developing countries. So dialysis centers are in lacking in the country and demand of kidney for transplant is increasing day by day.

As per Ayurveda, Mootra is one of the drava Mala i.e waste product of the body [4]which remove the excessive Kleda.[5] doing excretion of waste products in liquid form i.e. Dravamala produced at the time of Pachan i.e. digestion.[6]

Sushrutacharya in Sharir Sthan has explained in detail, the formation of Mootra. According to this the formation of the urine starts from Pakwashaya. (7) The two main diseases of Mutravaha Srotas explained by Ayurvedic text are Mutrakrucchra & Mutraghata. Mootrakrucchra vyadhi is considered as the primary disease of mootraghaat which leads to CKD i.e. chronic kidney disease.

While describing them, the ancient scientists did not explain each step of *Samprapti* of this disease in detail regarding, so it is difficult to understand this disease. On another side when we study a disease other than disease of *Mootravaha Srotas,* we certainly get some type of *Dushti* in *Shabda, Rupa, Sparsha, Gandha* which is related to *Mutra*& the well explained view behind that.

As per modern science, the confirmed diagnosis of a particular disease of Urinary Tract is depending mainly upon the laboratory as well as radiological examinations and the signs & symptoms. Classification of types of disease depends upon the parametric result of the clinical examination & laboratory tests.

Our science explained well all diagnostic tools with proper symptoms for early diagnosis. The causative factors also told by classical text. *Ayurveda* focused *hetu vichhar* very well which plays very important role in measuring control for the particular *srotodushti. Ayurveda* explained well the causative factors of *annavaha srotas dushti* which plays important role in etiopathology of mootrakrucchra(8) (Charak. Chikitsasthan 26)

Annavaha srotas is one of the important *srotas* in body described well with *srotodushti hetu, lakshan, moolsthan* in *viman sthan* by *charaka.*The Stressful and busy lifestyle denotes almost all *hetus* of *annavaha srotas dushti* in terms of *annapachan vikruti.*

In present scenario, the food habits, food contents, life style are changing very rapidly which is the maintained causative factor for *annapachan vikruti* and the persistence of the same factor denotes the maintenance of the diseases of the system which denotes the permanent damage of the organ.

In *charak chkitsa sthan* 15, *charaka* predicted *mootraroga* are denoted *annapachan vikruti (specially agnimandya) which* is most common *hetu* in each age groups. Hence, the exact role, pattern and effect of *annavaha srotasdushti* in *mootrakrucchra vyadhi* is the one of the important and socially significant subject for study.

The relation of the disease and the present types of *annavaha srotas dushti* and its study will surly contribute in control program of renal diseases globally.

Therefore the topic selected for the dissertation is,

‘**ASSESMENTOF ANNAVAHASROTAS DUSHTI IN MOOTRAKRUCCHRA VYADHI**

1.*The Global Burden of Disease:Generating Evidence,Guiding Policy.* Seattle, WA: IHME, 2013.Institute for Health Metrics and Evaluation.
*https://www.business.unsw.edu.au/.../**2013-Global**-Burden-of-**Disease**-*
2..Vivekanand Jha[1]Meeting [1]Professor of Nephrology, Postgraduate Institute of Medical Education and Research, Chandigarh, India Current status of end-stage renal disease care in India and Pakistanreport*Kidney International Supplements* (2013) **3,** 157–160; doi:10.1038/kisup.2013.3
3.Sushrut Nidan 9/18-19
4.6.Sharanghadhar Poo.6/6
5.Ashtang Hridaya soo.11/15
7.Sushrut.soo.21/10
8. (Charak. Chikitsasthan 26

CHAPTER

2

AIMS AND OBJECTS

AIM

1. To assess *Annavaha srotas dushti* in *Mootrakruchhra Vyadhi*

OBJECTS

1. To study the *Annavaha srotas* in Detail as per Ayurvedic classical text
2. To study the *Annavaha srotas dushti* and its various types with *Nidanpanchak* as per Ayurvedic classical text
3. To study the annavahasrotas dushti as the diagnostic tool of mootrakrucchra
4. To study the diagnostic tools of mootrakrucchra vyadhi by Ayurvedic as well modern laboratory investigations point of view
5. To study the nidanpanchak of mootrakrucchra vyadhi
6. To study the *Mootrkrucchra Vyadhi* and its causes related to *annavaha srotas dushti to establish the sahacharya in between them*
7. To study the relevant modern text for *Annavaha srotas dushti* and *Mootrakrucchra vyadhi*

CHAPTER

3

MATERIALS AND METHODS

MATERIALS AND METHODS

1. **There will be 2 groups. Each group will have 160 patients**
   The effect of Annavaha srotas dushti in mootrakrucchra vyadhi can be well establish by taking two groups in a study.
   Causative factors of more than one year annavaha srotas dushti and its upadrava should studied well with respect to mootrakrucchra vyadhi in first group
   As well we have to study the mootrakruchhra vyadhi in the patient to check or define the role of annavaha srotas dushti in it in second group.

2. **One of them is the group of the patients with the diagnosis of Annavaha srotas dushti and another is the group of patients of Mootrakrucchra vyadhi**

   We want to establish the correlation and pattern between annavaha srotas dushti and mootrakrucchra vyadhi on the basis of classical text[1]

3. **Selected the patients for Mootrakrucchra vyadhi with well designed a special case record form with the help of literature of Mootrakrucchra Vyadhi described in texts as well the modern criteria based on previous work done**

   Designed a special text proforma for the diagnosis of patients of Mootrakrucchra vyadhi
   Ayurvedic classical criteria for mootrakrucchra vyadhi
   Symptoms-mootradaha, sakashta mootraprarutti basti gaurav, painful,dribbling micturation we will diagnose clinically

   Classical causative factors- excessive vyayaam ,tiksha aushadha, rukshanna, madya, prasanga, aanupmansa sevan, adhyashan, ,ajirna were evaluated.

   As well the modern diagnostic criteria decided on the basis of previous work done on mootrakrucchra [2]

1. Charka chikitsasthan grahani chapter 15, charak chikitsa 26-32
2. Jai Kiran KIni, Ancient Science of LIfe 2012, Vol. 32, Issue 2, (Suppl1):32

Diagnostic criteria of Mootrakrucchra vyadhi on the basis of previous work done-

- Especially difficulty in micturation in terms of heaviness in urogenital and basti region, burning or acute paining at the time of passing of urine is the symptoms. no fever in all types of Mootrakrucchra
- Pathologically no change in serum urea and serum creatine in all types of mootrakrucchra vyadhi
- Painful micturation, toda as symptoms and amorphous material and epithelial cells in microscopy in urine labeled as vataja mootrakrucchra
- Pus cells in urine, specific gravity on higher side, basti gaurav, heaviness at genital area, shofa, in kaphaja mootrakrucchra.
- Calcium oxalate in urine, RBC in microscopy and occult blood in urine chemistry, color specifications in urine with daha labeled as pittaja mootrakrucchra
- Crystals in urine microscopy, very much unbearable radiating pain towards groin and mid suture as well genital area labeled as ashmarija mootrakrucchra.
- All symptoms altogether in sannipataj mootrakrucchra

4. **The blood samples of the patients collected for serological tests** like **serum electrolytes (Na,K,Cl) ,serum creatinine and serum urea & blood tests for CBC with ESR,** the tests will be performed by standard laboratory techniques and related reagents with the help of Nihon Koden for CBC and chem.7 from transasia for serology and Roche's electrolyte analyser. Kept all record of results carefully

5. **Collected the urine sample for the investigations and urine test like microscopic and routine etc. done with the laboratory techniques and related regents.**
   Collected the early morning midstream urine sample of the patients and performed microscopic and routine examinations of urine.

   Made record of results of blood and urine tests after performing them

6. **Designed a special proforma for diagnosis of patients of Annavaha srotas dushti with the help of classical text [(3)]**

   Classical types of annavaha srotas dushti-
   annanabhilasha, arochak, avipaka, amlapitta, chardi,Ajirna
   Agni Pariksha criteria[(4)-]
   A. **Jaranshakti-** Jaranshakti pariksha done by agni parikshan with the help of quantity of food, frequency appetite ,annapachan kaal
   B. **Jirnahaar lakhanani**- Deha laghav, Kshudha, pipasa, Uchit kaal malotsarga and its consistency as well symptoms at the time of malotsarga
   C. **Kshudbodh and its kaal**- frequency of appetite and required time

3.Charak samhita vimaan sthan chapter 5/11-20
4.Charak samhita vimansthan 6/12-13

Hetu of agni dushti-

Ahita bhojan, Atimatra bhojan Akaal bhojan,Pavakasya vaigunyaat

Criteria for diagnosis of Agnimandya[5]

Hetu - Daurbalya, Chinta,Jaagran, Shrama, Avyaam,Aahaar Niyambhanga, Apatarpana and S**ymptoms -** Gaurava, Aalasya, Kshudhamaandya, Shoola, Daurbalya

Criteria for diagnosis of Ajirna [6]

Hetu - Guru,Snighdha,Madhur,Viruddha ,type of food and its quantity and frequency Anna,Adhyashana,Prabhutashana

Symptoms - Gaurav,Aadhmaan,Aatop,Shoola,Trishna

Hetu- Hrallas,Utklesh,Chardi,Jwara,Dravamala Pravrutti

Criteria for diagnosis of Alasaka [7]

Hetu- viruddha Anna,Garavisha,Adhyashana,Vegavidhaaran

Symptoms - Mala and Vata Apravrutti,Aadhmaan,Udgaarnirodh,Shoola,Arati

Symptoms –

Prasek,Amlaudgaar,Tiktaudgaar,Katuudgaar,Urovidaha,Amlika,Shoola,Chardi,Shiras hoola,

Bhrama

5.Charak Samhita chikitasthan 15/42-44, Charak vimansthan 6/12-13, Madhav nidan agnimandya2to4

6. Madhav Nidan agnimandya 5-10,14,ashtanga sangraha sootrasthan 11/47, charak samhita nidan sthan 2/10, charak chikitsa sthan 15/45, satik sushruta sootrasthana 46/502-503, ashtanga sangraha sootrasthan 11/44 indutika, Kashyapasamhita 32-39,madhav nidan page 104

7. charak viman satik 2/14, vagbhat sootrasthan 8/10-14, sushrut uttarsthan 56/78

Criteria for diagnosis of Chardi [8]

Hetu - Atidrava,Asaatmya,Tikshna Sevan,Ashuchi,Mrudbhakshan,Krimi,Ajirna

Symptoms - Hrallas,Prasek,Utklesh,Chhardi,Daha,Trushna,Shoola

Criteria for diagnosis of Amlapitta[9]

Hetu - Katu,Amla,Lavan,Ushna,Abhishyandi,Oily,Viruddha,Vishamasevan,Shile sprout **Symptoms** –

Prasek,Amlaudgaar,Tiktaudgaar,Katuudgaar,Urovidaha,Amlika,Shoola,Chardi,Shirashoola,

Bhrama

Criteria for diagnosis of Shool[10]

Hetu - Viruddha,Vishamaseva,Adhyashana,Sproutes,Ajeerna, Vegavarodha

Symptoms - Aruchi,Malavashtambha,Aaadhmaan,Aatopm,Shoola

Criteria for diagnosis of Grahani [11]

Hetu - vishamashana

Symptoms - Avipaaak,Aarochaka,Aalasya,Chaardi,Daurbalya,Malapravrutti-Dravya and aniyamit,Aantrakoojan,Shoola, Mukhapaak,Trishna,Arati

8.vagbhat Nidansthan5/30, shushrut smhita uttarsthan 49/3-7, Madhav nidan chardi page 153, charak samhita chikitsasthan 20/9

9. Madhav nidan amlapitta , Sharangadhar samhita prathamkhanda ch. 7/103-105, Kashyapa page 335-339

10.sushrut uttarsthan 42 satik /75-85, Madhav Nidan shool /6-16

11.Charak cxhikitsa sthan 15/ 56-62, ashtanga sangraha sharir sthan 5/23, sushrut uttarsthan 40-169, sushrut sharirsthan 4/18, vagbhat sharir sthan 3/51, charak chikitsasthan 28/8, vagbhat sootrastthan 12/8

Criteria for diagnosis of Krimi[12]

Hetu- Snigdha,Madhur,Vishama,Ashuchi sevan

Symptoms - Jwara,Udarvrana,Shotha,Kamala,Gulma,Baddhodar,Vidradhi

Criteria for diagnosis of Arochak[13]

Hetu- angimandya, ajirna, atiguru, atisnighdha, atimadhur, ekrasatmaka aahar, Chinta , shoka , Bhayadi,Impure , pungent food

Symptoms- Loss of taste, pain and burning in heart region, bitter taste, tastelessness,excessive thirsty, vertigo, breathlessness, shortness of breath

7. **Diagnosed the patient of Annavaha srotas dushti and its type in that patient** as well upadrava also noted well as per the refrences in text[4]

8. **The blood samples of the patients collected for serological tests** like **serum electrolytes (Na,K,Cl),serum creatinine and serum urea & blood tests for CBC with ESR,** the tests performed by standard laboratory techniques and related reagents with the help of Nihon kohden for CBC and chem.7 from Transasia for serology and Roche's electrolyte analyser

9. **Collected of the urine sample for the investigations and urine test like microscopic and routine etc. and done with the laboratory techniques and related regents.**
   Collected the early morning and midstream urine sample of the patients and performed microscopic and routine examinations of urine.

10. **Observed the correlation of type of Mootrakrucchra vyadhi and annapachan vikruti clinically as well as pathologically also in the both groups**

    Observed the relationship of these both vyadhi in terms of hetu, prakruti ,pathological investigations of blood and urine

11.Charak chikitsa sthan 15/ 56-62, ashtanga sangraha sharir sthan 5/23, sushrut uttarsthan 40-169, sushrut sharirsthan 4/18, vagbhat sharir sthan 3/51, charak chikitsasthan 28/8, vagbhat sootrastthan 12/8

12.Charak vimansthan satik 7/9, Madhav Nidan krimi 1/7-10,Sushrut uttarsthan 54/3-5, 12-14

13.Sushrut samhita uttarsthan 57/3, bhavprakash madhyam Khanda page 474, Vagbhat Nidansthan 2/17-19

SAMPLE SIZE –

160 patients of each of the both group

160 patients from annavahasrotas dushti more than one year

160 patients of mootrakrucchra vyadhi

Age group-**18-40**

STUDY CENTRE-

1. YMT AYURVEDIC MEDICAL COLLEGE AND HOSPITAL NAVI MUMBAI KHARGHAR

INCLUSION CRITERIA

1. All types of difficult micturation from the age group - 18-40
2. All types of Annavaha srotas dushti patients for more than 1year from the age group -18-40

EXCLUSION CRITERIA

- Hypertension
- Known DM
- Neoplasia of Urinary Tract and Annavaha srotas
- Immunocompramised patients
- Patients of ARF and CRF
- Patients of mootraghaat

STATISTICAL TEST

Chi –square test

Z test

CHAPTER

4

LITERARY REVIEW

Review of Ayurvedic Literature

Human body is made up of three Doshas, seven Dhatus and three Malas. In Samyavstha these Dosha, Dhatu and Mala maintains the health of the body and their imbalance creates various types of disease conditions.

विकारो धातुवैषम्यं साम्यं प्रकृतिरूच्यते ।

सुखसंज्ञकमारोग्यं विकारो दु:खमेव च ।। चरक सूत्रस्थान ९।४

The state of balance of constitutional doshas is called as Aarogya another hand imbalance of Dosha generate state of Roga.

रोगस्तु दोषवैषम्यं दोषसाम्यमरोगता । वाग्भट सूत्रस्थान १।२०

The condition which explains the different stages of formation of Vyadhi e.g. from vitiation of Doshas up to the state of differential diagnosis is called as Vyadhi.

Nidan Panchak

Defination-व्याख्या

निदानं पूर्वरूपाणि रूपाण्युपशयस्तथा ।

सम्प्राप्ति च इति विज्ञानं रोगाणां पंचधा स्मृतम् ।। वाग्भट निदान १

According to Ayurved there are five tools for understanding etiopathology of a disease known as ‘Nidanpanchak’.

These five tools are as follows;

1) Nidan (causative factors)
2) Purvarupa (prodromal symptoms)
3) Rupa (symptoms)
4) Upashaya (prognosis)
5) Samprapti (process of formation of disease or etieopathology)

These five tools are extremely important for diagnosis of a disease. It also helps to understand the stage and details of a disease.

Nidan –निदान

Nidan is one of the most important factor in the development of a disease.

Defintion– व्याख्या

a. निर्दिश्यते दीयते व्याधि अनेन इति निदानम् ।। माधव निदान

The factor which indicates 'Vyadhi' called as 'Nidan'

b.व्याधि उत्पत्ति हेतु निदानम् ।

The factors responsible for development of a disease is called as 'Nidan'.

c. सेति कर्तव्यताको रोगत्पादको हेतु निदानम् । माधन निदान

The factors which forms a disease by generating the stages of Sanchaya, Prakopa, Prasara etc. of Doshas is called as Nidan of that particular disease.
Nimitta, Hetu, Aayatana, Pratyaya, Uthana, Karan are the synonym of Nidan.

Purvarupa - पूर्वरुप

After the stage of Hetu Sevan during the development stage of a disease, vitiated Doshas develops some symptoms known as Purvarupa or prodromal symptoms. As these symptoms are not well developed, it is difficult to judge the type of disease but one can get a rough idea about the development of a disease in the body. For e.g. Bodyache, Yawning, burning sensation of eyes etc. are the prodromal symptoms of Jwara.

Definition – व्याख्या

a.**प्राग्रूपं येन लक्षते। अ.हृ.नि. १-३**

Prodromal symptoms of a Vyadhi developed before the actual symptoms of the specific disease.
The characters by which one can get that the disease is going to be happened in body is called as Purvarupa.
It is the stage at which one cannot make out the Doshavishesh of Vyadhi but it is the stage which shows that disease is going to be present in the body

b. उत्पित्सुरामयस्यासौ दोषविशेष्येणाधिष्ठिता ।
लिङ्गमव्यक्तल्पत्त्वात् व्याधिनां तघथा यथम् । वाग्भट निदान १-४

At this stage symptoms presents in the way that they indicates Vyadhi is going to be happened in the body

But these symptoms are not clearly indicate any ‘Doshavishesh’
At this stage symptoms are not clearly but minorly present in the body.

Rupa -रूप

Definition -

तदेवा व्यक्ततां याति रूपमित्याभिधियते ।

संस्थानं व्यंजनं लिंगं लक्षणं चिन्हम् आकृति ।। अ.ह्र.नि. १–५

After Purvarupa (predominal symptoms) when these all symptoms get clearly expressed in body, those called as ‘Rupa’ of the Vyadhi

Sansthan , Vyanjan, Linga, Lakshan, Chinham,Aakruti are the synonyms of ‘Rupa’

If all Rupas of a disease mentioned in texts appears in the body then it is consider that the disease in that body is not curable.

Samprapti of Vyadhi -संप्राप्ति

व्याख्या –

Samprapti i.e the etiopathology of a Vyadhi. Samprapti explains the step by step development of a disease, which is useful from the point of view of treatment.

संप्राप्ति –

यथा दुष्टेन दोषेण यथा चानुविसर्पिता ।

निवृत्तिरामयस्यासौ संप्राप्रिर्जातिरागति: ।। वाग्भट निदान १-८

The evolution of a disease due to vitiation of Doshas, which are continuously circulating in the body is known of the body is known as Samprapti of that particular disease, also called as Jati and Aagati.

Types of Samprapti

संख्याविकल्पप्राधान्य बलकालविशेषता । वा. नि. १–९

There are five types of Sampraptis mentioned in Ayurvedic literature viz. Sankhya, Vikalpa, Pradhanya, Bala & Kala.
Aacharya Charaka has mentioned one more type i.e. Vidhi Samprapti.

१. संख्या सम्प्राप्ति –

When the Samprapti gets explained with the help of Sankhya is called Sankhya Samprapti. e.g. Ashta Jwara – i.e eight types of Jwara.
This type of Samprapti counting the varieties of each disease in number.

2. Vikalpa Samprapti - विकल्प सम्प्राप्ति –

दोषाणा समवेताना विकल्पोऽशांशकल्पना । वा. नि. १–१०

Vitiated Doshas comes together & forms Vyadhi then the Anshnsha kalpana of the Doshas makes the type called Vikalpa Samprapti. This indicates the different aspect of vitiated Doshas involved in formation of a Vyadhi.

3. Pradhanya Samprapti– प्रधान सम्प्राप्ति–

स्वातंत्र्यपारतत्र्याभ्यां व्याधे: प्राधान्यमादिशेत । वा. नि. १–१०

One can determine the type by dependence of disease. If disease is independent then the Samprapti is called as Pradhan Samprapti

4. Balabal of Samprapti - बल सम्प्राप्ति –

हेत्वादिकात्स्न्नावयवैर्बलाबलविशेषणम् । वा. नि. १–११

The presence of Hetu, Purvarupa, Rupa with all characters shows balabal i.e severity of Samprapti. It is based on the nature of the cause.

5.Kala Samprapti- काल सम्प्राप्ति–

नक्तदिनर्तुभुक्तांशैर्व्याधिकालो यथामलम् । वा. नि. १–११

According to diurnal & dirhythmic variations type of Samprapti also gets changes. It signifies the time during which the disease and Dosha.

Upashaya –

व्याख्या –

१. गूढलिंगम् व्याधिम् उपशयानुपशयाभ्याम् परिक्षेत् । माधव निदान

It is useful to diagnosed the Vyadhi Avasthat i.e. the symptoms of a disease which is not fully expressed in the body but actively present during etiopathology is called as Gudhalinga.

To examine the Gudhalinga of a disease one can use the management of Upashaya. According to Charaka the disease whose symptoms are not well expressed can be examined by Upashaya or Anupashaya pariksha

२. हेतुव्याधिविपर्यस्तविपर्यस्तार्थकारिणाम् ।
औषधान्न विहाराणामुपयोगं सुखावहम् ।।
विध्यादुपशयाम व्याधे स हि सात्म्यमिति स्मृतम् ।
विपरितोऽनुपशयो व्याध्यसात्म्यमिति स्मृतम् ।। वा. नि. १–६,७

Administration of either medicine, food or activity which is Viparita i.e. opposite to the causes of the disease or both and do not actually but of identical nature i.e. Viparitharthakari – yet produces the effect of the opposite that giving comfort to the patient is called as Upashaya.

It is also called as Satmya (suitable for the disease and the patient).

Mutrakrucchra Vyadhi - मूत्रकृच्छ्र व्याधि

Definition -

a मूत्रयतीह कृच्छ्रात् । च.चि. २६-३३

As per Aacharya Chrarak the Vyadhi in which the patient gets painful and difficulty in micturition is is called as Mutrakrucchra Vyadhi.

b.मूत्रकृच्छ्रे कृच्छ्रत्वमतिशयतिम् ईषद्विभन्दः । मधुकोष टीका

In this Vyadhi pain is more than obstruction in this Vyadhi. Obstruction is also there but the severity of pain is more at the time of urination.

c.मूत्रकृच्छ्रे मूत्रं कृच्छ्रेण वहति। चक्रदत्त

As per Chakradatta in this Vyadhi urination process becomes difficult and painful.

Nidan of Mutrakrucchra Vyadhi- निदान –

व्यायामतीक्ष्णौषधरूक्षमघप्रसंनित्यद्रुतपृष्ठ्यानात् ।

आनुपमांसाध्यशनाद्जीर्णात्स्युर्मूत्रकृच्छ्राणिनृणाम् तथाऽष्टौ ।। च.चि. २६–३२

Hetus (Causative factors) of Mutrakrucchra Vyadhi as mentioned by Charaka -

1. Vyayam-
   Excessive exercise
2. Tikshna Aushadha-
   Consumption of strong medicines frequently or continuously
3. Rukshanna-
   Intake of excessive, dry or nonunctious food
4. Madya-
   Excessive intake of liquors for a longer duration
5. Prasanga-
   Excessive sexual indulgence, which is also considered as ‘Sahas’
6. Nityadrushtaprushtayanat-
   Regular journey by vehicles
7. Aanupmansat-
   Regular and heavy intake of aquatic food
8. Adhyashanat-
   Frequent & excessive eating before previous digestion
9. Ajirna-
   Repeatedly indigestion and irregular dietary habits

These are the factors, responsible for the generation of Mutrakrucchra Vyadhi.

As per Aacharya Charak

कषायतिक्तोषणारूक्षभोज्यौ संधारणाभोजनमैथुनेश्च । च. चि .२६/५

1) Kashaya, Katu, Tikta Anna -

Repeatedly excessive intake of Kashaya, Katu, Tikta Rasatmaka food Charakacharya has mentioned the above three extremely important causative factors responsible for the generation of Mutrakrucchra.

2) Vegavidharan -

Natural urges are called as Vega. The various Vega in body generated by normal movement of Vayu . Suppression of natural urges is called as Vegavidharan.

These both factors vitiate the normal Vata in body & disturb the normal function of Vata.

Swabhava (Tendency)- स्वभाव –

दारूण, चिरकारी

It is chronic disease which needs a long term treatment as it gives severe discomfort & pain

Marg - मार्ग –

मध्याम

It develops in Koshtha as well as Dhatu like Rakta , Mansa, & Meda hence called as Madhyam Margaja Vyadhi.

As per the urine formation process mentioned by Sushrutacharya the distribution of waste material developed during the process of Pachan is started from the Koshtha, which is a Madhyam Rogamarga.

Purvarupa of Mutrakruchhra Vyadhi

1. Heaviness in lower abdomen-

As the vitiated 'Kapha' gets accumulated in Srotas at lower abdomen, the symptom heaviness rises

2. Dribbing urination

As vitiated Doshas gets accumulated in lower abdomen, the normal direction & motion of urine gets disturb & the drubbing urination present

3. Paining at inguinal region

As vitiated Vata gets accumulated in lower abdomen and creates pain at inguinal region

4. Paining at basti region

As Aapan Vayu gets vitiated in lower abdomen, it gives pain at Basti region

5. Paining at umbilicus

Vata at lower abdomen gets vitiated & turns at upwards direction gives paining at umbilicus region

6. Paining at lower abdominal region at the time of micturition

As excretion is one of very important function of Vata & when Vata gets vitiated, it disturbs micturition & makes it painful

As the place of Aapan is Basti (urinary bladder), Pakwashaya (large intestine), Vankshan (inguinal region), Uru (thigh region), & Kukshi

When these Aapan gets vitiated, its generates throbbing pain in these regions at the time of micturition

Rupa of Mutrakruchhra Vyadhi- Rupa-रूप –

१. बस्तिवंक्षणमेढ्रार्तियुक्तोऽल्पाल्पं मुहुर्मुहुः ।

मूत्रयेत् वातजे कृच्छ्रे पैत्ते पीतं सदाहरूक् ।।च.चि. ३६

रक्तं वा कफजे बस्तिमेढ्रगौरवशोफवान ।

सपिच्छं सविबन्धं च सर्वैः सर्वात्मकं मलैः ।। अ.ह्.नि. ४–५

In Mutrakrucchra caused by Vata, the obstruction is more than any other type. In Pittaja, urine is yellow and accompanied with burning sensation and haematuria.

A person suffering from Kaphaja Mutrakrucchra has heaviness and oedema at the region of bladder and penis and voids urine which is slimy and disrupted.

When all Doshaj gets vitiated at a time or symptoms above mentioned appears together in the body.

The Sannipataja type is critical by condition and treatment point of view. Hence it is difficult and severe amongst the other types of Mutrakrucchra.

रूप –

२. तीव्रा रूजो वंक्षणबस्तिमेढ्रे स्वल्पं मुहुर्मूत्रयतीह वातात् ।

पीतं सरक्तं सरूजं सदाहं कृच्छ्रान्मुहुर्मूत्रयतीह पित्तात् ।।

बस्तेः सलिङ्गस्य गुरूत्वशोथौ मूत्रं सपिच्छं कफमूत्रकृच्छ्रे।

सर्वाणि रूपाणि तु सन्निपाताद्भवन्ति तत् कृच्छ्रतमं हि कृच्छ्रम् ।। च.चि.३४,३५

Most of the Aacharya have explained Rupa as per type of VyadhiGenerally there are 8 types of Vyadhi

1. Vataja
2. Pittaja
3. Kaphaja
4. Sannipataj
5. Ashamarija
6. Purishaja
7. Kshataj
8. Kshayaja

वातेन पित्तेन कफेन सर्वै: यथाऽभिधानै: शकृदश्मरीभ्याम् ।

तथाऽपर: शर्करया सुकष्टे मूत्रोपघात: कथितोऽष्ठम स्यु: ।। सु.उ. ५९-३

1) Madhavnidankar has included Sharkaraja mutrakrucchra in Ashmarija Murakrucchra.
2) Aacharya Sushruta has mentioned Sharkaraja Mutrakrucchra as an independent type
3) Charaka has not descrivbed Purishaja Mutrakrucchra

Acharya Charak has explained four types clearly

1. Vataj Mootrakruchhra

Due to vitiated Vata, severe throbbing pain (Tivra Ruja) generates at the region of urinary bladder (Basti), lower abdomen, penile (Medhre) & inguinal region (Vankshan)

These also causes slow urination in less quantity (dribbling micturition)

2. Pittaja Mutrakruchhra

Due to accumulation of vitiated Pitta, urination gets yellowish (Pittam), painful (Sarujam), with bleeding (Saraktam) ,with irritation (Sadaha),as well as dysuria (Kruchhrata) also present

3. Kaphaja Mutrakruchhra

Due to accumulation of vitiated Kapha, heaviness (Gurutva) & odema (Shotha) presents at the Inguinal region, penis & by that urine gets sticky in consistency(Sapiccha)

4. Sannipatik Mutrakruchhra

In Sannipatik Mutrakruchhra, all above mentioned symptoms presents simultaneously & with these all symptoms, this type of Mutrakruchhra gets more chronic & more difficult to get cure

Aacharya Sushruta has also mentioned the above types but there are some variations in the symptoms of these types.

The variations of Sushrutacharya other than Charakacharya as as follows;

Vataja –

During severe pain patients squeezes his scrotum, penis and inguinal region at the time of urination.

Kapahaja –

In this type of Mutrakrucchra there is a specific symptom is mentioned by Aacharya i.e. Horripilation at the time of urination.

Sannipataja -

In Sannipataja type of Mutrakrucchra, urine shows various shades of colours.

Purishaja Mutrakrucchra- पुरीषज –

शकृतस्तु प्रतीघाताद्वायुर्विगुणतां गत: ।

आध्मानं वात च मूत्रसङ्गं करोति च ।। सु.उ. ५९

Aacharya Shushruta has been described the above mentioned type of Mutrakruchhra. By frequent suppression of natural urge for bowel, Vayu at that place gets vitiated & moves in opposite than normal direction i.e. upward direction.

It creates obstruction in urination process, retention of gases & gives severe pain in Basti region it gives severe constipation also

Shukraja Mutrakrucchra- शुक्रज मूत्रकृच्छ्र –

१. शक्रं मलाश्चैव पृथक् पृथग्वा ।
मूत्राशयस्था: प्रतिवारयन्ति ।।
तदव्याहतं मेहनबस्तिशूलं ।
मूत्रं सशुक्रं कुरूते विबद्धम् ।।
स्तब्धश्च शूनो भृशवेदनाश्च ।
तुघते बस्तिर्वृषणौ चतस्या ।। च.चि. २६–४२

According Aacharya Charak, Shukra i.e. semen & Mala i.e. stool separately obstructs the urinary bladder. That arises frequent radiating pain, in lower abdomen & penis & urine with semen obstructs that path so semen also doesn't come easily.

२. रेतोऽभिघाताभिहतस्यपुंस प्रवर्तते यस्य तु मूत्रकृच्छ्रम् ।
स्याद्वेदना वंक्षबस्तिमेढ्रे तस्यातिशूलम वृषणातिवृत्ते ।।
शुक्रेण सरूध्द्गतिप्रवाहो मूत्रं स कृच्छ्रेण विमुञ्चतीह ।
तमङ्गयो: स्तब्धमिति ब्रुवन्ति रेतोभिघातात प्रवदंति कृच्छम् ।। च.चि. २६–४०,४१

In this type of Mutrakrucchra, the infected Shukra obstructs the urinary & seminal path,so he gets severe unbearable pain at inguinal region,Basti region, scrotal as well as penile region.
Infected Shukra obstructs urinary tract & urination process makes painful & generates dysuria

३. शुक्रो दोषैरुपहते मूत्रमार्गे विधारिते ।
३. सशुक्रं मूत्रयेत् कृच्छ्राद्वस्ति – मेहनशूलवान् ।। सु.उ. ५९

By vitiated Vata etc. Dosha, Shukra gets infected & obstructs urinary path, disturbs urination process and patient gets very severe pain at Basti region at the time of seminisation as well as urination.

Kshataja - क्षतज –

१. क्षताभिघातात् क्षतजं क्षयाद्वा ।
प्रकोपितं बस्तिगतं विबद्धम् ।।
तीव्रार्ति मूत्रेण सहाश्मरीत्व–मयातिस्मिन्नतिसंचिते च ।
आध्माततां विन्दति गौरवं च ।
बस्तेर्लघुत्वं च विनि:सृतेऽस्मिन् ।। च.चि. २६–४३,४४

When a urinary tract gets injured by external or intrinsic factor, internal haemorrhage or stricture takes place. Bleeding due to Kshata i.e. injury and Shukrakshaya by excessive sexual activity Vayu gets vitiated and obstruct at Basti region.
In such circumstances retention of urine & Vayu takes place along with heaviness at Basti region.

२. मूत्रवाहिषु शल्येन क्षतेष्वभिहतेषु वा ।
मूत्रकृच्छ्रम् तदाघाताज्जायते भृशदारूणम् ।। सु.उ.५९/८–१०

Kshataja Mutrakrucchra can arise by injury to urinary path by external factor or accidental injury to urinary path.
This type of Mutrakrucchra is very much painful & difficult to manage.

१. क्षोभात् क्षते मूत्रयतीह स्त्रासृक् तस्या: सुखम् मेहति च व्यपायात्।

अश्मरीज मूत्रकृच्छ्र –

By injury, the urinary path gets irritation; haematuria as well as dysuria are presents.

Ashmarija Mutrakrucchra - अश्मरीज मूत्रकृच्छ्र –

१. विशोषयेद्बस्तिगतं सशुक्रं मूत्रं सपित्तं पवन: कफं वा ।
यदा तदाऽश्मर्युपजायते तु क्रमेण पित्तेष्विव रोचना गो: ।। च.चि. २६–३६

According Charak Chikitsa Sthan,
When by vitiated Vata, Mutra in bladder gets absorbed & gets dry with semen (Shukra), Pitta or Kapha, then they generates Ashmari which seems like Gorochan

This way Aacharya Charak has been explained the formation of ashmari with well Samprapti. He had also explained the types & characters of Ashmari

२. कदम्बपुष्पाकृतिरश्मतुल्या: श्लक्ष्णा त्रिपुट्यप्यथवाऽपि मृद्वी ।
मूत्रस्य चेन्मार्गमुपैति रूद्धा मूत्रं रूजं तस्य करोति बस्तौ ।।
ससेवनीमेहनबस्तिशूलं विशीर्णधारं च करोति मूत्रम् ।
मृद्नाति मेढ्रं स तु वेदनार्तो मुहु: शकृन्मुञ्चति मेहने च ।। च.चि. २६–३७,३८

In vataja type Ashmari seems like Kadamba Pushpam, some are triangular in shape.
In Pittaja type they are round & slippery.
These Ashmari comes in urinary tract & obstructs the urinary path & generates pain at Basti region & pain at the time micturation
In this case one gets severe throbbing pain in inguinal region, penis, raphae urinary bladder & it makes urination in more than one stream (Vishirnadharam Mutrapravritti) and patient squeezes his penis by severe pain at the time of urination.
When Ashmari moves forward in urinary system, it gives severe pain while moving so bowel movements also gets painful

वातपित्तकफैस्तिस्त्रचतुर्थी शुक्रजाऽपरा ।

प्राय: श्लेष्माश्रया: सर्वा अश्मर्य: स्युर्यमोपमा ।। च.चि. २६/३६

Vata, Pitta, Kapha & Shukrashmari are the 4 types of Ashmari.
Generally in these all types Ashmari gets formed by Kapha Dosha as their receptacle these all types of stones are giving severe and verities of pains.

अश्मरीरोग– पूर्वरुप

बस्त्याध्मानं तदासन्नदेशेषु परितोऽतिरुक् ।

मूत्रे च बस्तगंधत्वं मूत्रकृच्छ्रं ज्वरोऽरूचि: ।। अ.हृ. ९/८

Prodromal symptoms of Ashmari are -
Retention of gases at Basti which generates distention of abdomen & surrounded region, severe pain at the Basti & surrounded area, loss of taste, fever & the smell of urine get change as the smell of goat urine as well as difficulty of passing urine.

Ashmarija Mutrakrucchra(अश्मरीज) मूत्रकृच्छ्र –

यदा वायुर्मुखं बस्तेरावृत्य परिशोषयेत् ।

मूत्रं सपित्तं सकफं सशुक्रं वा तदा क्रमात् ।।

सञ्जायतेऽश्मरी घोरा पिताद्गोरिव रोचना ।

श्लेष्माश्रया च सर्वा स्यात् ..... ।। अ.हृ.नि. ९–६,७

According Astang Hridaya Nidan Sthan,
When the normal Snigdhattva of Kapha Dosha at the opening of the Basti gets absorbed or dry by surrounded vitiated Vata as well as urine infected by Pitta, Kapha or by Shukra respectively, then painful Ashmari gets gets formed by Vikrut Pitta Dosha & these all Ashmaries having Kapha as their receptacle.

रूप –

सामान्यलिङ्गंरूङ्गनाभिसेवनिबस्तिमूर्ध्वसु ।

विशीर्णधारं मूत्रं स्यात्तथा मार्गनिरोधने ।।

तध्यपायात्सुखं मेहेदच्छं गोमेदकोपमम् ।

तत्सक्षोभात्क्षते सास्त्रमायासाच्चतिरूग्भवेत् । अ.हृ.नि. ९–१०

Symptoms of Ashmarija Mutrakrucchra according Ashtanga Hridaya

1) Upwards pricking paining at umbilical or para-umbilical region, suture, lower abdominal region (basti Pradesh).

2) As the path of urine & stool gets obstruct by vitiated Doshas, the stream of urination gets disturb & so urine comes out in multiple streams.

3) When the path gets free clear urine comes out which seems like Gomedaka gem (dolomite in colour). The viscosity of urine gets changes & due to vitiated Kapha Dosha urine gets Gomutra Varna

4) Due to vitiated Doshas urinary tract gets irritation & bleeding also gets present & these urination process gets more difficult & extremely painful

After the common symptoms, we will discuss all types of Ashmaries viz.

1. Vataj ashmari
2. Pittaj ashmari
3. Kaphaja Ashmari
4. Shukrashmari

Vataj Ashmari– वातज अश्मरी –

तत्र वातादृशं चार्तो दन्तान् खादति वेधति ।

मृद्धाति मेहनं नाभिं पीडयत्यनिशं क्वणन् ।।

सानिलमं मुञ्चति शकृन्मुहुर्महति बिन्दुश: ।

श्यावारूक्षाऽश्मरी चास्य स्याच्चिता कण्टकैरिव ।। अ.हृ.नि. ९–११,१२

Vataj Ashmari is very painful then any other type of Ashmari.

Due to vitiated Vata whole lower abdomen gets severe pain so that person grinds his teeth, shivers, squeezes the penis, rubs umbilicus, cries with pain, voids fecal with flatus, urinates too frequently and in drops.

The stone is black in colour, rough and studded with thorny projections.

Pittaja Ashmari – पित्तज अश्मरी –

पित्तेन दह्यते बस्ति: पच्यमान इवोष्मवान् ।

भल्लातकास्थिसंस्थानाम् रक्तापीताऽसिताश्मरी ।। अ.हृ.नि. ९–१३

By these all phenomena Ashmari gets generated which can be like seeds of Bhallatak, red or yellow coloured and seems like Sita (Khadisakhar)

Kaphaja Ashmariकफज अश्मरी –

बस्तिनिस्त्युघत इव श्लेष्मणा शीतलो गुरू: ।

अश्मरी महती श्लक्ष्णा मधुवर्णाऽथवा सिता ।। अ.हृ.नि.

In this type of Ashmari, Basti gets pricking sensation as well as one can get heaviness at Basti region & coolness at that place

The Ashmari of Kaphaja type is cool (Sheetal), Slakshana, Mahati (Big sized), Honey or sugar coloured

Shukrashmari – शुक्राश्मरी –

शुक्राश्मरी तु महतां जायते शुक्रधारणात् ।

स्थानाच्च्युतं युक्तं हि मुष्कयोरन्तरेऽनिल: ।।

शोषयत्युपसंग्रह शुक्रं तच्छुष्कमश्मरी ।

बस्तिरूक्कृच्छ्रमूत्रत्वमुष्कश्वयथुकारिणी ।।

तस्यामुत्पन्नमात्रायां शुक्रमेति विलीयते ।

पीडिते त्ववकाशेऽस्मिन् ..... ।। अ.हृ.नि. ९–१६,१७

Seminal stone gets formed in adults due to suppression of the natural urge of semination. The semen gets dried by vitiated Vata inside the scrotum and forms Shukrashmari. This generated pain in bladder, dysuria and the swelling at the scrotal region.

Sharkaraj Mutrakrucchra– शर्कराज मूत्रकृच्छ्र –

अश्मरी शर्करा चैव तुल्यसंभवलक्षणे ।

विशेषणं शर्कराया: शृणु किर्तयतो मम् ।।

पच्यमानाऽश्मरी पित्ताच्छोश्चमाणा च वायुना ।

विमुक्त कपसंधाना क्षरन्ति शर्करा मता ।।

हृत्पीडा वेपथु: शूलं कुक्षीविक्षश्च दुर्बल: ।

तथा भवति मूर्च्छा च मूत्रकृच्छ्रं च दारूणम् ।।

मूत्रवेगनिरस्ताभि: प्रशमं याति वेदना ।

यावदस्या: पुनर्नेति गुडिका स्त्रोतसो मुखम् ।। मा.नि. मूत्रकृच्छ्र

While describing about Sharkraja Mutrakrucchra , Aacharya Madhava says, the symptoms of Ashmari & Sharkara are same.

By vitiated Vayu & heat of vitiated Pitta , Ashmari gets ripened & Kapha also gets absorbed & Ashmari turns into Sharkara

This type of Mutrakruchhra is very painful. In this type one gets yokning, pain at heart region, pricking sensation, weakness & paining in lower abdomen.This also gives vertigo. So patient feels excessive weakness & he may get faint

When Sharkara gets displaced from urinary path, the pain gets relived & urine comes out easily

श्लेष्मणोवयवाभिन्ना शर्करा इति संज्ञिता । सु.उ. ५९–१२

According Aacharya Shushruta, Sharkara is mainly made by Shleshma i.e. vitiated Kapha in the body.

शर्करा निर्माण –

१. अणुशो वायुना भिन्ना सा तस्मिन्ननुलोमगे।

निरेति सह मूत्रेण प्रतिलोमे निरुध्यते।। आ.ह्र.नि.९/१९

When Ashmari gets fully dry by vitiated Vata Dosha,it turns into Sharkara
When Vayu acts downwards it comes out of urinary tract with the help of that vitiated Vayu, otherwise it lies inside of the body when vitiated Vata stops inside of body

२. एषाश्मरी मारूतभिन्न मूर्ति स्याच्छकरा मूत्रपथान क्षरंति।

When Ashmari gets spiltened by vitiated Vata, Sharkara gets formed which scrubs the urinary tract

शर्करा उपद्रव–

मूत्रस्रोत प्रवृत्ता सा सक्ता कुर्यादुपद्रवान्।

दौबल्याम् सदनम् काश्र्यम् कुक्षिशूलमथारुचिम्।

पांडुत्वमुष्णवातत्वम् च तृष्णाह्रत्पीडनं वमिम्।। सु.नि.३–१६/१७

When Ashmari gets converted into Sharkara, it gives following situations-

1. Weakness
2. Lossness in body organs
3. Leanness
4. Pain in abdomen
5. Loss of desire of food
6. Pale yellowish skin
7. Ushnavata
8. Excessive thirst
9. Paining at heart region
10. Vomitting

Samprapti of Mutrakrucchra Vyadhi– सम्प्राप्ति

१. पृथङ्गमला: स्वै: कुपिता निदानै: सर्वेऽथवा कोपमुपेत्य बस्तौ ।
मूत्रस्य मार्ग परिपीडयिन्ति यदा तदा मूत्रयतीह कृच्छ्रात् ।। मा.नि.

By Madhav Nidan,
Vata, Pitta & Kapha gets vitiated in body & acts as Malaby frequent intake of above mentioned Nidan or Hetu
These Mala gets vitiated lonely or together & go to the Basti (At Basti region Khavaigunya should be there)
There they disturbs the function & normal physiology of urinary tract & makes urination painful & generates 'Mutrakrucchra Vyadhi'

२. पक्वाशये कुप्यति चेदपान: स्त्रोतांस्य अधोगति बलि स रूद्धवा।
करोति विण्मारूत मूत्रसङ्गं क्रमादुदावर्तमन: सघोरम् ।
रूग्बिस्तहृत्कुक्ष्युदरेष्वभीक्ष्णं सपृष्ठपार्श्वेश्चतिदारूणास्यात् ।।
आध्मानहृल्लासविकर्तिकाश्च । तोदोऽविपाकाश्च स बस्तिशोथ: । च.चि. २६-५

By Charak Samhita Nidan Sthan-
Aapan Vayu which controls Pakwashaya, Basti, Uru (thighs), Vankshan (inguinal region) gets vitiated in Pakwashaya by Hetu- Sevan & it obstructs all downward system
It obstructs the regular motion of stool, urine, flatus & generates severe Udavarta
So patient gets very severe, unbearable pain in lower back, buttock as well as heart, Basti & lower abdomen region
Same time patient gets nausea, fissure, pricking pain & retention of flatus in abdomen as well as indigestion & odema at Basti region.

Mootravaha Srotas - मूत्रवह स्रोतस् दुष्टी कारणे –

मूत्रितोदकभक्ष्यस्त्रीसेवनान्मूत्रनिठाहात् ।

मूत्रवाहिनी दुष्यन्ति क्षीणस्याभिहतस्य च ।। च.वि. ५–२८

Suppression of urge of urination, excessive water intake, consumption of excessive Mansa i.e meat, sexual activity even after the urge of urination resulting in urinary tract vitiation in turn develops the various symptoms and diseases.

मूत्रवह स्रोतस् दुष्टी लक्षणे –

अतिसृष्टमतिबद्धं प्रकुपितमल्पाल्पमभीक्ष्णं वा बहलं सशूलं मूत्रयत्नं दृष्ट्वा

मूत्रवहान्यस्य स्रोतांसि प्रदुष्टमीति विघात् । च.र. ५–१४

If the Mootravaha Srotas gets vitiated following symptoms can be observed –

1. Painful and burning micturition
2. Increased urgency and frequency
3. Less or high quantity urine passes at a time
4. Dribling or incontinence of urine

ANNAVAHA SROTAS

According Charak Vimansthaan 5-11 Annavaha Srotas contains much, gala, vamaparshwa (annanalika) , amashaya and laghuantra

Food is required for life and the digestion of food gets starts right from mouth itself. Tridosha, Dhatu, upadhatu and mala are the important factors in each digestion in the body

According ayurveda Bodhak Kapha at tounge, kledak kapha in aamashaya plays very important rolke in mixing and churning of food as well as secrtetion of gastric juices which plays very important role in food digestion (1)

From the distal part of aamashaya, food gets processed by pavcak pitta, jatharagni, bhootagni as well samaan vayu and this process of digestion gets continued till the end of small intestine.(2)

Till this part of the body, the food nutrients gets digested and converted by related secretions and aahararasa gets ready for the nutrition to another Dhatus(3)

Henceforth food gets converted into mala and thus katu avastha paaka gets completed and normal vatadosha gets formed and thatafter in pakwashaya , the separation of drava and kitta mala takes place

Causes of Annavaha srotas dushti-(4)

Excessive diet without the concern of kaal , matra, rashi guna. Intake of opposite characteristics at a time , excessive sweet, excessive bitter or salty food, repeatedly consumption of food, chinta, shoka, bhaya causes annavaha srotaqs dushti

Lkshana-

Indigestion, loss of apetite, Nausea ,pain, butrning,belching

Here is some important practically diseases discussed by classical text

Agnimandya-

Definition-

The vitiation of guna of agni causes the malfunction of properties of Agni so the further process of nourishment of dosha dhatu mala gets disturbed and the function of metaboilism on every cells affects ultimately is called agnimandya(5)

Vishamagni always creats dhatu vaishamya as its work and processes always in irregul;ar manner so regularity which requires for the secretion and proper digestion do not occurs(6)

Tikshagni always works very fast and within short time of period on the food material so the time for processing of digestion requires very little as well as the endproduct also don't work as per predicted and it leads to dhatukshaya

Inspite of taking small quantity of food, still indigestion and related symptoms occurs then the situation or status of agni will be called as mandagni

Charaka well explained the effect of agnimandya in chikitsa 15^{th}(7)

1.sushrut su.21-14 page 102, Va. Su. 12-17 page 125

.Su.su.21-14 pg.102, va.su. 12-16pg.194

2.asht.Su.20/2 pg.146

3. va. Ni 5-22 pg.479

4. Ch. Vi. 5-20 pg. 528

5.Ch. vi 6-12-13 page 536

6. ch. Chik. 15-50 page 1194

7.Ch.Chiki.15-46to 49 page 1193

AJEERNA

Defination-

When food gets undigested and remains as it is in stomach is called ajirna(1)

Causative factors-

Excessive water intake, food at w3rong time with wrong combinations, irregularity in quantity and intake time of the food, excessive spicy food, afternoon nap, excessive thinking, food intake without hunger leads to ajirna(2)

Lakshana

Loss of apetite, heaviness in abdomen, tastelessness,excessive mental and physical exhaustion,constipation, mild fever,headeache,indigestion liquid also, hyperemisis,vertigo,excessive thirst(3)

Types-

Aamajirna-

Kapha dosha is vitiatated so indigestion occurs and mild odema on supraorbital region, itching in all over body,hyperemesis are the main symptoms(4)

Vidagdhajirna-

Irriation and burning sensation in chest and throat, Over perspiration, vomiting, nausea,vertigo,fever, excessive thrist(5)

Vishtabdhajirna

Pain inabdomen, body , headache, lower abdomen, waist also(6)

Rasasheshajirna

Loss of taste and apetitie , heaviness in all over body, shortness of breathing, mental exhaustion, mild headache, constipation(7)

Dinpaki ajirna

Retention of gases mild distention of abdomen after taking the lunch or heavy breakfast(8)

1.Ma.ni ajirna 5-6 page 101
2.Ma.Ni agnimandya7-9 page 103
3.Ma.ni. agnimandya14 pg.104 ch.vi. 2-8 page 501
4.ch.chiki. 15-45 pg.1193
5.Su.su.46 commentry 46-502-503 page 251
6.Ch. vi. 29/pg.502
7.Ma. Agni. 10-12 pg.103
8.ashtg.su.11/44 induti.pg.107/108

AROCHAK

Inspite of hunger if one find the loss of taste is called as arochak(1)

Types-a. vataj b. pittaj c. kaphaj d. sannipataj e. manasaja (2)

Lakshan-

a. Vataj- pain in chest, bitter mouth. Tastelessness
b. Pittaj- Burning in chest region, bitter test, excessive thrist, vertigo
c. Kaphaja- sweetness in mouth, shivering,itching, heaviness,
d. Sannipataj- all tests equally, tridoshaja symptoms at atime
e. Manasaj- Loss of desire of food ,tastelessness(3)

1.Bhav.Madh.page 474

2.tika su.u.57-3 page.784

3. ch. Su. 28-24pg.378,ch.chik.8-60pg.1076,su.ut.57-3-6

CHARDI-

Retention of food due to vitiated dosha is called as chardi(1)

Types-

a.Vataj- vata gets vitiated byexcessive exertion,high dose medicines,shock, different types of diseases,scare,shock and takes out all vitiated doshas from mouth(2)

b.Pittaj- sour,bitter and hot to touch food items vitiate pitta and its irritates trasavaha srotas and take out all food with burning sensation in chest.(3)

c.kaphaja-Excessive daysleep, excessive intake of oily and sweet items,excesswive eating of raw materials leads to vitiation of kapha and vitiated kapha takes out all food in stomach is called as kaphaja chardi(4)

d.Sannipataj- Mixing of symptoms of all vitiated doshas together(5)

e.Dwishtarthaja-unpleasant food items and things leads to this type of chardi(6)

f. aagantooja- Very spontaneous and casual type of chardi(7)

Causative factors-

Excessive liquid diet,excessive oily,eating food on wrong time,excessive sour,bitter and salty food,eating food in hurry indigestion,worms,pregency leads to chardi(8)

Vyan and udaan takes all vitiated doshas from stomach to mouth .Stomach gets irritated by vitiated kapha and pitta , so vayu also gets retained and chardi takes place.(9)

1.Su.u.49-6 satik

2.va.ni 5-30 satik

3. Su.utt. 49-7

4. Ch.chik.20-20

5. Ch.chik.20-9, su.utt.49-9

6. Ch.chik.20-10, su. utt. 49-10

7. Ch. chik 20-12, su.utt.49-11

8. Satik ch.chi.20-14

9. Cha.chi.20-18 satik

AMLAPITTA-

Due to vidgadhavastha pitta dosha increases with sour guna and forms amlapitta(1)

Types-

As per Marga

a. Urdhwaga
b. Adhoga

As well as as per dosha

a. Vatanubandhi
b. vatakaphanubandhi
c. kaphanubandhi(2)

Causative factors-

Rasa virya vipaka virudhha food , contaminated, poisionous,excessive sour, excessive spicy ,salty, oily excessive liquid, roasted sprouts, excessive milk,colostrums poha, nonfermented alcohol, dried vegetables, raagi as well as holding of natural urges, frequently eating, excessive water intake, longer time hot water bath, mental stress, immediate sleep after dinner or lunch vitiates drava guna of pitta and abhishyandi and vidahi guna increases this forms agnimandya and its further vitiates and achives vidagdhavasthaand forms amlapitta in aamashaya(3)

Symptoms-

Indigestion,exhaustion,irritation , burning sensation,stomachache, headache, liquid stool, excessive belching and tremors, heaviness in abdomen(4)

Urdhwaga amlapitta-

Vomiting is the main symptom. The liquid in vomit always colorful, irritable belching, headache,fever, tastelessness,rashes on the skin, nausea are the symtoms of urdhwaga amlapitta(5)

Adhoga amlapitta

Liquid stool, colorful stool, very much foul and pungent smell, excessive thist, burning sensation, irritation excessive sweating(6)

Vatanubandhi amlapitta

Tremours, kampa,moorcha,vertigo, numbness, darkness infront of the eyes,(7)

Kaphanubandhi amlapitta

Hemaptosis,heaviness of body,distention of abdomen,loss of apettite, weakness,feling always sleepy are the main symptoms

1.Ma.ni amla.1

2.Sha.pra.7 -103

3.kashyp 335, Ma.ni amlapi.1pg.246

4.ma.Mi. Amla.4,5,6

5. ma.ni amla. 3 page 346

6. Asht.su.5-33 indu tika

7. Ma.amla.8-123,pge348

SHOOLA-

Piercing pain in abdomen is called as shool(1)

Dosha is vata and doshya are rakta or majja

Symptoms-

Indigestion. abdomen pain, distention of abdomen, constipation

Types-

a) Vataja-Excessive exertion, jerking journey, frequent sextual act, excessive drinking of chilled water,exceeive dry food items,dry vegetables, overfasting,excessive and continous talk,excessive laugh creats vitiation of vata with ruksha guna and forms shool(2)
b) This aggrevates in constiopation and when it gets relived it gets decresed.
c) Pitta- ksharyukta, Excessive ruksha, excessive tikshna ,ksharyukta ,continuous work besides hot places or fire stations, excessive sexual intercourse, excessive exertion leads to vitiation of pitta and its gets mixed with vitiated vata and form shoola with daha(3)
d) Kapha- Intake of excessive oily food, repeatedly intake of sweet food items, afternoon nap, immediate sleep after dinner or lunch leads to vitiation of kapha and its with vitiated vata forms kaphaja shool(4)
e) Sannipataj- All symptoms of tridosha simultaneously form the saanipatik type of shool.(5)
f) Vatapittaj- Vitiation of vata and pitta forms vatapittaj shool(6)
g) Vatakaphaja- Vitiation of vata and kapha shows the symptoms of vatakaphaja shool(7)
h) Kaphapittaj- Vitiation of kapha and pitta shows the mix symptoms of kapha and pittaja shool(8)
i) Aamaja- indigestion of ingested food generates Aama by vitiation on agni characters leads to aamaja shool it is very painful and significant(9)
j) Parinaamashool- At the digestion time of food when the shool gets increased then that is called as parinamashool(10)
k) In Parinamashool, the predominance of vata pitta and kapha as well as permutation and combination of dosha shows the respective characters of vitiated state (11)

l) Annadravashool –Continous severe pain in abdomen at the time of food intake, after food intake at the time of digestion (12)

m) Kukshi shool-Vitiated vata form agnimandya which forms indigestion as well as shortness of breath. Feels discomforts in every position of body. This type of shool generates by Aama and vayu(13)

n) Annadoshaja shool-In indigestion orat the state of agni guna(14)

1) 1.Satik S. ut.42-81. Pge.723, Ma.ni. shool 1 tika
2) Ma. Ni shool 2-5
3) 3.ma. Ni. Shool 6-8, Su.ut.42-84 pge.723
4) 4.Ma. Ni shool9, 10, Su.ut.42-85,86pge 723
5) 5.Satik Ma.ni. Shool 11, Satik Su.utt.42-87 pge 723
6) 6.Ma. Ni. Shool 13, Pge 224
7) 7. Ma.Ni shool 12, pge 224
8) 8. Ma. Ni shool15,16
9) 9. MA. Ni shool 17-20
10) 12. Ma. Ni.Shool 21,22
11) 13.Satik su.utt.42,123-125
12) 14.Satik Su. Utt. 42, 142-144

GRAHANI-

In this disease the organ grahani gets affected so the name is grahani(1)

Types-

a. Vataj- excessive intake of bitter, sour, excessive dry, chilled food leads to indigestion by weakness of Agni or digestive power.(2)
b. Pittaj- excessive intake of sour, amla, kshaar food leads to vitiation of drava guna of pitta and agni gets affected(3)
c. Kaphaja - Excessive heavy, excessive oily, chilled food , sleep immediate after lunch leads to kaphaja grahani(4)
d. Sannipataj - Mixed symptoms of all doshas at a time is called as sannipataj (5)
e. Sangrahani- Excessive weakness, low backache, are the significant symptoms(6)
f. Aamavastha-Heaviness, tastelessness is the significant symotoms in this type occurs.(7)

Improper or wrong way of eating habits leads to agnimandya so absorption, holding and separation as well motivation of food in the next part of digestive system these all functions gets disturbed. This repeating situation weakens this organ. So function of paachakagni and saman vayu also gets vitiated and the absorption for stool formation gets disturb so the eaten food spells out from stool as a main content so this condition is called as grahani(8)

The consistency of stool becomes semisolid. When pitta gets vitiated by drava guna then stool becomes more watery

Mala gets saama and so it gets foul smell, patient becomes more weaker day by day

1.Ma. ni. Grahani 3
2. satik su.u.40-177
3. ch.chik.15/56-64, ma.Ni grahani tika page 83
4. Ch.chik.15/65
5. Ch.chik.15/67-70, Ma.ni.grahani 16
6. Mi.Ni.grahani 17.pge 85
7. Ma. Ni grahani pge 85
8. Ch. chik.15-73
9. Ma. Ni.grahani pg 85

REVIEW OF INVESTIGATIONS

REVIEW OF INVESTIGATIONS

As per study designs following investigations are important –

1. Blood test for Haemogram**& ESR**
2. Blood tests for Serum **Creatinine & Serum Urea**
3. Urine tests **Microscopic & Routine**

Blood test for **ESR and Haemogram**

Haemogram contains

1. Haemoglobin % (Hb %)
2. White blood cell count (WBC)
3. Red Blood Cells count (RBC)
4. Packed Red Cells Volume (PCV – Absoloute indices)
5. Mean Corpuscle Volume (MCH – Mean Corpuscle Haemoglobin)
6. MCV
7. MCHC
8. Differential Leukocyte Count (DLC)
9. Platelet Count

Erythrocyte sedimentation Rate (ESR)

Haemoglobin

Introduction-

Haaemoglobin is the main constituent of the red blood cells and transports oxygen from lungs to various parts of the body. It also transports carbon Dioxide from the body to the lungs

Fully saturated each gram contains 1.34 ml of oxygen.

The red cell mass of an adult contains approximately 600gm of haemoglobin, capable of carrying 800ml of oxygen

Estimation methods–

- Sahali's Method
- Cynmethaeglobin Method
- Sheard Samford
- Oxyhaemoglobin Method

Other Methods

- Alkali Haematin Method
- Gasometric Method
- Specific Gravity Method

Chemical Method –

- Haemoglobin is estimated by finding the iron content
- Sodium Lauyl Sulphate Method

1. **Sahali's method-**

   This method is based on conversion of haemoglobin to acid haematin, which as brown color in the presence of N/10 HCL

   1. Fill haemoglobin tube till 20 mark with N/10 HCL
   2. Add 20 mul blood in that with the help of Haemoglobin pipette
   3. Wait for 5-45 minuets
   4. Keep stirring this mixture
   5. Add distilled water until the match is obtained with the brown glass standard (comparator) provided.
   6. Lower level of meniscus is the report of Haemoglobin in gm/100ml of blood
   7. if Haemoglobin is less than 2gm% take double quantity of blood and divide the result by 2
   8. If Haemoglobin concentration is extremely high dilute blood with equal amount of saline, take reading & multiply by 2

   Limitations-

   This method however does not estimate carboxyhaemoglobin, methemoglobin and sulphhaemoglobin. Non-haemoglobin substances (protein, lipids) in plasma & cell stroma may influence the color of blood diluted with acid.

Cynmethoglobin method-

Drabkin solution contains-

Sodium Bicarbonate-	1.0 gm
Potassium cyanide	0.05 gm
Potassium Fericyanide	0.2 gms
Distilled water	1000cc

Precaution-

This solution should be made once in a month & should be stored in a brown bottle

Method-

1. To 5 ml of Drabkin's solution add 20 mul of blood.
2. Mix well
3. Read in a photocolorometer at 540 nm (green filter)

Precaution-

Pipette carefully and take care not to discard cyanide solutions into sinks or receptacles contains acid (green filter)

Sheard- Sanford method

Method-

1. Mix 20 ml of 0.1 % sodium carbonate & 0.1 ml of blood or aliquots of these (eg. 4ml dilutent for 20 ml of blood)
2. Read optical density in photometer at 540 nm within 30 minutes

Precautions-

Photometer calibration should be based on blood iron determination or oxygen capacity determination.

Other methods-

1. Alkali haematin methods-

It does not estimate foetal haemoglobin

2. Gasometric method-

It is an indirect method, which estimates the amount of haemoglobin from amount of oxygeon it absorbs

3. Specific gravity method-

The normal specific gravity of blood ranges from 1.048 to 1.066

The average for men is 1.057 & for women it is 1.053

From specific gravity of the unknown sample its haemoglobin is calculated

Chemical method-

Haemoglobin is estimated by finding the iron content

2. Pulmonary disease resulting in **Normal Haemoglobin Values –**

Men-	14 – 18 gm %
Women-	11.5- 16.5gm %

Infants (Full term)-

Cord blood-	13.5 – 19.5 gm %
Children (1 year)	11 – 13 gm %
Children	11.5 -14.5 gm % (10 – 12 years)

Anemia

Definition –

It is defined as reduction in concentration of hemoglobin in the peripheral blood below the normal for the age & sex of the patient

Diurnal variations-

Hb values are highest in the morning & lowest in the evening

A change in the Hb must be 1.5 gm % or more to be considered definitely significant

Causes of Anaemia –

1. Blood loss

- Acute post- hemorrhagic anemia
- Chronic post – hemorrhagic anemia

2. Impaired blood cell formation

a.Disturbance of bone marrow due to deficiency of substances essential for erythropoesis

- Iron deficiency of anemia
- Megaloblastic macrolytic Anemia due to deficiency of Vit. B 12 or Folic acid
- Anemia associated for scurvy
- b.Disturbance of bone marrow function not due to deficiency of substances essential for erythropoiesis

Anemia associated with

- Infection
- Renal failure
- Liver disease
- Disseminated malignancy
- Aplastic anemia
- Anemia associated with bone marrow infiltration e.g. Leukemia, malignant lymphoma, multiple myloma, mylosclerosis
- Anemia associated with mysodema & hypopitutirism
- Sideroblastic anemia
- Congenital dyserythropoetic anemia
- Incresed destruction of red cell (Haemolytic anemia)
- Haemolysis due to corpuscular defects (intracorpuscular or intrinsic defect)
- Haemolytic anemia due to abnormal haemolytic mechasnisims
- (Extracorpuscular or extrinsic factors)

Polycythemia –

Definition –

Raised haemoglobin value for age & sex of the patient is called Polycythemia

Polycythemia (erythrocytosis)

Increased in Hb

- Above 18 gm % in males
- Above 16.5 gm % in females

In addition there is

- Increase in red cell count;
- Above 6 million/cu mm in females
- Increase in Haematocrit

  Above 55 % in males

  Above 47 % in females

Causes of Polycythemia –

Primary – Polycythemia Vera

Secondary –

A. Associated for hypoxia

1. Cardiovascular disease, usually congenital resulting in significant venous admixture

- Impaired gas perfusion
- Perfusion of poorly aerated lungs
- Pulmonary arteriovascular fistula

3. High altitude residence

4. Hypoventilation associated with obesity

5. Haemoglobin is variants with increased affinity for oxygeon

6. Heavy smoking

7. Methaemoglobinomea

B. Due to imappropriate erythropoietin increased in

1. Benign / malignant tumors of

- Kidney
- liver
- CNS
- Uterus
- Ovary

2. Renal disease (besides magnancies)

- Hydronephrosis
- Vascular impairment
- Cysts

C. Associated with adrenocortical steroids or Androgens

1. Adrenal hypercorticism (all types)
2. Virilizing tumors
3. Androgens used therapeutically (rarely corticoids)

D. Associated with chronic chemical exposure

1. Nitrites, sulphonamides , other substances producing methanoglobin and sulphomeglobin .
2. Cobalt, shellac components, various alcohols

E. Relative

1. Stress or spurious polycythemia
2. Dehydration: water deprivatiobn , vomiting
3. Plasma loss: burns, enteropathy

Blood Cell Count -

WBC –

A white cell count (TLC) estimates the total number of white cells in cubic millimeters of blood.

The diluting fluid-

It contains a weak acid to lyse the red blood cells and a stain for staining the nucleus of white blood cells

Turke's fluid-

Glacial acetic acid	1.5 ml
1 % aqueous solution of Gentian Violet	1.0 ml
Distilled water	98.0 ml

Counting chamburs-

Newbaur chembur- area 9 sq/mm Depth- - 0.1 mm

Methods –

1. Using a WBC pipette of a haemacytometer draw a well mixed venous blood or capillary blood & fill it till the 0.5 mark. Clean the tip of the tube. Now draw the WBC diluting fluid till the 11 mark
2. Mix the fluid & blood mixture gently, avoiding bubbling
3. Place the cover slip on the counting chamber at the right place
4. Shake the fluid blood mixture and transfer the mixture using a fine bore Pasteur pipette on to the counting chamber (called charging the chamber), taking care that the mixture does not overflow.
5. Allow the cells to settle to the bottom of the chamber for 2 minutes
6. For counting, clean the under part of the chamber if it was left on petridish and place it on the stage of the microscope.
7. Using 10 X or low power objective count the WBC uniformly in the four layer corner squares
8. Cells present on the outermost lines should be counted on the one side and those present on the line opposite should not be counted

Calculate the number of cells per cubic millimeters of blood as follows

$$\frac{\text{Cell counted} \times \text{blood dilution} \times \text{chamber depth}}{\text{Area of chamber counted}}$$

$$= \text{number of cells counted} \times \frac{20 \times 10 \text{ (depth factor)}}{4}$$

= number of cell counted × 50

- (Dilution factor is 20 for there is no mixing of cells till first 1 mark of the WBC pipette, hence 0.5 parts of blood are present in 10 parts of the diluting fluid dilution factor is 20)

Correcting the white cell count for nucleated red cells-

Calculation-

$$\frac{\text{Number of nucleated RBCs} \times \text{TLC}}{100 + \text{nucleated RBCs}}$$

= Nucleated RBCs /cu. mm

Corrected count = TLC - Nucleated red cell count

Normal total leucocytes count=

4,000-11,000 cells /cu. mm

TLC undergoes minor physiological and diurnal variations

It increases slightly in the afternoon ‘afternoon tide’. Various stimuli that may increase the count are**:**

- Food intake
- Physical exercise
- Emotion
- Pregnancy & following parturition

Pathological variations in white cell count neutrophillia

Infections

- Pyogenic bacteria
- Staphylococcal
- Streptococcal
- Pneumococcal
- Meningococcal
- Gonococcal

Non-Pyogenic

- Acute rheumatic fever
- Diphtheria
- Scarlet fever
- Acute poliomyelitis
- Cholera
- Herpes zoster
- Mycobacterial
- Fungal
- Spirochetal
- Parasitic

Metabolic disorder

Due to varied causes leading to

- Acute yellow atrophy of liver
- Uremia
- Diabetes
- Acidosis
- Gout
- Aclampsia

Neoplasms

- Myeloproliferative disorders
- Myeloid leukemia
- Lymphomas
- Polycythemia Vera
- Myleosclerosis
- Other malignancies
- Carcinomas
- Sarcomas

Conditional causing cell necrosis or distruction

- Acute haemolysis
- Infractions
- Drugs intoxications
- Nephrotoxins
- Hepatotoxins

Various drugs chemicals implicated are

- Phenacetin
- Digitalis
- Quinine
- Organic arsenics
- Lead
- Mercury
- Carbon monoxide

Trauma & hemorrhage

- Hemorrhage
- Acute hemorrhage
- Trauma
- Operatives
- Fractures
- Crush injuries
- Burns

Cardiac disorders

Collagen diseases

- Polyarthritis nodosa
- Acute phase of Rheumatoid arthritis
- Dermatomayosistis

Miscellaneous

- Serum sickness
- Acute anoxia
- Spider venom poisoning
- Histiocytosis

Eosinophillia

Allergic states

- Asthma
- Hay fever
- Exfoliative dermatitis
- Erythema multiforms
- Urticaria
- Food sensitivity
- Angioneurotic odema
- Serum sickness
- Drug allergy

Parasitic diseases Interstitial

- Hookworm
- Roundworm
- Tapeworm

Tissue form

- Toxocora
- Trichiina
- Storongyloides
- Echinococcus
- Filiarisis
- Malaria

Skin disorders

- Pemphigus
- Dermatitis herpetiformis
- Psorasis
- Scabies
- Prurigo

Drug administrations

- Liver extracts
- Penicillin
- Stre4ptomycin
- Chlorpromazine

Neoplasms

- Myleoproleferative
- Eosinophillic leukemia
- Chronic Myeloid leukemia
- Polycythemia

Others

- Hodgkin’s syndrome
- Multiple myloma
- Metastatic and necrotic tumors
- Occult abdominal tumour

Miscellenous

- Familial eosinophillia
- Eosinophillic syndrome
- Eosinophyllic granulomatosis
- Scarlet syndrome
- Polyarthritis nodosa
- Tropical eosinophyllia
- Pernicious anemia
- Post- splenectomy
- Post- transfusion mononucleosis
- Idiopathic neutropenia

Lymphocytosis

Acute infections

- Infectious mononucleosis
- Infectitious lymphocytosis
- Pertussis
- Mumps
- Chicken pox
- Rubella
- Infective hepatitis
- Convalescent stage of many acute infections
- Toxoplasmosis
- Influenza

Chronic infections

- Brucellosis
- Tuberculosis
- Syphilis

Endocrine disorders

- Thyrotoxicosis
- Adrenocortical insufficiency
- Hypopitutriasm
- Myaesthenia gravis

Neoplasms

- Non- Hodgkin’s lymphomas
- Chronic lymphatixc leukemia
- Lymphosacroma
- Multiple myeloma

Monocytosis

Infections

Bacterial

- Brucellosis
- Tuberculosis
- Subacute bacterial endocardities
- Typhoid fever
- Recovery stage of an acute infection

Rickketisial

- Rocky mountain spotted fever
- Typhus

Protozoan

- Malaria
- Kala- azar
- Trypanosomiasis
- Oriental sore

Viral

- Infectious mononucleosis

Neoplasms

- Monocytic leukemia
- Hodgkin’s and other lymphomas
- Myeoproleferative disorders
- Multiple myleoma
- Carcinomatosis

Collegen diseases

- Rheumatoid arthritis
- SLE

Miscellenous

- Chronic ulcerative colitis
- Regional enteritis
- Sarcoidosis
- Lipid storage diseases
- Haemolytic anemias
- Hypochromic anemias
- Recovery from agranulocytosis

Basophillia

- Chronic myeloid leukemia
- Myleosclerosis
- Polycythemia vera
- Hypersensitivity status
- Myxedema
- Iron deficiency
- Haemolytic and topxic anemias of long standings
- Pre- leukemia

Neutropenia & agranulocytosis

Neutropenia is reduction of circulating neutrophills below 2500 cells /cu. mm

Causes of neutropenia

Drugs-

1) Drugs that cause aplastic anemia
2) Drugs that induce selective neutropenia
    - Antipyretics
    - Analgesics
    - Antithyroids
    - Tranquillizer & antidepressant
    - Antibacterial
    - Anti- coagulants
    - Anti –tubercular
    - Anti –malarial
3) Chronic idiopathic neutropenia
4) Infections
5) All causes of aplastic anemia
6) Myeleophthalsis
7) Neutritional deficit
8) Hypersplenism
9) Miscellaneous
    - SLE
    - Anaphylaxis
    - Antileukotic antibodies
    - Immunodefficiencies
    - Pancreatic exocrine deficiency
    - Cystic neutropenia

RBC-Red blood cell count

Diluting fluid-

This should be isotonic so that RBC are not haemolysed.

Normal saline can be used but it may cause crenation of the RBC's and allow rouleaux formation

One can use-

1. Sodium citrate 3gm
   Formaline 1ml
   Distiilled water to 100 ml

OR

2. Hayem's fluid
   Mercuric chloride 0.5 gm
   Sodium chloride 1.0 gm
   Sodium sulphate 5.0gm
   Distilled water to 200 ml

Methods –

1. Draw blood to the 0.5 mark in the RBC pipeete
2. Wipe tip clean and draw diluting fluid to the 101 mark
3. Shake for 3 minutes
4. Charge the chamber
5. Count the RBC's using 40* objective in the 80 smallest squares as indicated in the diagram of the chamber

RBC count-

$$\frac{\text{No of cells counted} \times \text{Dilution factor} \times \text{Depth factor}}{\text{Area counted}}$$

Where dilatation is 1 in 200, depth is 1/10 mm

Area counted is 80/400=1/5 sq. mm.

$$\frac{\text{Number counted} \times 200 \times 10}{1/5}$$

=Number counted ×10,000

RBC counts are low in anemia and high in polycythemia, the cause of these have been already discussed

Haematocrit or Packed Cell Volume (PCV) –

Definition –

Haematocrit is the volume of red cells expressed as the percentage of the volume of the whole blood in sample

The venous haematocrit is almost same as that obtained from a skin puncture

Dried heparin, EDTA or double oxalates are satisfactory anticoagulants

Methods –

A.Using Wintrob's tube-

1. Fill the Wintrob's tube till the 100 mark on top with the Pasture pipette ensuring that there are no air bubbles in air column
2. Centrifuge this tube for 15 minutes at 3500 rpm (or longer at lower speed)untilled the packing is complete
3. After centrifuging the blood is separated into 3 layers , a column of red blood cells at the bottom, a narrow middle layer – buffy coat of white blood cells & plateletsvand topmost fluid level of plasma
4. The percentage of the height of the column of the blood occupied by packed red cells constituents with haematocrit

Errors-

1. Inadequate mixing of blood
2. Irregularity of the bore of the tube
3. Incomplete packing

Values

Men- Range 42-52 % Avarage- 47 %

Women- Range 37-47 5 Avarage 42 %

Causes of reduced Haematocrit-

1. Blood loss
2. Acute post- hemorrhagic anemia
3. Chronic post – hemorrhagic anemia

4. Impaired blood cell formation
5. a.Disturbance of bone marrow due to deficiency of substances essential for erythropoesis
6. Iron deficiency of anemia
7. Megaloblastic macrolytic Anemia due to deficiency of Vit. B 12 or Folic acid
8. Anemia associated for scurvy

b.Disturbance of bone marrow function not due to deficiency of substances essential for erythropoiesis

- Reduced haematocrit associated with
- Infection
- Renal failure
- Liver disease
- Disseminated malignancy

Causes of raised Haematocrit-

Associated for hypoxia

1. Cardiovascular disease, usually congenital resulting in significant venous admixture

2. Pulmonary disease resulting in

- Impaired gas perfusion
- Perfusion of poorly aerated lungs
- Pulmonary arteriovascular fistula

3. High altitude residence
4. Hypoventilation associated with obesity
5. Haemoglobin is variants with increased affinity for oxygeon
6. Heavy smoking
7. Methaemoglobinomea

B.Due to imappropriate erythropoietin increased in

1. Benign / malignant tumors of

- Kidney
- liver
- CNS
- Uterus
- Ovary

2. Renal disease (besides magnancies)

- Hydronephrosis
- Vascular impairment
- Cysts

C.Associated with adrenocortical steroids or Androgens

1. Adrenal hypercorticism (all types)
2. Virilizing tumors
3. Androgens used therapeutically (rarely corticoids)

D.Associated with chronic chemical exposure

1. Nitrites, sulphonamides , other substances producing methanoglobin and sulphomeglobin .
2. Cobalt, shellac components, various alcohols

E.Relative

1. Stress or spurious polycythemia
2. Dehydration: water deprivatiobn , vomiting
3. Plasma loss: burns, enteropathy

If packed cell volume has been determined by Wintrob's tube, one can obtain some more information

Buffy coat-

A buffy coat of thickness 1 mm approximately corresponds to a total leucocyte count of about 10,000. Absent or minimal buffy coat implies lecopenia, a thickness more than 1mm implies leucocytosis. In addition in subleukemic leukemia, a film can be made from the buffy coat where the greater concentration of white blood cells will be available,& identification of atypical cells would become easier & less time comsuming. Another advantage is for performing L.E. cells or phenomena test for which also white blood cells can be picked from buffy coat. The platelets form a very thin layer above the white cells, the coat is pinkish white but is of no us clinically, one has to do platelet counts if necessary.

Plasma layer

The topmost layer of plasma can give important clues by observing its colour
Its normal colour is pale yellow or straw

Yellow-	Jaundice
Pink-	Haemolysis
Creamy white-	Hyperlipidemia , especially chylomicrons
Brown-	Methaemalbuminaem

Platelets –

Preferably use venous blood for platelet count. Finger prick may cause clumping of platelets

The blood is diluted in 1% ammonium oxalate stored refrigerated at 4degree which haemolysed the red blood cells

Methods –

1. Fill the blood & dilutent
2. Charge the chamber with the help of the pipette employed
3. Using 40 ×* objective with reduced condenser aperture count the platelets in the same squares as indicated for RBC counting
4. calculated as (if RBC pipette used)

$$\frac{\text{Cells counted} \times \text{blood dilution} \times \text{chamber depth factor}}{\text{Area of chamber counted}}$$

$= N \times 200 \times 5 \times 10$

$= N \times 10{,}000$

(However, if a WBC pipette is employed the appropriate formula &method should be used. Platelet counts are made in the small 5 RBC squares only

Platelet count = $= N \times 20 \times 5 \times 10$ or $N \times 1{,}000$

Diluting fluid

Consists of

Trisodium citrate	3.8gm
Neutral formaldehyde	0.2 ml
Briliant crystal blue	0.1 gm
Distilled water	100ml

Precautions-

All glass wares are must be compulsory clean. Dust or dirty particles can resemble & may be counted as platelets

Procedures

1. Take 3.98 ml of dilutent
2. Add to the dilutent 0.02 ml of well mixed anticoagulant blood with the help of a Sahli's pipette wipe out the outer tip of the pipette before dilutes.
3. Wash out the contents in the pipette into the diluents tube 3-4 times
4. Immediately mix the diluents with the specimen for at least 5 minutes or so
5. Employ the Sahali's pipette for charging either side of the chamber
6. Keep the charged Haemocytometer inside a moist chamber
7. Let stay for about 15 minutes. This permits the platelets to settle down, and the moistened chamber does not allow evaporation of the fluid
8. Place the haemocytometer on the stage of the microscope; focus the RBC counting area under low magnification. Now move to the corner square of the red cell area carefully to high dry objective
9. Platelets are bluish and must be distinguished from debris. They are oval ,round or comma shaped retractile bodies that vary in size normally from 1 to 5 microns
10. Count the platelets in the finely ruled centre area (1mm2)of each side of the chamber
11. Take the average count of 2 sides

Failure of production of platelets (Thrombocytopenia)

1. Causes of platelet production failure
   a. Selective megakaryocytic depression
   b. Drugs
   c. Chemicals
   d. Viral infections

Part of general bone marrow failure

a. Aplastic anemia

b. Leukemia

c. Myleosclerosis

d. Marrow infiltration e.g. carcinoma, lymphoma

e. Multiple myloma

f. Megablastic Anemia

2. Increased destruction of platelets

a. Acute or chronic (ITP) idiopathic thrombocytopenic purpura

b. Secondary immune thrombocytopenia

(Post infection, SLE, CLL, and lymphomas)

3. Abnormal distribution of platelets

Splenomegaly

4. Dilutional loss

Massasive transfusion of old blood to bleeding patients

Raised platelet count (Thrombocytosis)

Can occur as a part of generalized myeloproliferative disorder, e.g. CML or following acute hemorrhage.

Erythrocyte indices

These can be calculated from

a. Haematocrit
b. Haemoglobin concentration
c. Red cell volume

1. The mean cell volume (MCV)

$$MCV = \frac{\text{Packed cell volume}}{\text{Red cell count per litre}} \times 10 \text{ fl.}$$

Normal values-

Adults	76-96 fi.
Infants, full term cord blood Av.	106 ft.
Children 10-12 years	76-93 fl.

MCV is reduced in microlytic Anemias

MCV is raised in macrocytic Anemias

2. The mean cell Haemoglobin (MCH)

$$MCH = \frac{\text{Hemoglobin in gm/dilution}}{\text{Red cell count /ml}} \text{ pg}$$

Normal MCH in adults is from 27 to 32 pg.

MCH is reduced in hypochromic Anemia

3. The mean cell haemoglobin concentration (MCHC)

$$MCHC = \frac{\text{Hb in gm \%}}{\text{PCV}} \times 100 = 31 - 35 \text{ gm \%}$$

This too, is low in hypochromic anemia

Diffrential leukocyte count- (DLC)

There are 5 types of leucocytes	Normal ranges
1. Neutrophills-	4000 to 10,000/cu mm
2. Eosinophills	1 to 6 %
3. Basophiles	0 to 2 %
4. Lymphocytes	20 to 40 %
5. Monocytes	2 to 10 %

Blood film preparation

A thin blood film is made by spreading a drop of blood evenly across a clean grease-free slide, using a smooth edged spreader.

Makiing of spreaders

1. Select a slide which has smooth edges
2. Using a glass cutter and a ruler, mark of 4 equal division, each measuring 19 mm
3. Break off at each division to give 4 spreaders

- For anemic blood a rapid smearing is needed whereas for thick concentrated blood smearing should be done slowly. A well spraed smear shows no lining extending across or downwards through the film and the smear should be tounge shaped
- While the thin smears are used for describing blood cells, the thick smears are used for detecting malarial parasites & microfilarae. A large drop of blood is taken on the centre of the slide & with the aid of the needle or slide corner spread the drop over ½ an inch square area. When dry the thickness should be such that printed matter can be seen through it.

Fixing of blood films

Before staining, the blood film ned to be fixed with acetone free methyl alcohol for ½ to 1 minute in order to prevent haemolysis when they come in contact with water while staining them with aqueous (water based) stains or when water has to be added subsequently. Alcohols denaturs the proteins & hardens the cell contents. For wright's stain and Leshmain's stain, no prefixation is required as these contain acetone-free methyl alcohol but for Geshmain's tain prefixation is a must because the alcohol content is only 5% in the ready to ue stain.

Staining of blood film

Blood cells have structures that are acidophilic and some basophilic structure, so they vary in their reaction (pH). The nuclei are basophilic (acidic) basophill granules also stain blue. Haemoglobin (being basic) stains acidophilic or red.

Stains that are made up of combinatyion of acid & basic dyes are called Romanwasky stain & various modifications are available.

Stain preparation & staining

WRIGHT'S STAIN

Wright's stain - 0.2 gm

Acetone free Methyl alcohol 100 cc

If the WBC granules donot stand out clearly try out a 0.25 or 3% solution

Method

Cover the slide with the stain for 1-2 minutes taking care that it does not dry on the slide. Now dilute this with equal amount of buffer water. The diluted stain is allowed to act for 3-5 minutes and then the flooded off with buffer or tapped water. The stain stain should never be poured off or a precipitate of the stain will be deposited on the slide. Should this occur, it can sometimes be removed by flooding the slide with undiluted stain for 10-15 seconds and then washing it off again by flooding the slide once more with buffer water

LEISHMAN'S STAIN

Powdered Leishman's stain 0.5 gm

Acetone free methyl alcohol 133 ml

All the stain should be dissolved , keep the stain in a glass stopperd bottle. Do not filter

Method

Like that for Wright's stain but with double dilution of the buffer water. Pour few drops on the slide. Wait for 2 minutes. Add double the amount of buffered water. Mix by rocking & not by blowing & wait for 7-10 minutes.

The stain is flooded off with distilled water and this should be complete 2-3 seconds

Longer washing will remove stain

Stand in the rack to drain & air dry

GIEMSA'S STAIN

Giemsa powder 0.3 gm

Glycerine 25.0 ml

Acetone free methyl alchol 25.0 ml

This makes stock solution and before use it has to be diluted by adding 1ml to 9 ml of buffered distilled water

Method

The blood film is fixed with Methyl alcohol for 3-5 minutes & dried. Pour on diluted stain and keep for 15 minutes or longer. Wash off with tap water or neutral distilled water and dry

Always note

RBC's

Size- Normocytes, microcytes, macrocytes, Anisocytosis

Shape- Abnormal,shape oval, pencil,tear, pear, oat and sickle shaped cells, fragmented cells, target cells, spherocytes, crenated cells, burr cells, acanthocytes, stomatocytes

Haemoglobin-

Normochromoc, hypochromic

Immature forms- Polychriomatic, stippled or nucleated red cells

Inclusion bodies- Howell-jolly bodies, Cabot rings,Pippenheimer bodies, Malarial parasites etc.

Diffrential leucocyte count (DLC)

For differential leucocyte count choose an area where the morphology of the cell is clearly visible. Ensure that there is no tailing of the WBC's or else a false DLC may be obtained. Do differential count by moving the slide as shown in order to include central & peripheral area of the smear

While doing DLC look for vacuolation, toxic granulation, size & maturity of the white blood cells. Count at least 100 cells and give percentage of the cells seen. Counting becomes easier if 100 squares are made on a paper & the letters P for neutrophil

L for lymphocyte

E for eosinophill

B for basophill

Can be entered in each square

Platelets

Preferably use venous blood for platelet counts. Finger prick may cause clumping of platelets. In small children clumping can be prevented by thinly smearing Vaseline over the area to be punctured.

The blood diluted in 1% ammonium oxalate stored refrigerated at 4℃ which haemolyses the blood cells (prepared by dissolving 1 gm of ammonium oxalate in 100ml of distilled water)

Method-

- Fill blood and dilutent (in the case 1% ammonium oxalate) as described for RBC count & using the RBC pipette. If platelets count is low, a WBC pipette can be used instead.
- Charge the chamber with the help of the pipette employd.
- Using 40 x objectives with reduced condenser aperture count the platelets in the same squares as indicated for RBC counting.
- Calculate as (if RBC pipette used)

  Cells counted × blood dilution ×

  Chamber depth factor

  Area of chamber counted

  =N x 200 x 5 x 10

  =N x10000

(However, if a WBC pipette is employed the appropriate formula & method should be used. Platelet counts are made in the small 5 RBC squares only

Platelet count- N x 20 x 5 x 10 or N x 1000

Normal platelet counts = 1.5 -3.5 lakhs / cu. mm

Rees- Ecker Method for Platelet count-

- Various components of the diluting fluid used have various functions, e.g. citrate prevents coagulation while formalin fixes the platelets and prevents their clumping together. Here no attempt is made to lyse RBCs. Platelets are identified by their size, shape and dark colour. Brilliant cresyl blue (the dye used) provides the background during cell counting. This dye does not stain the platelets and therefore, is not essential for the counting procedure.

Diluting fluid-

Consists of

Trisodium citrate-	3.8 gms
Neutral formaldehyde	0.2 ml
Brilliant cresyl blue	0.1 gm
Deionized water	100 ml

- Dissolve the ndegrients in 100 ml volumetric flask, filter centrifuge, transfer to a well stopper bottle & keep at 2-8℃ (refrigerate)
- This fluid if not contaminated will stay good indefinitely. Filter aliquot of the diluting fluid immediately before use.

Caution-

All glassware must be compulsory clean. Dirt or dust particals can resemble & may be counted as platelets

Procedure-

- Take 3.98 ml of dilutent (freshly filtered) into a test tube
- Add to the dilutent 0.02 ml (20ml) of well mixed anticoagulant blood with the help of a Sahali pipette wipe out the outer tip of the pipette before dilution. Wash out the contents in the pipette into the dilutent tube 3-4 times
- Immediately mix the dilutent with the specimen for at least 5 minutes or so.
- Employ the Sahili's pipette for charging either side of the chamber
- Keep the chargerd haemocytometer inside a moist chamber (can be a petridish with the moistened or wet filter paper-or which the chamber can be kept). Let stay for

about 15 minutes. This permits the platelets to settle down, and the moistened chamber does not allow evaporation of the fluid

- Place the haemocytometer on the stage of the microscope; focus the RBC counting area under low magnification. Now move to the corner square of the red cell area carefully to high dry objective
- Platelets are bluish & must be distinguished from debris. They are oval, round or coma shaped, retractile bodies that vary in size normally from 1 to 5 microns
- Count the platelets in the finely ruled centre area (1 mm2) of each side of the chamber.
- Take the average count of two sides
- (In the new improved Newbaur ruling, there are 25 small squares & each of these contains 16 small squares. The area covered by 25 squares is equal to 1 sq. mm.)

Platelet count/ml or cu mm=

Number of platelets counted x dilution

Volume of the fluid

Where volume of the fluid for 1 mm area=

1 x.1= 0.1 ml (cu mm)

Dilution = 200

So platelet count / cumm =

Number of platelet count x 200

0.1

=Number of platelet counted x 2000

Rough estimation of platelet count from stained thin smear

- A well – prepared peripheral blood smear can be used to check the results of direct counting. Determine the ratio of platelets to red cells on a thin blood smear used for differential leucocytes count. If the average number of platelets is 8 to 25 in 10 fields, it is reported to be adequate, & if it 0 to 5, it is reported as inadequate.

Causes of thrombocytopenia

1. Causes of platelets production failure-

   Selective megakarytocyte depression

- Drugs
- Chemicals
  - Viral infection

Part of general bone marrow failure-

- Aplastic anemia
- Leukemia
- Myleosclerosis
- Marrow infiltration, e.g. in carcinoma, lymphoma
- Multiple myloma
- Megaloblastic anemia

2. Increased destruction of platelets
    - Acute or chronic (ITP) idiopathic thrombocytopenic purpura
    - Secondary immune thrombocytopenia

      (Post- infection, SLE, CLL,and lymphomas)

3. Abnormal distribution of platelets

- Spleenomegaly

4. Dilutional loss
    - Massasive transfusion of old blood to bleeding patients

Raised platelet count -

(Thrombocytosis)

Can occur as a part of generalized myleoproleferative disorder e. g. CML or following acute haemorrhage.

Erythrocyte sedimentation rate (ESR) –

This is the rate at which erythrocytes sediments on their own weight when anticoagulated blood is held in a vertical columns, it is expressed as the fall of RBC's in mm at the end of first hour

Methods –

Wastergreen Method

1. Westerngreen's pipette is about 30 cm long with a bore diameter of about 2.5 mm
2. The lower 20 cms are marked from 0 to 200
3. Anticoagulant used is 3.8 % trisodium citrate solution. One part of anticoagulant is added to 4 parts of blood
4. The pipette accepts about 1ml of blood. Fill the pipette by sucking till the mark 0 & clamp it vertically in the Westerngreen rack

   Read the upper level of the red cells exactly after 1 hour

Normal values-

Males- 0 to 5 mm at the end of 1 hour

Females- 0 to 7 mm at the end of 1^{st} hour

Wintrobe's Method

1. The Wintrob's tube is about 11 cms long, bore diameter is 2.5 mm and the bottom 10 cms are graduated
2. Graduations are from 0 to 100 for ESR and 0 to 100 for PCV

EDTA blood is used and the tube is filled til zero mark on the top with the help of the Pasture pipette

Set it up vertically & read exactly after 1 hour

As has already been said that this tube can also be used for PCV estimation

Normal values-

Males- 0 to 9 mm at the end of 1 hour

Females-0 to 20 mm at the end of 1^{st} hour

Interpretation of ESR

The value of ESR is that it indicates the possible presence of organic disease, or to follow the course of disease

Its main use is as a prognostic tool

Rapid ESR is found in-

1. In any chronic infection
2. Any extensive inflammation
3. Pregnancy after the second month
4. Purpeuim returns to normal within 2 months
5. Active myocardial infraction
6. Acute myocardial infraction
7. Acute Rheumatoid arthritis
8. Nephrosis
9. All types of shock
10. Active syphilis
11. Postoperative status
12. Any active infectious disease, acute or chronic
13. Salphangitis, appendicitis
14. Infected necrotic or malignant tumors

15. Liver diseases
16. Menstruation

Slow ESR is usually seen in

1. Newborn infants
2. Polycythemia
3. Congestive heart failure
4. Allergic status
5. Sickle cell Anemia

Factors that plays a role in ESR

1.Plasma factor

- An accelerated ESR is favored by elevated levels of fibrinogen, & to a lesser extent of globulin
- These plasma factors cause increased formation of rouleaux which due to more weight sediments more rapidly than do single cell
- Albumin retard sedimentation
- Extreme increase in plasma viscosity
- 1`Cholesterol accelerates and Lecithin retards the ESR

2. Red cell factors

- Anemia is responsible for accelerated ESR .The change in erythrocyte- plasma ratio favors rouleaux formation
- Microcytes sediments more slowly & macrocyte somewhat more rapidly than normocyte. The sediment rate is directly proportional to the weight of the cell aggregate & inversely proportional to cell area
- Poikilocytosis retards ESR because abnormal shape hampers roulex formation

3. Anticoagulants

- Sodium citrate & EDTA do not effect ES but oxalates & heparin may.

Interfering factors –

1. The blood sample should not be allowed to stand for more than 2 hours before the test is started because rate will increase

2. In refrigerated blood the sedimentation rate is greatly increased. Refrigerated blood should be allowed to return to room temperature before the test is performed

3. Factors leading to reduced rates

- High blood sugar
- High albumin level.
- High phospholipids

- Decreased fibrinogen level level of the blood in newborn
- Certain drugs

4. Drugs.

a. That increase ESR level

- Dextran
- Methyldopa
- Methysergide
- Oral contraceptive
- Penicilamine
- Theophyline
- Trifluperidol
- Vitamine A

b.Those that decrase levels

- Ethamabutol
- Quinine
- Salicyclates
- Drugs that cause a high blood glucose level

Urine examination –

Composition of Urine –

Urine composition is affected mainly by 3 factors

1. Nutritional status
2. State of the metabolic processes
3. Ability of the kidney to selectively handle the material presented to it

Physico-chemical characteristic of urine –

Dry weight	55-70 gm/ 24 hours
Osmolality	38- 1400 mOsm /kg water

(Avarage = 500-800 mOsm / kg water)

pH	4.6-8.0 (mean= 6.1)

Specific gravity

Neonates	1.012
Infants	1.002-1.006
Adults	1.003-1.030

Volume	**Per day**
Neonates	30-60ml
10-60 days	250-450 ml
60- 365 days	400-500ml

Children

1-3 years	500-600 ml
3-5 years	600-700ml
5-8 years	650-1000 ml
8-14 years	800-1400 ml
Adults	600-2500 ml

Inorganic constituents per 24 hours

Iron	0.06-0.1 ml
Chlorides	6(4-10) gm on usual diet

Phosphate		0.8-1.3 gm on usual diet
Sulphar	2gm	
Calcium		<150 mgs

Organic constituents per 24 hours

Nitrogenous total	25-35 gms
Urea	15-30 gm
Creatinine	1.4 gms (1- 1.8 gms)
Ammonia	0.7 gms (0.3-1)
Uric acid	0.45 gms.3- 0.6 gms)
Protein (Albumin)	0-0.1 gms
Creatinine in children	10-15mg
Glucose fasting range	2-20 mg %
Amylase (diastase)	40-260 units/hour

Collection of urine

The urine sample should be collected in a clean, dry container & should be examined fresh. For cultures sterile containers should be used.With time RBC, leucocytes tend to be destroyed due to hypotonicity of urine. Casts too tend to get decomposed. Bacterial contamination of stale urine is frequent & causes alkanization of the urine due to conversion of urea to ammonia & loss of glucose

This rise in pH accelerates loss of leucocytes and epitheluial cells. For ordinary qualitative tests a random sample is enough.

Preservation of sample

Urinary decomposition occurs quickly in warm temperatures. Hence fresh specimens should be examined, if not, then it should be refrigerated. As far as possible the need for preservation should not arise.

Physical examination –

Colour and appearance

Normal urine colour is clear & pale yellow

1. Colorless-Dilution, Diabetis Melitis / Inspidus, nervousness, Diurates or alcohol intake
2. Milky pru;ent-Genito urinary tract disease, chyluria
3. Orange Urobilinogenuria ,fever ,excessive sweating ,concentrated urine
4. Red Beet root ingestion ,haematuria, haemoglobinuria, phenolphthelin, pyridium, sulphonal
5. Greenish jaundice ,phenol poisioning
6. Dirty blue or green Putrefying urine, in typhus or cholera, methelyne blue
7. Dark brown, brown red or yellow. Very concentrated urine. Acute febrile disease .Bilirubinuria
8. Brown yellow or brown red (if acidic) or bright red (if alkaline)
9. Brown, brown black or black. Haemorrhage in urinary tract if urine is acidic (acidhaematin), haemoglobinuria, porphyria, Methamoglobinuria,myoglobinuria, melanine,phenol poisioning, homogentistic acid (alkoptonuria). In porphyria ,urine turns dark brown on exposure to sun light or boiling

Reaction-

Avarage range- 4.6- 8, average pH= 6.0

Urine pH	Acidic urine
Ketosis	Diabetes, starvation, febrile illness in children
Systemic	Except with impaired renal tubular function, respiratory or Metaboilic acidosis provokes intense urine acidity and decreased NH4+ excretion

Acidification therapy

Use in treating urinary tract infections, and to prevent precipitation of calcium carbonate or phosphates or magnesium ammonium phosphate

Alkaline urine-

Post prandial- Normal finding in specimens

Alkaline tide- Voided shortly after meals

Vegitarianism- Meats produce fixed acid residue, vegetarian diet does not

Systemic alkalosis

As may occur in severe vomiting, hyperventilation, excess alkali ingestion

Urinary tract-

Proteus or Pseudomonas infection they spilt urea to HCO3 and Ammonia

Alkalization-

Used to prevent crystallization of uric acid, oxalate, cystines, sulphonamides and streptomycines

Stale specimen-

Bacterial overgrowth. If true infection exists the sediment should show pus cells

Renal tubular-Impaired tubular acidification causes inappropriately high urine pH with extreme acidosis and low serum HCO3

Odour-

Important in fresh specimens only and is aromatic because of volatile fatty acids.

Bacterial actions causes ammonical odour, while ketosis leads to a fruity odour in urine

Specific gravity-

It depends upon the concentration of various solutes in urine

1. Urinometer-

   Urine should be foamless. Transfer urine (about 70-80 ml) into the urinometer container and let the urinometer float freely without touching the sides or the bottom of container

   Read graduations at the lowest level of urinary meniscus.

2. Refractometer-

   Only small an\mount of urine is needed. It measures the concentration of solutes (related to refractive index)
3. Can be tested with Dipsticks also
4. Osmometry

   Give the most accurate assessment

Urine of low specific gravity are called hyposthenuric (< 1.007) while urine of fixed specific gravity of about 1.010 are known as isothenuric

High specific gravity

1. Excessive sweating
2. Glycosuria
3. Acute nephritis
4. Albuminuria
5. All causes of oliguria

Low specific gravity

1. Excessive water intake
2. Chronic nephritis
3. Diabetis inspidus
4. All causes of polyuria except Diabetes mellitus

Low & fixed specific gravity (1.010 to 1.012)

1. Chronic nephritis (end stage kidney) when concentration power of renal tubules is low
2. ADH deficiency
3. Atherosclerotic kidney

Urinary volume

The average 24 hour urinary output in an adult is around 1200 to 1500 ml and the night urine should not be more than 400ml urine

Polyuria

A volume more than 200ml is termed polyuria

Oliguria

Oliguria implies excretion of urine less than 500ml

Anuria

Complete cessation

Nocturia

Excretion by an adult of urine more than 500ml with a specific gravity of less than 1.018 at night

Polyuria-

- Neurotic polydypsia
- Diabetis mellitus/ inspidus
- Diuretics
- Intravenous salines / glucose
- Chronic renal failure
- Addison’s disease of adrenocortical hormones

Oligouria -

1. Dehydration

- Vomiting
- Diarrhea
- Excessive sweating
- Renal ischemia
- Acute renal tubular necrosis
- Acute glomerurinephritis
- Obstruction to urinary outflow

Turbidity

Normal value-Fresh urine is clear

Cloudy- presence of pus, RBC's or Bacteria

Alkaline urine may appear cloudy because of presence of phosphates and urine may be appear cloudy because of urates

- Pathologic urines are often turbid or cloudy
- Occasionally urine turbidity may result from urinary tract infections
- Abnormal urines may be cloudy on account of presence of red blood cells , pus cells or bacteria

Chemical examination –

Test for protein

Normal values- Negative (2-8 mg/ dl)

If urine is not clear filter or centrifuge the specimen. Both bile & protein cause urine to froth

Heat and Acid test

1. Take a test tube 2/3 rd full with urine, boil upper portion of urine for 2 minutes
2. Now turbidity can arise because of phosphates, carbonates or proteins
3. Add a few drops of 10 % acetic acid , persistence or development of turbidity implies protenuria

Sulpho – salicylic acid test

- Urine should be clear & acid
- To 1 ml of urine add 3 drops of 205 sulphosalysalic acid
- Absence of cloudiness means absence of proteins
- If the turbidity persists after boiling it is due to protein
- If the cloudiness vanishes on heating and reappears on cooling, it is due to Bence John's protein]

Paper slip method

Paper stripes impregnated with bromphenol blue and salicylate buffer are dipped in urine.

Presence of protein indicated by change of colour from pale yellow to blue

Quantitative estimation of protein in urine

1. Turbimetric and chemical procedure

   Provide an accurate estimation. Colorimetric readings taken against blanks and calculations done accordingly give the result
2. Esbach's Quantitative method
3. Acidify the urine if necessary
4. Cover the bottam of the Esbach tube with pumice, fill urine till the U mark and add esbach's or Tsuchiya"s reagent till the R mark

5. Stopper the tube and invert it about a dozen times slowly
6. Set the tube vertically and read after 30 minutes
7. The tube is graduated to read in percent or grams of protein per liter at the top of sediment

Bence John's protein test

Seen in multiple myloema classically and rarely in chronic leukemia, osteomalacia, osteosarcoma, cancer metastases to bone and hypertension

Interpretation of protenuria

Minimal proteinuria (<0.5 gm /day)

1. Following exercise or in highly concentrated urine, in healthy persons
2. Fever , severe emotional, thermal stress, in otherwise healthy person
3. Postural proteinuria, young adults may pass protein while ambulatory but not at lying
4. Hypertension
5. Renal tubular dysfunction, include genetic & drug induced
6. Polycystic kidneys
7. Lower urinary tract infections
8. Haemoglobuneria with severe infections

Moderate protenuria (0.5- 3 gm / day)

1. Chronic glomerulonephritis, moderate
2. Congestive heart failure
3. Diabetic nephropathy mild
4. Pyleonephritis
5. Multiple myeloma
6. Pre- eclampsia

Marked proteinuria (>3 gm/day)

1. Acute glomerulonephritis
2. Chronic glomerulonephritis, severe
3. Lipoid nephrosis
4. Severe diabetic nephropathy-----other causes of nephritic syndrome
5. Renal amylodisis
6. Lupus nephritis

Tests for Glucose

Normal values

Random –Negative

24 hours specimen- 100 mg/ 24 hours

Benedict's qualitative glucose test-

- The cupric iron reduced to cuprous oxide
- If only 1 % or less of glucose is present, the precipitate may not appear until cooling
- To 5 ml of Benedict's qualitative reagent add 8 drops of urine (0.5 ml)
- Heat it boiling water bath for 5 minutes or else boil it over a flame for 2 minutes

Read as follows

Blue to cloudy

Green colour-	Negative o
Yellow- green	+ (< 0.5 % glucose)
Greenish yellow	+ + (0.5 – 1 % glucose)
Yellow	+ + + (1-2 % glucose)
Orange to brick red	+ + + + (over 2 % glucose)

Benedict's Quantitative test

Piece a small quantity of powdered pumice, 10 gm of anhydrous sodium carbonate and 25 ml of quantitative Benedict's reagent in a 250 ml container & heats

While the mixture is boiling, add urine rapidly from a burette until the blue colour begins to fade, then add urine drop by drop until all blue colour is gone & only a blue colour remains

At this point, all cupric iron originally in solution is reduced

The amount of urine used contains 0.05 gm of glucose

To calculate grams of glucose per 100ml of urine divide 5 by the number of ml of urine used

Significance of sugar in urine

- Glycosuria with Hyperglycemia

- Diabetes Mellitus

Other Endocrine disorder

- Acromegaly
- Cushing syndromes
- Hyperthyroidism
- Pheochromocytoma

Pancreatic disease

- Cystic fibrosis- advanced stage,
- Haemochromatosis
- Severe chronic pancreatitis
- Carcinoma

CNS dysfunction

- Asphyxia
- Tumors or haemorrhage
- Especially of hypothalamus

Massive metabolic derangement

- Severe burns
- Uremia
- Advanced liver disease
- Sepsis
- Cardiogenic shock

Drug induced

Corticosteroids & ACTH

Thiazides

Oral contraceptives

Glycosuria without hyperglycemia

Renal tubuklar dysfunction

Pregnency (differentiate from gestational Diabetis)

Nonglucose sugars in urine

Galactose

Detecting galactosaemia in newborn period may prevent irreversible liverand CNS damage

Galactose spills into urine if only milk is being taken

Fructose

Essential fructosuria (rare)

Pentose

Very high fruit intake may cause pentosuria in normal persons

Haematuria (Blood in urine)

Haematuria can be gross, urine appears reddish due to blood, it can also be microscopic, when it is not visible by naked eyes, here various tests are performed for confirmation

1. Guaic test

In one test tube, mix 2 ml of 10 % acetic acid, 5 ml of urine and 5 ml of ether.

In a second test tube place 5 ml 95 % alcohol, 2 ml fresh Hydrogen peroxide and a pinch of powdered guaic.

Now pour the guaic solution slowly done the side of the first tube. Blood I urine causes blue colour to appear at the zone of contact between the guaic & ether

Ether

2. Benzidine test

Saturate 2 ml of glacial acetic acid with beneficing and pour off the clear supernatant fluid

Add 1 ml of fresh, hydrogen peroxide and 2 m,l of urine

3. Paper stripe test

Blood reacts with the peroxide – orthotolidine reagent to produce a blue colour

Causes –

a. Bleeding diathesis
b. Local disorders of kidney and genito- urinary tract

1. Trauma
2. cystitis
3. renal calculi

c. Genitourinary tumors
d. Heritable disorders

1. Haemoglobinopathis
2. Osler- weber- Rendu disease
3. Polycystic kidney

Nitrates or bacteria –

Normal Value – Negative for bacteria

Explanation of tests

There are two methods that are used to detect bacteria in the urine during routine urine analysis

Microscopic examination & clinical testing

Procedure

A first morning specimen is preferred because urine that has been in bladder for several hours is more likely to yield a positive result

1. Follow procedure as stated by the dipstick manufacturer

Clinical implications

1. The finding of 20 or more bacteria per high power field may indicate urinary tract infection
2. The presence of only a few bacteria should be interpreted with caution & suggests a urinary tract infection that cannot be confirmed or excluded until more definitive studies, such as cultures and sensitivity tests are performed
3. A positive result from nitrate test is a reliable indication of a significant bacteriuria and is an indication for urine culture
4. A negative result should never be interpreted as indicating absence of bacteriuria because

a. If an overnight sample was not used there have may been insufficient time for the conversion nitrate to nitrate to have occurred
b. There may be a rare instance when nitrite doesn't appear in urine , and a person of this type could have significant
c. Bacteria without the positive test
d. Some strains of urinary pathogens do not produce enzymes necessary to change nitrate to nitrite and can cause a negative result

For Calcium in Urine –

- Fasting or random samples may be tested. Before the test the patient should be on neutral low calcium diet for 3 days
- Collect 24 hours urine sample
- Mix equal parts of urine and Sullkowitch reagent, let stand for 2-3 minutes and read as under

  0= no precipitate, no urine calcium, serum calcium level 5- 7.5 mg %

  1+ =fine white cloud, normal urine and blood calcium level

  2+ & 3+= Thicker, coarser precipitate, raised urinary calcium

4+=precipitate like milk, strongly positive

Normal values

24 hours levels

100-250 mg /24 hours on & average diet <150 mg /24 hours on low calcium diet

Clinical relevances

Increase level

1. Caused by

- Hyperparathyroidism (results in constant 3+to 4+ Sulkowitch test)
- Sarcoidosis
- Primary cancers of breast & lung
- Metastasis malignancies
- Myeoloma with bone metastasis
- Wilson's disease
- Renal tubular acidosis
- Glucocorticoid excess

2. Increased urinary calcium almost always accompanies elevated blood calcium level
3. Calcium excretion greater than intake is always excessive , and excretion above 400-500mg/24 hours is reliable abnormally
4. Increased levels of calcium occurs whenever calcium is mobilized from the bone, as in metastasis cancer and prolongedskeletan mobilization
5. When calcium is excreted in increasing amounts , a potential for nephrolithasis or nephrocalcinosis is created

Decrease level

Caused by

- Hyperparathyroidism
- Vitamine deficiency (Vitamine D is essential for absorption of calcium)
- Malabsorption syndrome
- Interfering factors
- Falsely high values are seen in

a. High sodium & magnesium intake

b. Very high milk intake

c. Levels are often high immediately after meals

Drug

- Androgen’s
- Cholestyramine
- Vitamine D
- Parathyroid injection
- Nandrolone, in some cancer patient

False negative values are seen in

Increased dietary phosphates

1. Alkaline urine
2. Drugs

a. Sodium phytate

b. Thiazides

c. Viomycins

Microscopy of urinary sediment

- Use a clean, fresh morning specimen.
- Obtain urinary sediment by centrifuging urine at 3000 rpm for 5 minutes
- Draw off the clear supernatant fluid, place a drop of sediment on a glass slide and cover it with a cover slip
- Examine first under low power then under high power, vary the light intensity for seeing casts
- If protein is present ,look for casts,RBC's , puscells and epithelial cells
- A drop of methelyne blue solution can be added to the sediment and would help in identifying cellular structure and bacteria
- Study of important urinary microscopy constituents

Red Cells –

- Under high power they appear as pale disc
- If the specimen is stale, because of dissolution of Hemoglobin, then cell will appear as ghost cells
- These red cells may show crenated margins
- RBC's may be confused with oil droplets or yeast cells
- Oil droplets are variable in size and are refractile
- Yeast cells usually show budding
- Alkaline haematine stains dark purple in alkaline urine.

Neutrophylic leucocytes (pus cells)

- Unstained neutrophilic leucocytes appear as round granular 12 mu spheres, larger then RBC. these may look like small epithelial cells- let a drop of glacial acetic flow under the cover slip-the segmented nucleus of a leucocytes becomes clearer
- Epithelial cells have a single, round nucleus
- Glitter cells are larger neutrophills, cytoplasm granules may show Brownian movement

Renal tubular epithelial cells

- Unstained cells are almost the same size as that of a neutrophill but contain a large round nucleus
- Oval fat bodies are these cells containing fat globules, the nucleus, then is not visible

Bladder epithelial cells

- Unstained cells are larger than renal tubular cells, have a round nucleus nucleus and vary in size depending on depth of origin in transitional epithelium
- Superficial cells are large & flat with small nucleus

Squamous epithelial cells

- Unstained these are large, flattened cells with abundant cytoplasm and a small round nucleus
- The cell may be folded or rolled

Casts

- These are cylindrical; diameter varies according to the size of the renal tubule or duct of their origin
- The ends are usually rounded but may be flat irregular or tapered

Hyaline

- Are colorless, homogeneous, transparent

Finely granular casts

- Contain fine granules in all or in part of cast

Coarse granular casts

- Contains fat, degenerated cells or protein aggregates which appear as dark granules

Fatty casts

- Contains highly retractile globules of varying size
- Fat droplets will stain bright orange with Sudan III

Red cell cast

- Yellow under LP objectives
- If many cells are present in each casts, the matrix will not be visible

Blood casts

- Contain hemoglobin from degenerated red blood cells
- Are yellow to orange incolour, best seen with LP objective

Leucocytes count

- Contain small granular cells in a clear matrix the leucocytes may be admixed with red cells or epithelial cells
- Clumps of leucocytes may sometimes look like casts

Tubular epithelial casts

- Resemble leucocytes or mixed cell casts
- They often appear as two rows of cells in a narrow cast

Waxy casts

- Are yellow and homogenous, have sharper outlines than hyaline casts with irregular ends & cracks

Detailed study of important urinary microscopy constituents

Red cells & red cell casts

Normal values of RBC's

1-2 /LPF (Low powered field)

0-1 /HPF (high powered field)

Red cell casts - Nil (zero)/ LPF

In healthy subjects red cells are only occasionally found in urine but persistent findings needs to be investigated

RBCs are studied under high power

Clinical relivances

Red cell casts

- Casts composed largely of RBCs are rarely found normally and indicate hemorrhage conditions of nephrons
- Red blood cells casts implay acute inflammatory or vascular disorder in the glomerulus

They may be the only manifestation of

- Acute glomerulonephritis
- Renal infraction
- Collagen disease
- Kidney involvement in sub acute bacterial endocarditis
- The usual finding in SLE is RBC casts and epithelial cell casts

Red blood cells

The finding of more than one to two RBCs per high powdered field is an abnormal condition that can indicate

- Renal systemic disease
- Trauma to kidney

Increased red cells are found in

- Pyleonephritis
- SLE
- Renal stones
- Cystitis
- Hemophilia
- Prostaitis
- Tuberculosis of urinary tract
- Malignancies of urinary tract

Red cells in excess of WBC's imply bleeding into the urinary tract as may occur in

- Trauma
- Tumors
- Aspirin consumption
- Anticoagulant therapy
- Thrombocytopenia

Interfering factors

- Increased numbers of RBC's can be found following violent exercise, a traumatic catheterization, passage of stones, or contamination of menstrual fluid
- Alkaline urine haemolysis RBC's and dissolved casts
- Many drugs can cause RBC appearance in urine
- Red cells casts may occur after strenuous physical activity and contact sports

White cells and white cells casts

Normal values

WBCs – 0=5 / high powered field

WBC casts- None (zero) / LPF

WBCs may come from anywhere in the genitourinary field. While white cell casts always come from renal tubules

Hyaline casts

Normal values

Occasional hyaline casts/ LPG may be found

These are clear, colorless casts and are formed when protein within the tubules precipitate & gels

Their appearance in the urine depends on the rate of urine flow, urine pH, and the degree of protenuria

Examine under low power

Waxy cysts

- Never seen in healthy subjects
- Seen in terminal diseases of kidney
- Chronic renal disease
- Tubular inflammation and degeneration

Granular casts

Normal values

Occasional granular cast may be seen

Granular casts results from the disintegration of the cellular material of WBCs and epithelial cells into coarse and fine particles

Crystals -

Crystals seen in normal acid urine-

1. Amorphous urates yellow red granules
2. Uric acid yellow or red brown irregular but usually whetstone crystals or rhomboids
3. Calcium oxalate- refractile, octahedral ‘envelope’

Crystals seen in normal alkaline urine-

1. Amorphous phosphates fine precipitate
2. Triple phosphate colourless, 3 to 6 sided prisms. Occasionly fern leaf
3. Ammonium biurate yellow brown spheres ‘throne apple’
4. Calcium phosphates stellate prisms
5. Calcium carbonate Colourless spheres or dumbbells tiny

Crystals seen in abnormal urine

1. Cystine – colorless ,refractile,hexagonal plate
2. Tyrosine: fine needles arranged in sheaves or clumps, usually yellow, silky.
3. Leucine-yellow,oil, appearing spheres with radial concentric striations
4. Sulphonamide crystals- yellow brown asymmetrical, straiated sheaves and round forms wiyhradial striations
5. Cholestrol appears as flat notched plates in acid urine, calcium oxalate and calcium hydrogen phosphatescrysals are found in neutral urine

Bacteria, fungus parasites

- Bacteria may or may not be important
- A dry film may be made by spreading a drop or two of the urine sediment on a glass slide, fixed 7 stained with gram's stain
- If bacteria are identified in an uncentrifuged specimen under an oil immersion lens, it suggests that more than 1, 00,000 organisms/ml are present i.e. significant bacteriuria
- Acid fast bacilli may be seen but urine should always be cultured as smegma also contains some acid fast bacilli
- Yeast cells may be seen in UTI (e.g. in diabetis mellitus)but yeasts are also common contaminants

Casts in urine-

Hyaline

Normal people after strenuous exercise

- Congestive heart failure
- Diabetic nephropathy
- Chronic renal failure
- Glomerulonephritis or pyleonephritis

Red calls

- Acute glomerunephritis
- Lupus nephritis
- Goodpastur's syndrome
- Subacute bacterial endocardiatis
- Renal infractions

White cells

- Acute pyleonephritis
- Interstuial nephritis
- Lupus nephritis

Epithelial cells

- Tubular necrosis
- Cytomegalovirus infection
- Toxicity from heavy metals
- Transperent rejection

Granular

- Nephroyic syndrome
- Pyleonephritis
- Glomerulonephritis
- Transparent rejection
- Lead toxicity

Waxy casts

- Severe tubular atrophy
- Renal failure
- Transplant rejection

Blood test for Serum creatinine

Alkaline picrate method

Principal

- Serum/plasma is diluted with distilled water & the proteins are precipitate by tungstic acid
- Alkaline picrate is added to a portion of protein filtrate
- The optical density of red colour is proportional to the amount of creatinine in the filtrate

Reagents

1. saturated picric acid solution
2. 10% sodium hydroxideMethod-
3. Alkaline picrate solution
4. Stock standard creatinine solution
5. standard creatinine working solution

Method

1. In a clean dry centrifuge tube place
   Plasma serum
   Distilled water
   10 % sodium tungested solution
   2-3 N- sulphuric acid

   Mix thoroughly by inversion and after a few minutes centrifuge at 2,500 rpm for 5 minutes

Calculations

Take standard reading that is nearest to the test

Using standard I

$$\frac{\text{OD Test}}{\text{OD standard}} \times 0.01 \text{ mg} \times \frac{100 \text{ ml}}{1.0 \text{ ml}} = \text{Creatinine mg \%}$$

$$\frac{\text{OD Test}}{\text{OD Standard}} \times 1.0 \text{ mg} = \text{creatinine mg \%}$$

Using standard II

$$\frac{\text{Test (reading) OD}}{\text{OD standard}} \times 0.03 \text{ mg} \times \frac{100 \text{ ml}}{1.0 \text{ ml}} = \text{creatinine mg \%}$$

$$\frac{\text{OD Test}}{\text{OD Standard}} \times 3.0 \text{ mg} = \text{creatinine mg \%}$$

Normal values (General refrence)

0.5 to 1.6 mg % of plasma or serum

Test for blood urea –

(Berthelot method)

(for quantitative estimation of blood urea)

Test Principle:

Urea is hydrolised to ammonia and carbon dioxide by Urease. Under alkaline conditions, ammonia so formed reacts with hydrochloride and phenol to form a blue coloured indophenol which is measured at 545 nm. Nitropusside acts as a catalyst during this reaction. The intensity of the coloured formed is directly proportional to the concentration of urea present in the sample.

Kit contains:

Reagent 1A : Diluent Buffer (1 Bottle)	
EDTA	100 ml
Sodium nitropusside	6 mM
Reagent 1B : Urease (1 Bottle)	
Urease	$\geq$ 4000 U
Reagent 2 : Phenol (1 Bottle)	
Phenol	2 M
Reagent 3 : Hydrochloride (1 Bottle)	
Sodium hydrochloride	0.1 %
Reagent 4 : Standard (1 Bottle)	
Urea	40 mg / dl

Preparation of working solution –

Solution 1

Reconstitute one bottle of reagent 1B with one bottle of reagent 1A. Mix thoroughly. Do not shake the mixture vigorously.

Solution 2

Dilute the contents of reagent 3 with 250 ml. of distilled eater. Store all the working solutions in dark colour bottles at 2-6^{0}C.

Storage and stability –

Reagents are stable till the expiry date mentioned on the lable when stored at 2-6^{0}C. The working solutions are stable for four months at 2-6 ^{0}C

Specimen –

Unhemolysed serum or plasma (with EDTA, heparin or oxalate). Do not use fluoride as anticoagulant. Sample should be used on the same day. If necessary, may be preserved up to 7 days at 2-8^{0}C.

Precautions –

Reagents are for in vitro use only. Avoid contact with skin or eyes. Do not pipette by mouth. Keep all the regents back in refrigerator immediately after use. Ensure that the cap of regents 4 is tightly closed after use. Do not open the regent bottles, if liquor ammonia is opened in the laboratory.

Test Procedure –

Pipette into Test Tures	Blank	Standard	Test
Sample	-	-	10µ
Standard	-	10µ	-
Solution 1	0.1 ml	0.1 ml	0.1 ml
Mix and incubate at 37ºC for 3 minutes			
Solution 2	1.5 ml	1.5 ml	1.5 ml
Mix			
Solutions 3	1.5 ml	1.5 ml	1.5 ml
Mix and incubate at 37ºC (water bath) for 10 minutes. Read the absorbance of the Test (A_T), Standard (A_s), and Blank (A_B), against the distilled water at 546 nm (530 to 570 nm) or with Green filter against distilled water.			

Calculations –

Blood Urea (mg / dl) = $\frac{A_T - A_s}{A_s - A_B} \times 40$

Normal Values –

Urea – 10 to 45 mg / dl

Serum Electrolytes

General consideration-70-95% of body weight is made up of water in normal individual. In body fluid ,water posses some important features due to polarity and hydrogen bonding properties.

1.It is powerful solvent for ionic compounds and neutral molecules.

2.On the state of disassociation of macromolecules of the cell,water has strong influence along with dilute salt solution.

3. in structre and function of the cell, it plays an important role.

4. water evaporation helps to keep moisture in skin and lung.

Body fluid distribution

Total body fluid distributed in two main parts-

a.Extra cellular fluid.

b. Intracellular fluid

A. Extracellular fluid

All the fluid outside the body cells is called as extracellular fluid

It is subdivided into

a. plasma-

It is the extracellular fluid of the blood.

b. Interstial and lymph fluid

Continous mixing and exchange of nutrients and metabolic waste products takes place through the walls of blood capillaries between intestitional fluid and plasma

c. Dense connective tissue, cartilage and bones

Due to specific dense structure and avascularity, dense connective tissues, cartilages and bones do not exchange fluid and electrolytes

d. Transcellular fluid

Salivary glands, liver ,pancreas, billary tree, thyroid gland, gonads,gastrointestinal tract,cerebrospinal fluid and fluid in the eye compartment

B. Intracellular fluid-

The fluid in the body cell is called as intracellular fluid

Intake and loss of body water

Intake of water and other fluids: 1200-1500 ml

Food : 750-100 ml

Metabolic water- 200-300 ml

Water loss from body in 4 ways

1. The skin
2. The lungs
3. The intestine
4. The kidneys

Factors affecting the distribution of body water

Water distribution in body subjected to change by the osmotic forces which direct the movement of water from one to another compartment of the body

These osmotic forces are controlled by

a. Electrolytes -

This factor influences the distribution and retention of body water. Sodium (Chief extracellular cation) potassium (chief intracellular cation) are the most important osmotic electrolytes.

b. Organic substances of large molecular weight and size (serum Proteins)

Serum protein maintains the osmotic balance between the tissue spaces and circulating blood. Albumin which hold 18ml fluid in blood stream. They act as acid and combine with blood at the normal pH of the body.They are amphoteric also

i. **Organic compound of small molecular size**

(Glucose, urea, amino acids)

They diffuse freely across the cell membrane as they they are not important in the distribution of water. If they present in large quantity , they may influence the body fluid and its pressure.

ii. Mineral metaboilism

The principal minerals elements are sodium , potassium chlorine, calcium, magnesium, phosphourous and sulphur they present 60-805 of all inorganic material of the body

iii. Sodium

This element is the the major comp[onent of cations of the extracellular fluid which is largely associated with chlorine and bicarbonates in the regulation of acid base balance

iv. Main source-

Sodium chloride in cooking andseasoning is the main source of sodium. Salted food, wheat germs, cheese, carrot, breads, eggs, milk, nuts, radish are the other sources

About 4 grams of sodium 910grms of sodium chloride0is ingested every day and about 95% sodium is excreted in urine.

v. Metaboilism-

The metaboilism of sodium is influenced by the adrenocortical steroid. With the exception of the androgens, all of the active corticosteroids increase the absorption of the sodium and chloride by the renal tubules and decrease their excretion by sweat glands, gastrointestinal tract and salivary gland.

Accompanying the retention of sodium by kidney, there is increased the excretion of potassium with extracellular sodium.

Sodium depletion Hyponatremia-

Occuirs in

a. Diminished intake of sodium
b. Genereal losss of water and sodium which is generally replaced by water
c. Massive sweating, burns and severe skin exudative skin leasions
d. Deficiencies of minerocorticoide
e. Salt losing chronic nephritis- diabetis ketoacidosis
f. Due to vomiting and diarrheoa loss of alimentary secretion

When sodium is lost from the body, the extracellular fluid becomes hypotionic, and water leaves extracellular fluid to restore plasma osmotic pressure. More water is lost from the tissue fluid. Symptoms begans to appear when the patient has lost the sodium equivalent of 4 liters of isotonic saline

Symptoms-

1. Vasoconstrictive shock
2. Intestitional dilatation, nausea, vomiting, cramps
3. The cause of death due to circulatory failure

Sodium excess(hypernitremia)

1. Excessive intake of sodium intravenously
2. Head injury with water depletion
3. Cushing syndrome

Symptoms-

1. Raised central venous pressure , peripheral odema and pulmonary odema with eventual respiratory faliure

Potassium

Potassium is the principle cation of intracellular fluid. It plays important role in maintainence of acid base balance , osmotic pressur and water retention . potassium plays an important role in several several important metabolic reactions catalysed by enzymes. It mainly affects the muscles activity mainly cardiac muscles

The normal intake of potassium in food is 4gm/day. High content of potassium is found in chiocken, dried apricots, dried peaches, bananas, organs, pineapple and potatoes etc.

Metabolism

Metabolism is controlled by mineralcorticoids. The kidney is the principle organ of excretion of potassium. It is filtered by glomeruli as well secreted by tubules. Variation of the extracellular potassium influences the activity of straiated muscle so

that the paralysis of skeletal muscle and abnormalities in conduction and activity of cardiac muscle occurs.

Elevated serum potassium (hyperkalemia)

Toxic elevation of serum potassium is observed in the case of the patient in a. renal failure

b. addison's disease

c. shock

d. advancd dehydration

e. if potassium is administered intravenously at an excessive rate

Symptoms-

The kidney is the principle organ of excretion of potassium. Variation of extracellular potassium influences the activity of striated muscle.

So the paralysis of straiated skeletal muscle and abnormalitites in conduction and cardiac muscke occurs.

Elevated serum potassium hyperkalemia

Toxic elevation of serum potassium occurs in the case of patients with renal failure, advanced dehydration and shock

Symptoms

a. Cardiac and central nervous system depression
b. Mental confusion
c. Weakness of respiratory muscles

Low potassium (hypokalemia)

Potassium deficiency in gastrointestinal losses, chronic wasting disease in malnutrition, metabolic alkalosis , prolonged IV administration of solutions which do not contain potassium, cushing syndrome

In these condition, the intracellular potassium is transferred to the extracellular fluid and the potassium is quickly removed by the kidney.

Prolong deficiency of potassium poduce damage to the kidney. During heart failure, the potassium content of myocardium becomes depleted.

Symptoms

1. Muscle weakness with irritability and paralysis
2. Tachycardia and dilatation of heart in ECG

Chlorine

It is the component of sodium chloride, the element chlorine is essential in water balance, osmotic pressure regulation and in acid base balance. In gastric juice the chloride also plays an important role in the production of hydrochloric acid

Requirement and metaboilism

The intake of chlorine is satisfactory as long as sodium intake is adequate since chloride occurs almost entirely as sodium chloride

Abnormalities of sodium always associated wioth the abnormalities of chlorine metaboilism. Hence chlorine ia always observed in deficit in condition as diarrhea, profsed sweating and addison's diseases when losses of sodium excessively.

This lead to compensative increases in sodium bicarbonate.(metabolic alkolisis)

Determination of serum sodium and potassium

Clincal significance-

Hyponytremia-

It is observed in

A. Severe prolonged diarrhea and vomiting
B. Salt losing nephritis
C. Addison's disease

Hypernitremia-

It is observed in

a. Severe dehydration
b. Diabetis inspidus
c. Salt poisioning
d. Cushing syndrome
e. Certain post renal conditions leading to the obstruction to the flow of urine

Hypokalemia-

It is observed in the condition of

a. Cushing syndrome
b. Real tubular damage
c. Metabolic alkolisis
d. Malnutrition

Hyperkalemia-

High potassium values observerd in

a. Addison's disease

b. Renal glomular disease

c. anuria and oliguria

Method- Flame photometry

Normal range-

Serum sodium- 133-148 m Eq/l

Serum potassium- 3.8-5.6 m Eq/l

Requirements-

1.test tubes- 15’’125 mm

2. dispenser or 10 ml volumetric pipette

3. 10 ml beakers or bulbs

4. 50 or 100 micro liter push button pipette

5.Flame photometer

6. specimen- serum or heparinized plasma specimen need not to be fasting one

Standards-

Mixed standards are prepared by

1. Stock standard by sodium :1000 m Eq/l . it is prepared by dissolving 5.85 grm of analar grade sodium chloride in glass distilled water and diluted to 100 ml by using the volumetric flask.
2. Stock standard by potassium- 100 mEq/l It is prepared by dissolving 0.740 gram of potassium chloride (AR0 in glass distilled water and diluted to 100 ml by using a volumetric flask.

Mixed working standards are prepared as follows

1. Sodium/ potassium120/2 m Eq/l It contains 120 m Eq of sodium and 2 m Eq of potassium per liter of distilled water.. It is prepared by 12 ml of stock standard I and 2 ml of stock standard II, in 86 ml of glass distilled water.
2. Sodium/ potassium – 140 mEq/l. it is prepared by 14 ml of stock standard I and 4 ml of stock standard II in 82 ml of glass distilled water.
3. Sodium / potassium- 160/6 mEq/l. it is preparec by 16 ml of stock standardI and 6 ml of stock standard of II in 78 ml of distilled water.

Flame photometer

A dual channel instrument, capable of quick simultaneous estimation of sodium and potassium is preferred for clinical chemistry purpose. Most are the equipments are equipeed with the facilities incorporated to select calcium in place of soidium and lithium in place of potassium.Simultaneous determination of two elements minimize sample quantity, cost of operation and operation time.

Principle

The solution under test is passed carefully under controlled conditions, as a very fine spray in the air supply to non luminious flame. In the flame the solution evaporates and the salt dissociates to give neutral ions, which emit light of the characterstic wavelenghth. The flame is simultaneously monitord by the both channels. Each channel consist of the detector which views the flame through narrow band optical filter. The photodectector outputs are connected to two independent digital; displkays which are calibered for direct concentration readout. Initial calibaration is done by using atleast three standards of different concentrations

Important components of the equipment

1. Main unit and compressor unit
2. A. main unit consist of a. an atomizer b. mixing chamber c. burners d. optical filters e. photodetectors f. two diugital displays g. air regulator h. gas regulator i.Gas gause
3. The compressor unit delivers oil free compressed air to the automizer
4. The automizer and flame are most important component in the flame photometer.The function of the autiomizer is to break up solution into the fine droplets so thatr the atoms will absorb the heat energy from the flame and become excited.
5. The gases used for flame photometry are a. Mixture of Hydrogen and Oxygen b. Natural gas c.Acetelyne and propane with air and oxygen d. liquid pertroleum gas (LPG)
6. Fix and transfer to beakers or bulbs for the flame photometric determination

Operatiuon of the plain photometer

a. Put the main switch on
b. Put on air compressor and adjust the required air pressure by adjusting the knob meant for gas.
c. Adjust the proper filter for the simultaneous determination of sodium and potassium
d. Make zero adjustment by introducing the distilled water.

e. Introduce the standard 120/2 and by using the knob meant for sodium the digits 120.0 and by using the knob meant for potassium the digits 2.0 are adjusted
f. Introduce the standard 140/4.0. If the standards are exactly prepared then the digital display will give the exact concentration of sodium and potassium
g. Introduce the standard 160/6 and confirm the accuracy of standard
h. Now introduce the test and record the readings for sodium and potassium

Determination of chlorides

Clinical significance

Low chloride values are observed in the condition such as

a. Prolonged vomiting
b. Salt loosing renal disease
c. Burns
d. Over hydration

High chloride values are observed in the condition such as

a. Dehydration
b. Renal tubular diseases
c. Condition causeing decresed blood flow

Method;

Schales and schales

Normal range

1. Serum chloride95-106 mEq/l

Principle of the method

The proein free filtrate of the specimen is titrated with mercuric nitrate solution in the presence of diphenyl carbazone as an indicator. The free mercury ion combines with chloride ions to form soluble but nonidodised mercuric chloride

After all chloride ions have reacted with mercury ions, any excee mercuric ions combine with the indicator diphenylcarbazine to form blue violet coloured comples. Color change of the reaction mixture is considered as the end point of the filtration

Requirements

a. Test tubes-15 x12.5 mm
b. 0.2 ml and 2ml graduated pipette

Reagents

a. Mercuric nitrate reagent: dissolve 2.9-3.0 of mercuric nitrate in about 800 ml of the distilled water, add 20 ml of 2N nitric acid and make up the one liter. It is stable at the room tempreture in an amber color bottle
b. Diphenyl carbozone indicator: 100 ml/dl in955 ethanol. It is stable in an amber color bottle at 2-8degree tempreture
c. Chloride standard:100mEq/l it is prepared by dissolving 5.85gm of the analar grade sodium chloride in one liter of glassdistilled water it is stavble at 2-8 degree
d. Additional reagents
e. 2/3 n sulphuric acid
f. 10g/dl sodium tungusten

Specimen - Serum

Procedure

Prepare protein free filtrate of the serum sample as follows

In acentrifuge tube, pipette

a. 4.0 ml distilled water
b. 0.5 ml mserum
c. 0.25 ml 2/3 NhSo4
d. 0.25 ml 10 g/dl sodium tungusten

Mix thoroughly and centrifuge at 3000 rpm for 10 minitues

Following steps

a.Pipette in a test tubes. 2.0 ml of protein free filtrate

b. add one drop of the indicator

c. Titrate against mercuric nitrate reagent

d. note the titration reading

e. dilute standard 1:10 by using glass distilled water.

f. pipeetee 2 ml of diluted standard in a test tube and titrate it against mercuric nitrate reagent, by using diphenylocabozine indicator

g. Note the titration

Precautions

Controlled quantity of Nitric acid is added to mercuric nitrate reagent, otherwise the endpoint will not be sharp

CHAPTER

5

OBSERVATION AND RESULTS

Observations and results

In mootrakrucchra patients

1. In patients of Vatik Mootrakrucchra (Mootrashmarijanya), rasakshaya and hridrava is seen predominantly, vatapradhaan prakruti patients seen noticeably.
    a) Rasa dushti, Raktashaya,Mansakshaya and medovaha srotas dushti is seen
    b) Arochaka, Ajirna, and aadhmaan is seen as annavaha srotas dushti more than one year in history (73 out of 160 i.e.46.25%).
    c) Urine microscopy shows casts (50 out of 73i.e.68.49%) and amorphous material
    d) Eosinophillia in blood seen (38 out of 73) which is significant in krimi roga(40 out of 73 i.e.54.79%)
    e) Malavashtambha seen in all patients.
    f) Serum sodium always on higher side in range but not high than normal range morning sickness and heaviness of body and puffiness seen on face in them

2. No change in level of urea and creatinine seen in patients of any type of mootrakrucchra as well as in annavahasrotas dushti more than one year

3. In asmarijanya mootrkrucchra bheda(78 out of 160i.e.48.75%),patients significantally pittashmarijanya from patients of pittashmarijanya mootrakrucchra (42 out of 43i.e.97.67%)shows calcium oxalate in urine microscopy

   Pittapradhan prakruti and vatashmarijanya in vatapradhan prakruti are seen predominatly.

4. Pittaj mootrakrucchra Pittapradhaan vatanubandhi patients seen noticeably (52 out of 160i.e.32.5%)

    a) This gives the history of significant avipaka (45 out of 52 i.e.86.53%) Amlapitta(45 out of 52 i.e.86.53%) since more than 1 year in the history

b) RBC seen in urine microscopy and occult blood (50 out of 52i.e.96.15%) seen in urine chemistry.
c) Serum potassium level seen noticeably (49 out of 50 i. e. 98%) on lower side. Urodaha, galadaha , angadaha seen in patients.

5. Kaphaja mootrakrucchra patients Kaphapradhaan prakruti seen noticeably
    a) These shows pus cells(32 out of 35i.e.91.42%) in urine microscopy, albumin traced(32 out of 35 i.e.91.42%)in urine and ESR on higher side ,specific gravity also seen on higher side
    b) Serum chloride (24 out of 35i.e.68.57%) seen lower side in normal range.
    c) Dysuria or dribbling urination especially at night time seen in these patients.
    d) In history of these patients, anannabhilasha seen with udargaurav and mandagni in all.
6. Shukraja mootrakrucchra is not noticeblely seen in this age group
7. Kshayaja, kshataja , purishaja also not found in this age group
8. Aalasya, angagaurav, trushna and irregular bowel movements are noticeably seen in the history of all mootrakrucchra patients.
9. Chardi lakshan not seen in any of the patient of Mootrakrucchra in this age group
10. Patients of annanabhilasha were 31out of 160 (19.37%), Arochak seen in 71 out of 160 (44.37%), Avipaka seen in 45% out of 160 (28.12%), Amlapitta seen in 45 out of 160 (28.12%), shoola seen in 59 out of 160 (36.87%), krimi seen in 40 out of 160 (25%)
11. Patients from vatapitta prakruti were 73(46.25%), pittavata 52(32.50%), kaphavta 22 (13.12%), Kaphapitta 13(8.12%)

In Annavaha srotas dushti

1. **Vata pradhaan pittanubandhi** patients were predominately seen in Annavaha srotas dushti which is more than 1 year.(99out of 160 i.e.61.87%)They all are having **metropia** and **squints** developed during last 1 years. In these all patients **udakavaha ,rasavaha, raktavaha, asthivaha, majjavaha srotas dushti** seen
2. 97 Out of 99 (97.97%) were having **HB% and RBC lower** than normal range
3. **Male** were seen in **higher percentage** (among 160,77 were female and 93were male patients)
4. **WBC** were seen in **normal** range in all types of annavahasrotas dushti more than 1 year patients
5. **Arochak is seen in**
    a) **vatapradhaan prakruti** patients,(62 out of 99i.e.62.62%)) and **amorphous material seen in urine** occasionally in this vikruti
    b) **Urine routine** showed **epithelial cells (52out of 99 i.e.52.52%)** as well as **epithelial casts** occasionally in **vatapradhaan annavahasrotas dushti** patients
    c) Serum electrolyte- **Na (sodium) is always** used to be on **higher side** within normal range in Annavaha srotas dushti patients of **Vata pradhanya prakruti (92 out of 99 i.e.92.92%)**
6. Chardi is seen in only 4 patients 2.5% till date as they are seen as predormal symptom in mootraghat (Oligouria, albumin + 3, stomache, dribbling unsatisfactory micturation)
7. Annanabhilasha is seen **in kapha pradhan prakruti** patients, there were Total 30 patients from kapha pradhan prakruti
    a) Kaphapiita18 and Kaphavata 12 and annanabhilasha seen in 27 patients
    b) In kapha pradhan prakruti 30 patients, 83.33% in urine routine pus cells are seen significantly (25 out of 30)
    c) Albumin traced(27 out of 30)
    d) MCHC is in the lower side
    e) Serum **electrolyte (Cl)** is seen on lower side. In 26(86.66%) patients

8. Avipaka is seen in
    a) all **pitta pradhaan prakruti** patients,
    b) 28 out of 33(84.84%) in **urine microscopic RBC is traced**,
    c) 12 out of 33 (36.37%)**calcium oxalate seen**
    d) 28 out of 33(84.84%)**occult blood traced**, **serum potassium (K+) is less**,
    e) 30 out of 33**Twakdushti seen in 89%** of patients,
    f) **Mansavaha srotas vikruti** is seen in these patients as well **basti daha** also seen noticeably

9. Amlapitta is seen in
    a) pitta pradhan prakruti patients.(32 out of 160)
    b) RBC in urine microscopy and occult blood is seen in chemistry
    c) Angadaha, mootradaha,aalasya, saruja mootrapravrutti seen seen noticeably

10. 61.25% patients were constipated

CHAPTER

6

STATISTICAL ANALYSIS

Statistical Analysis

As per observation and results with chi square and z test we calculated p value with the mx excel

Table No. 1

Parameters	Mootrkrucchra Proportion	Annavaha Srotas Proportion	Z statistic	P value	Significance
PUS	24	25	-0.15523595	0.438317654	Non Significant
EPI	32	52	-2.541027354	0.005526364	Significant
OCC BLOD	50	28	2.864459496	0.002088608	Significant
CAST	50	62	-1.406421693	0.92	Non Significant
CRYSTALS	43	12	4.593380277	2.18062E-06	Significant
PROTEIN	32	27	0.720773372	0.76447552	Non Significant
SP GR	32	27	0.720773372	0.76447552	Non Significant
ANNANA	31	27	0.580457381	0.71919689	Non Significant
AROCHAK	71	62	1.020871019	0.846342224	Non Significant
AVIPAKA	45	33	1.562432452	0.940906927	Non Significant
AMLAPI	45	33	1.562432452	0.940906927	Non Significant

P value less than 0.05% is significant and P1=P2 are non significant

As per table no 1 the p value of epithelial cells in urine, occult blood in urine is different i.e. significant in both groups (<0.05%)

The proportion of urinary pus cells , urinary cast , proteins in urine and specific gravity of urine in Annavaha srotas dushti and Mootrakrucchra Vyadhi is same i.e. nonsignificant

Annanabhilasha, Arochak, Avipaka and Amlapitta also having the non significant p value (<0.05) so in these scale also proportion is same in Mootrakruchra Vyadhi and Annavaha Srotas Dushti

Chart No. 1 - Annavaha Srotas Dushti – Gender

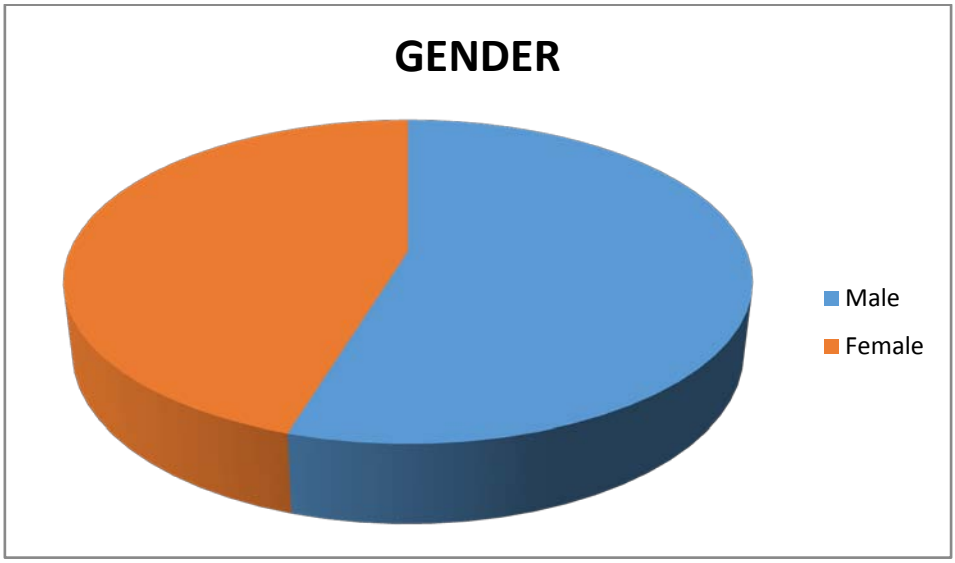

Chart No. 2 - Annavaha Srotas Dushti – Prakruti distribution

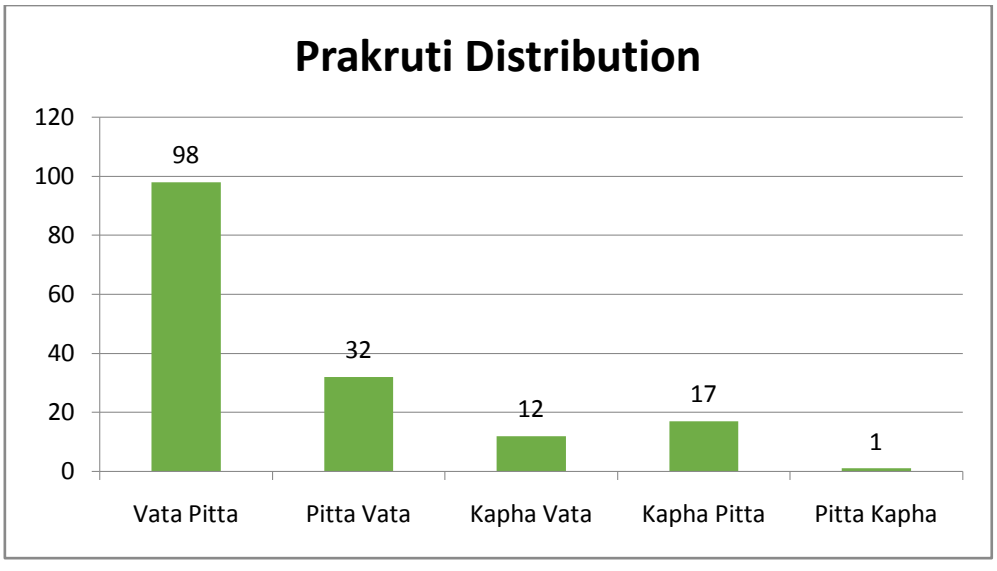

Chart No. 3 - Annavaha Srotas Dushti Lakshan

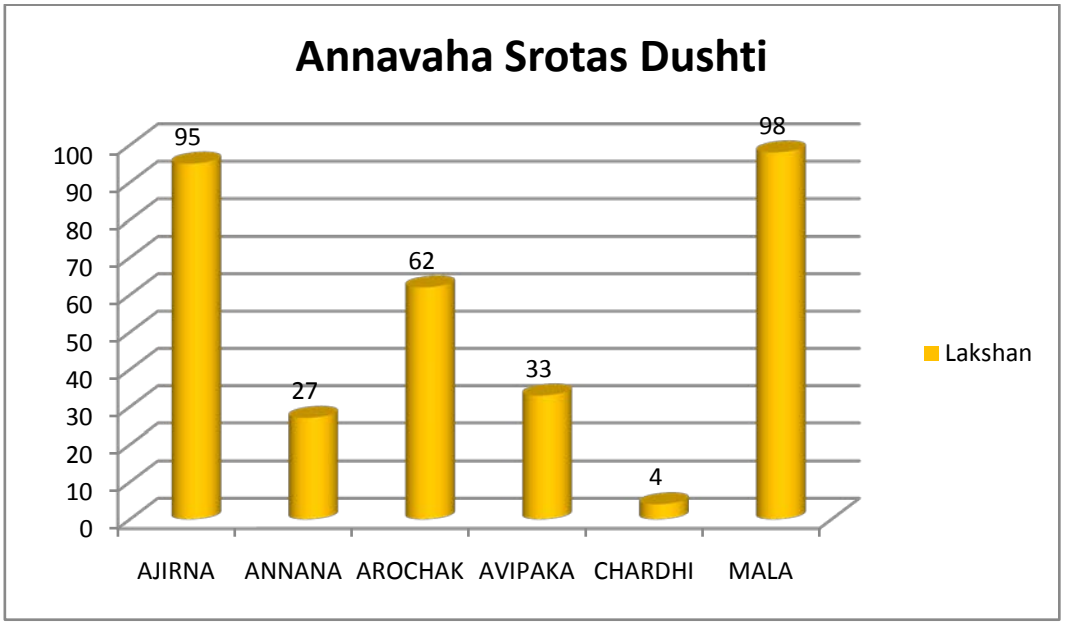

Chart No. 4 - Annavaha Srotas Dushti – Urine Analysis

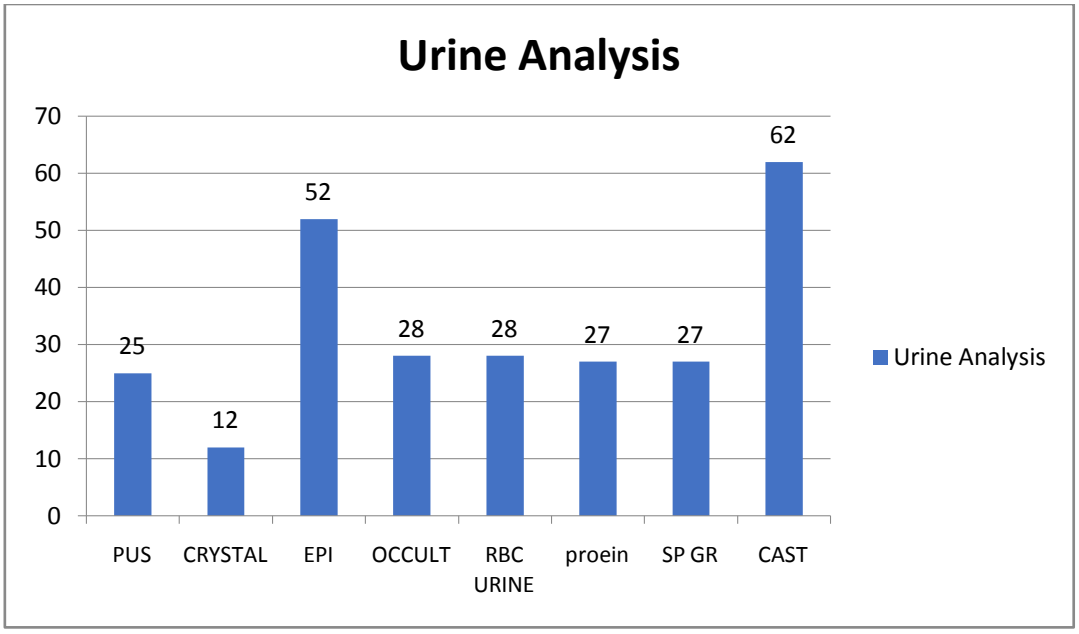

Chart No. 1 - Mootravaha Srotas Dushti – Gender

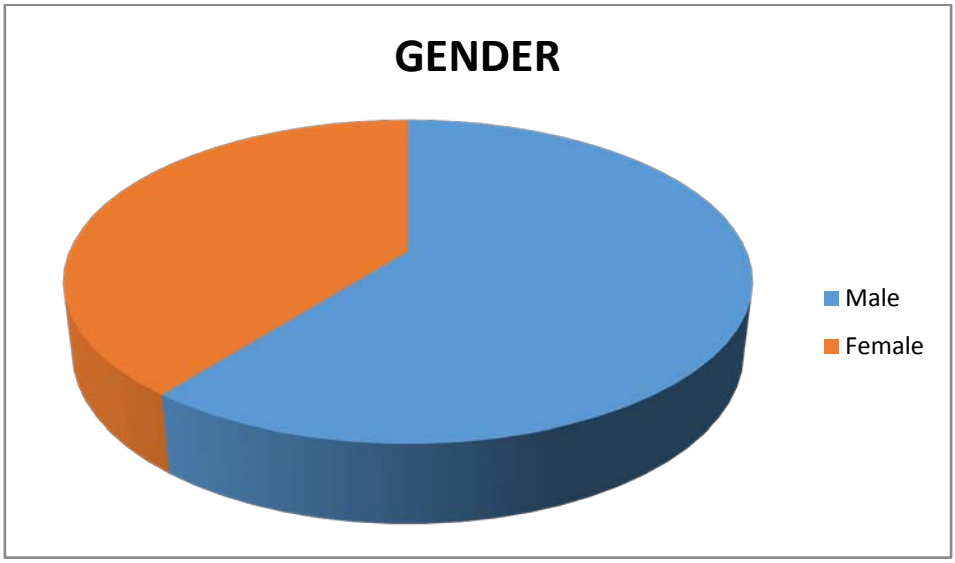

Chart No. 2 - Mootravaha Srotas Dushti – Prakruti distribution

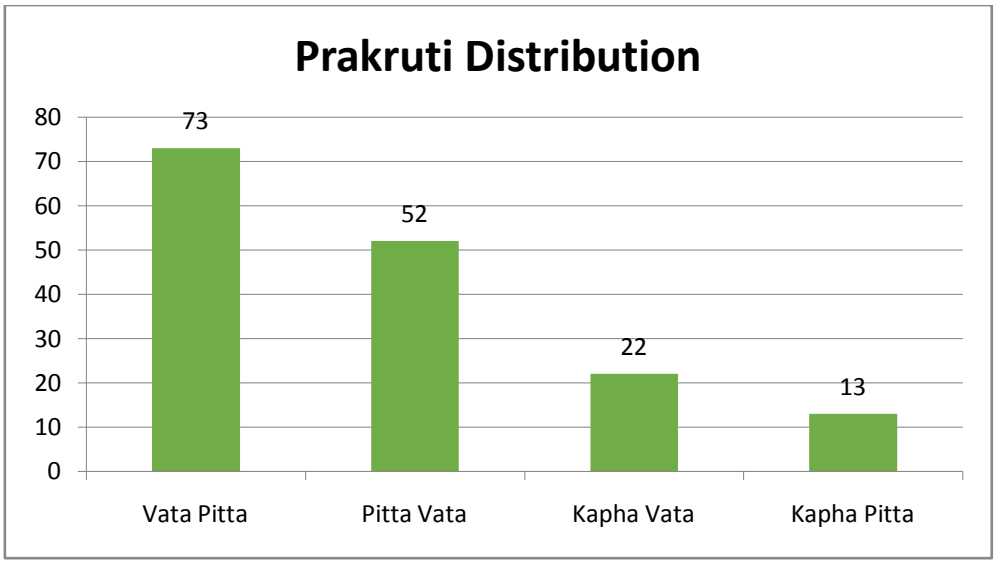

Chart No. 3 - Mootravaha Srotas Dushti Lakshan

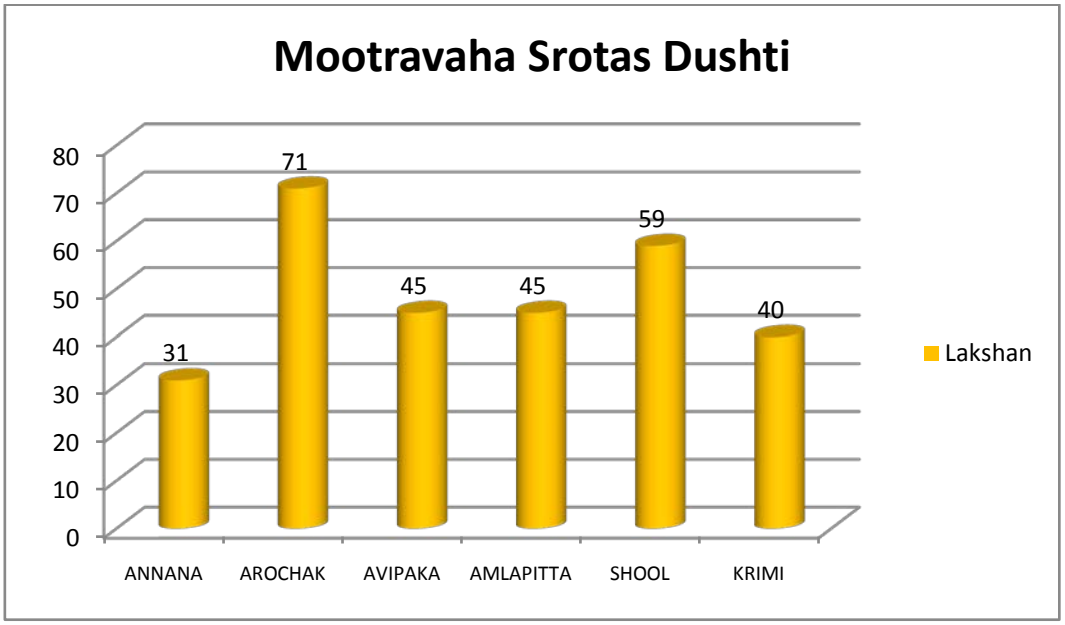

Chart No. 4 - Mootravaha Srotas Dushti – Urine Analysis

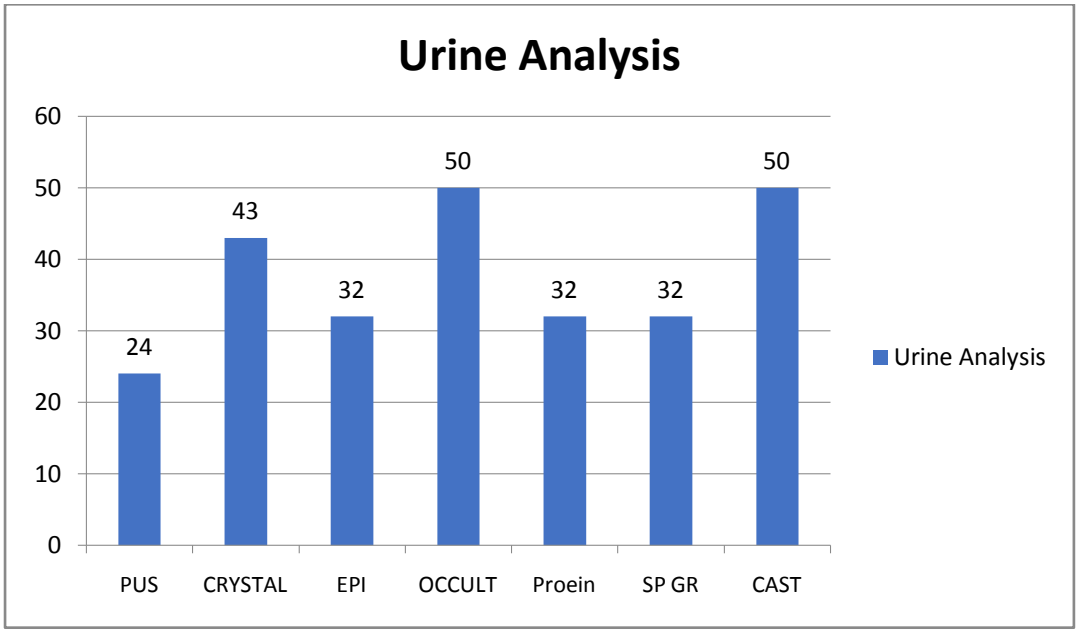

Table1: Frequency distribution of patients according to Age Groups

The frequency distribution of patients according to Age Groups is given below along with it's bar graph.

Age Groups	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
16 to 20	7	4.38	4	2.50
21 to 25	21	13.13	26	16.25
26 to 30	16	10.00	33	20.63
31 to 35	80	50.00	71	44.38
36 to 40	18	11.25	26	16.25
41 to 45	18	11.25	0	0.00
Total	160	100	160	100

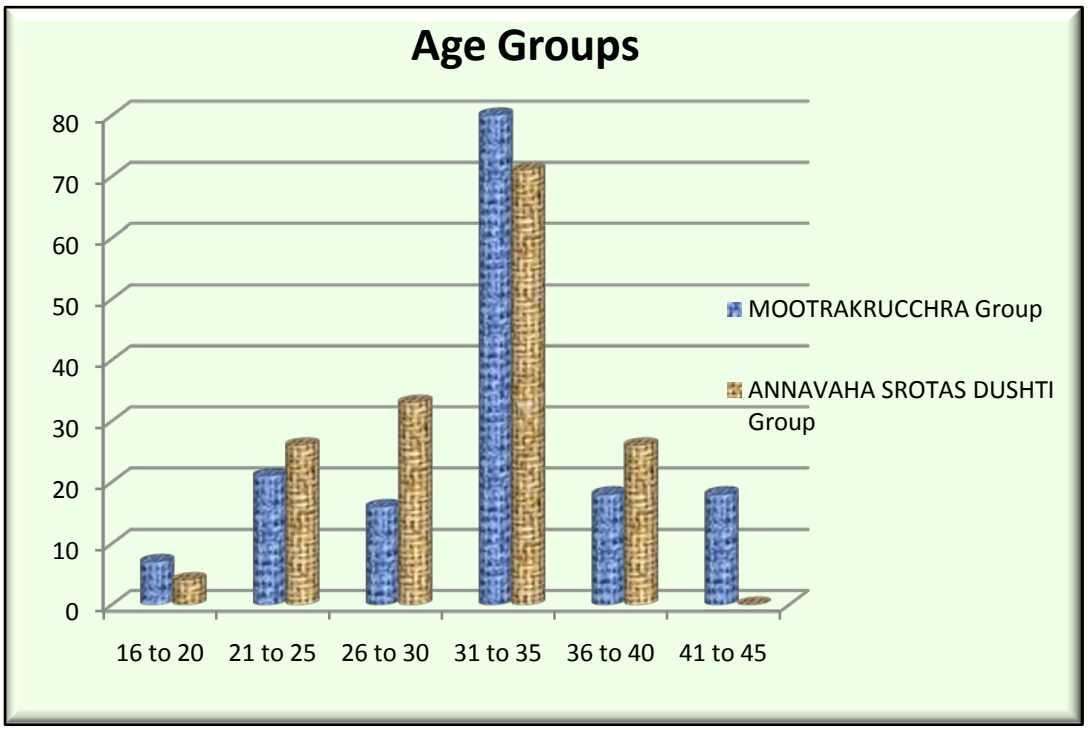

Table2: Frequency distribution of patients according to Sex

The frequency distribution of patients according to Sex is given below along with it's bar graph.

Sex	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
Female	63	39.38	77	48.13
Male	97	60.63	83	51.88
Total	160	100	160	100

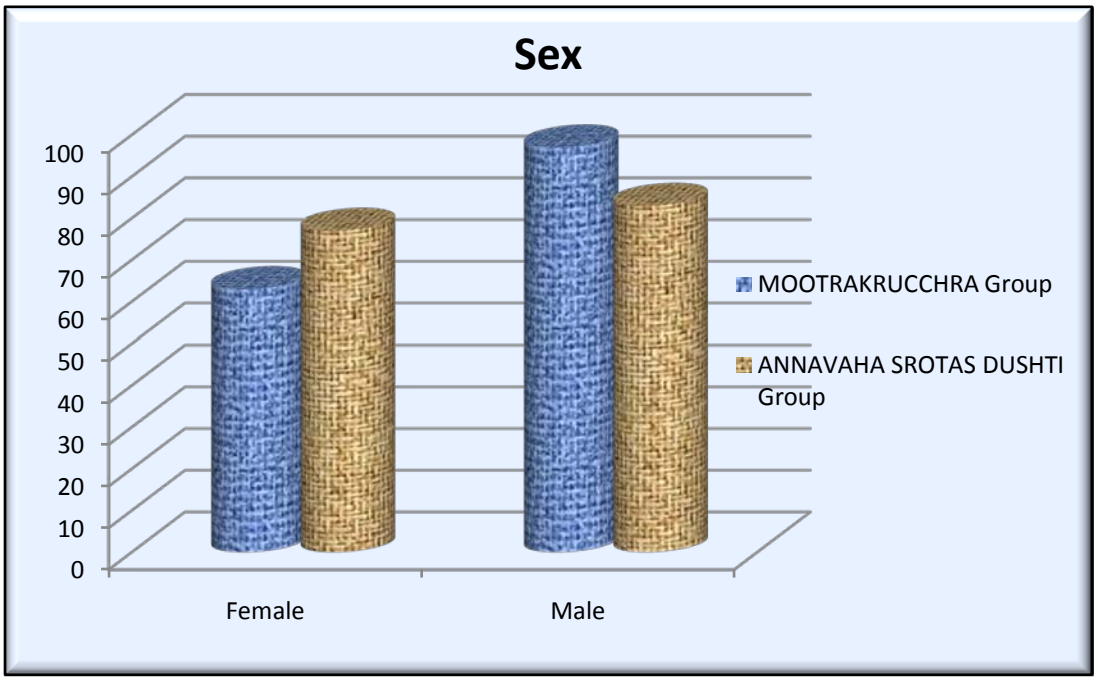

Table3: Frequency distribution of patients according to Occupation

The frequency distribution of patients according to Occupation is given below along with it's bar graph.

Occupation	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
HW	28	17.50	39	24.38
PROF	35	21.88	2	1.25
SERVICE	68	42.50	82	51.25
STUDENT	29	18.13	37	23.13
Total	160	100	160	100

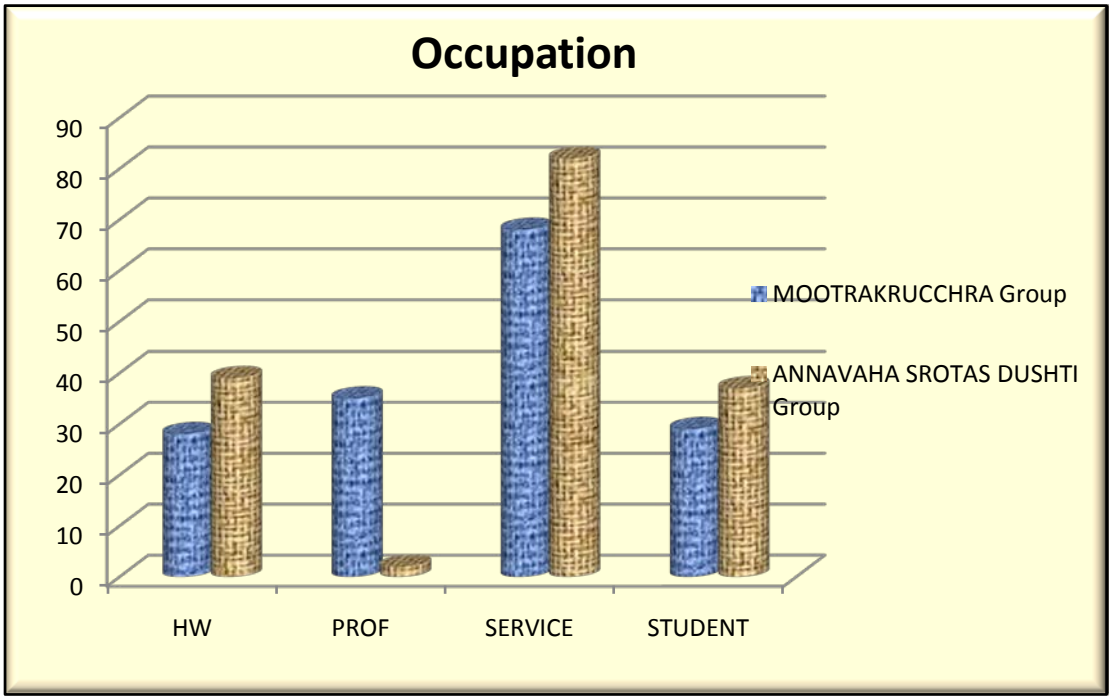

Table4: Frequency distribution of patients according to PRAKRUTI

The frequency distribution of patients according to PRAKRUTI is given below along with it's bar graph.

PRAKRUTI	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
KP	13	8.13	17	10.63
KV	21	13.13	12	7.50
PK	0	0.00	1	0.63
PV	52	32.50	32	20.00
VP	74	46.25	98	61.25
Total	160	100	160	100

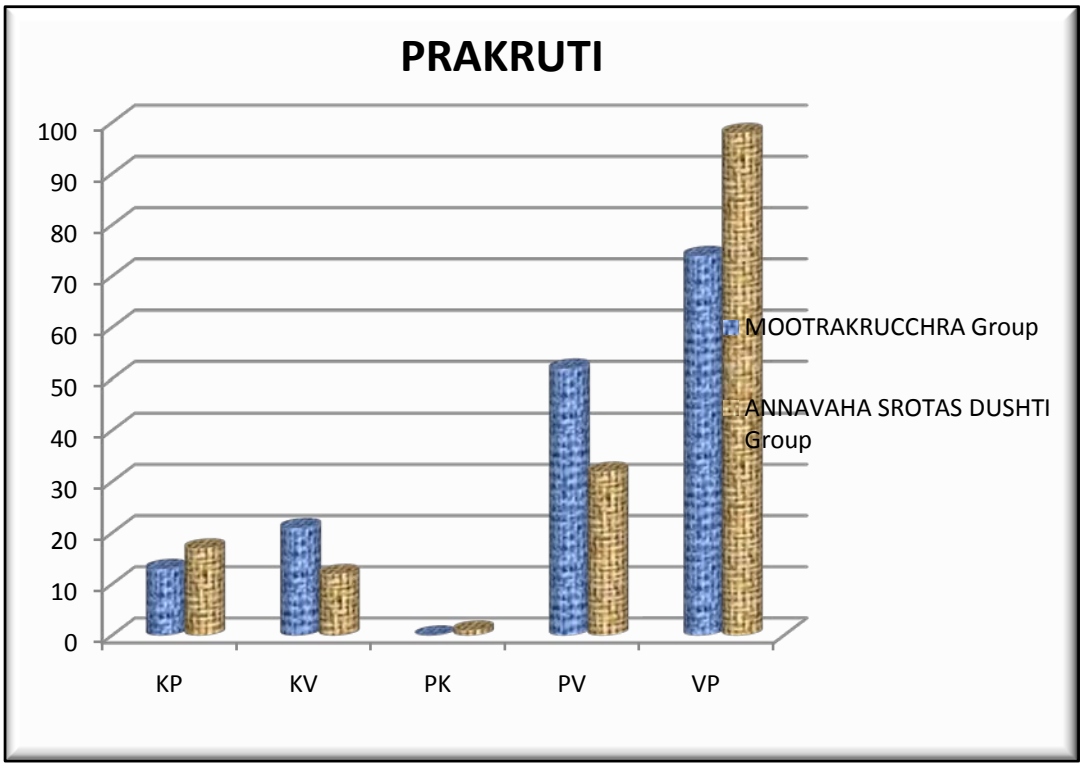

Table5: Frequency distribution of patients according to JIVHA

The frequency distribution of patients according to JIVHA is given below along with it's bar graph.

JIVHA	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
N	0	0.00	42	26.25
S	160	100.00	116	72.50
SN	0	0.00	1	0.63
SS	0	0.00	1	0.63
Total	160	100	160	100

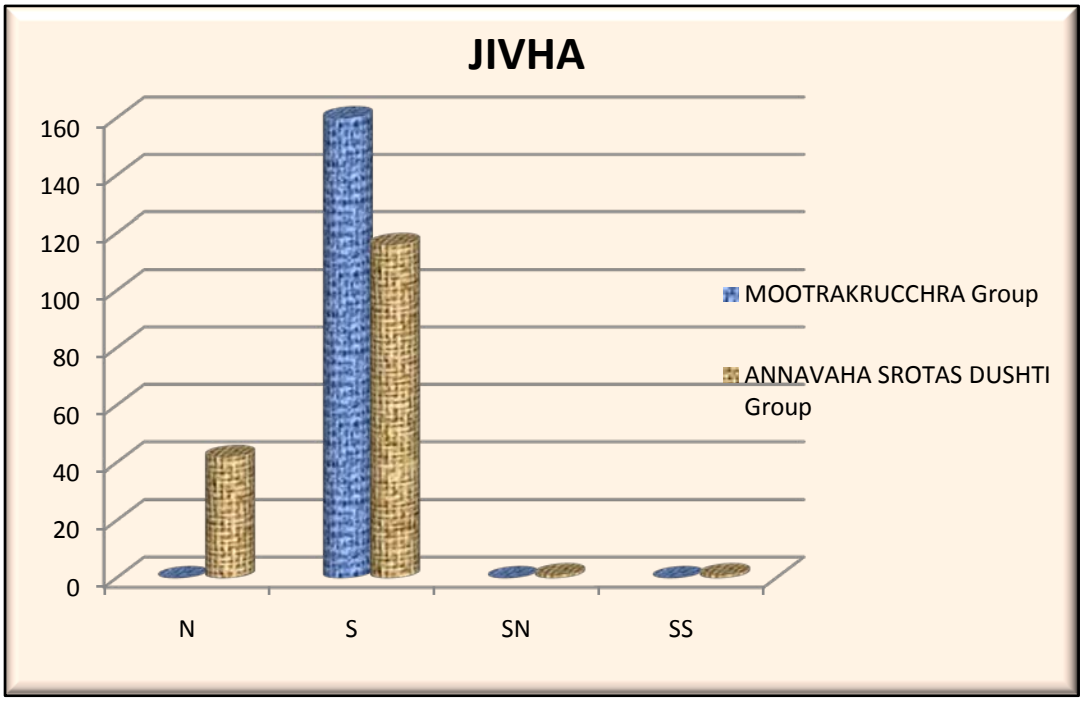

Table6: Frequency distribution of patients according to WBC

The frequency distribution of patients according to WBC is given below along with it's bar graph.

WBC	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
4001 - 5000	6	3.75	7	4.38
5001 - 6000	29	18.13	21	13.13
6001 - 7000	21	13.13	30	18.75
7001 - 8000	27	16.88	42	26.25
8001 - 9000	62	38.75	35	21.88
9001 - 10000	15	9.38	25	15.63
Total	160	100	160	100

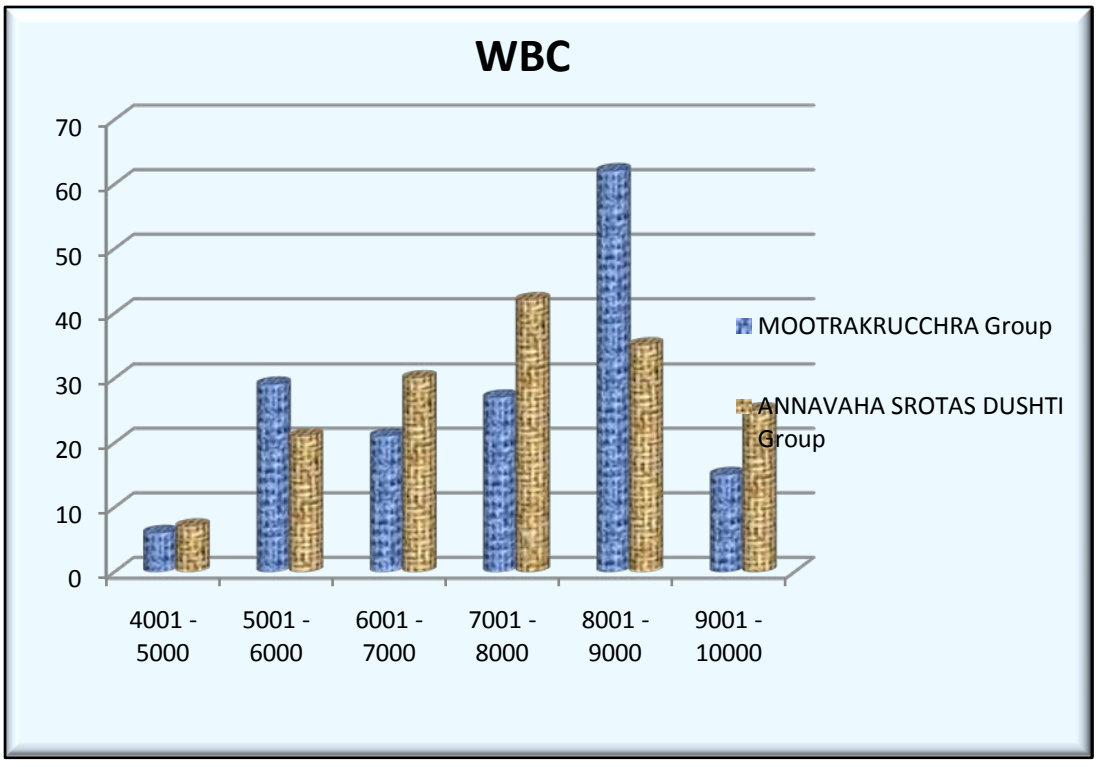

Table7: Frequency distribution of patients according to INDICES

The frequency distribution of patients according to INDICES is given below along with it's bar graph.

INDICES	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
None	96	60.00	63	39.38
MH	63	39.38	97	60.63
N	1	0.63	0	0.00
Total	160	100	160	100

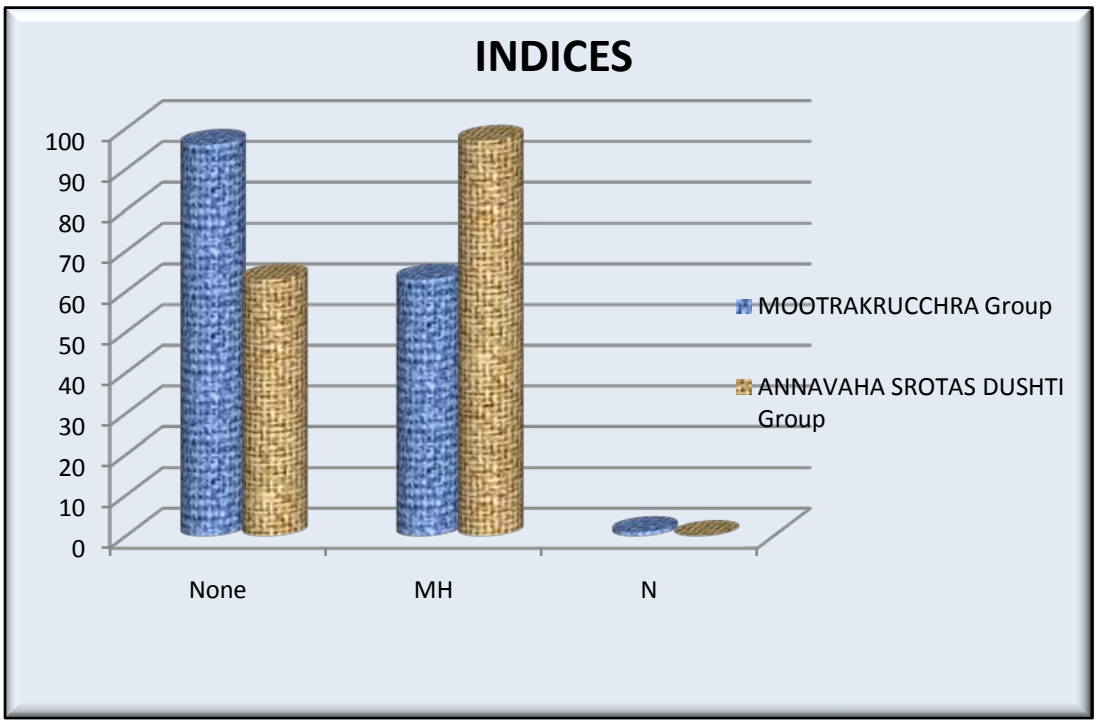

Table8: Frequency distribution of patients according to UREA

The frequency distribution of patients according to UREA is given below along with it's bar graph.

UREA	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
15 to 20	29	18.13	9	5.63
20 to 25	51	31.88	52	32.50
25 to 30	26	16.25	38	23.75
30 to 35	48	30.00	49	30.63
35 to 40	2	1.25	7	4.38
40 to 45	4	2.50	5	3.13
Total	160	100	160	100

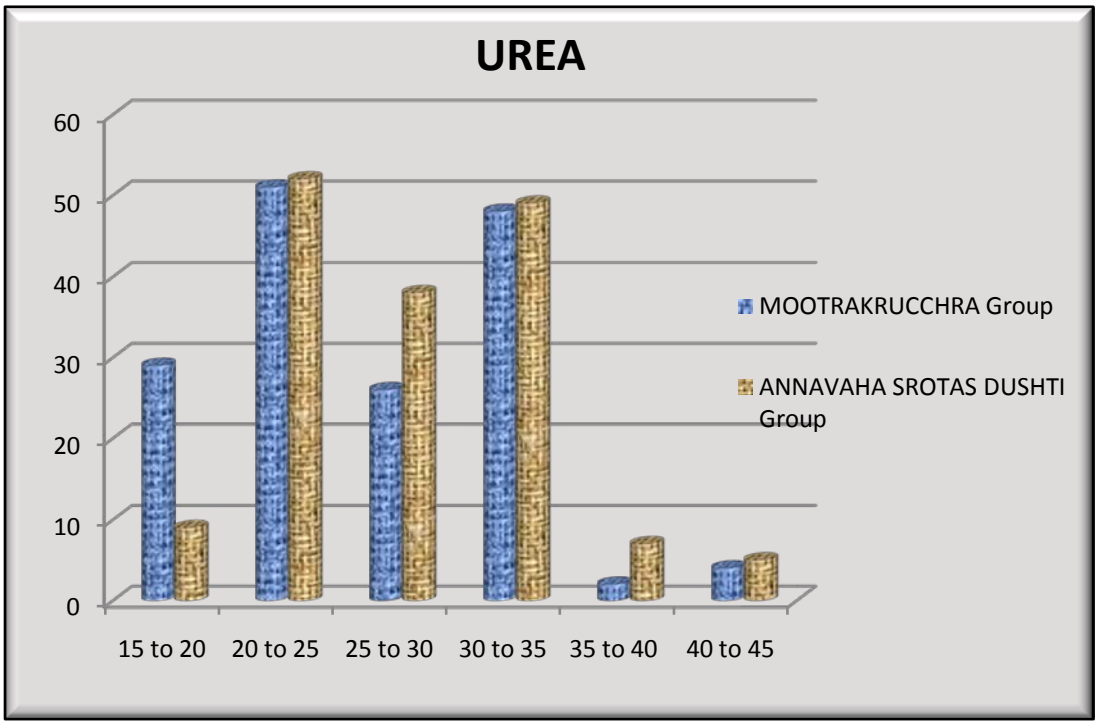

Table9: Frequency distribution of patients according to Creatinine

The frequency distribution of patients according to Creatinine is given below along with it's bar graph.

Creatinine	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
0.5 to 1.0	53	33.13	79	49.38
1.0 to 1.5	105	65.63	81	50.63
1.5 to 2.0	2	1.25	0	0.00
Total	160	100	160	100

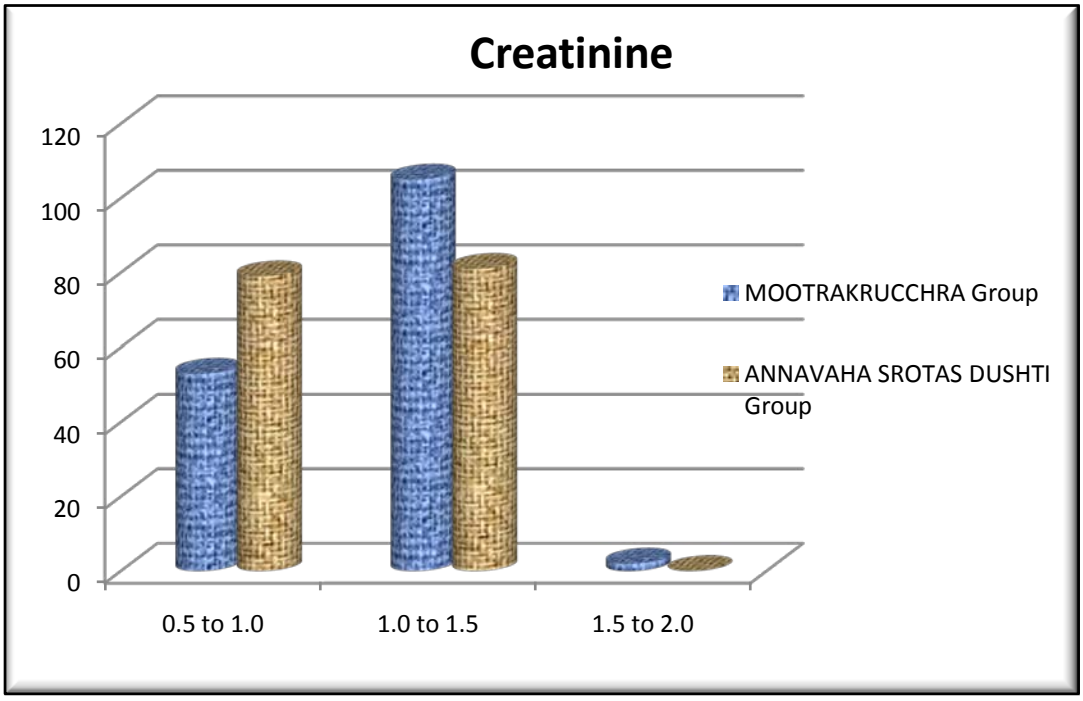

Table10: Frequency distribution of patients according to NA

The frequency distribution of patients according to NA is given below along with it's bar graph.

NA	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
Normal	87	54.38	68	42.50
130 to 135	7	4.38	0	0.00
135 to 140	27	16.88	1	0.63
140 to 145	28	17.50	51	31.88
145 to 150	11	6.88	36	22.50
150 to 155	0	0.00	4	2.50
Total	160	100	160	100

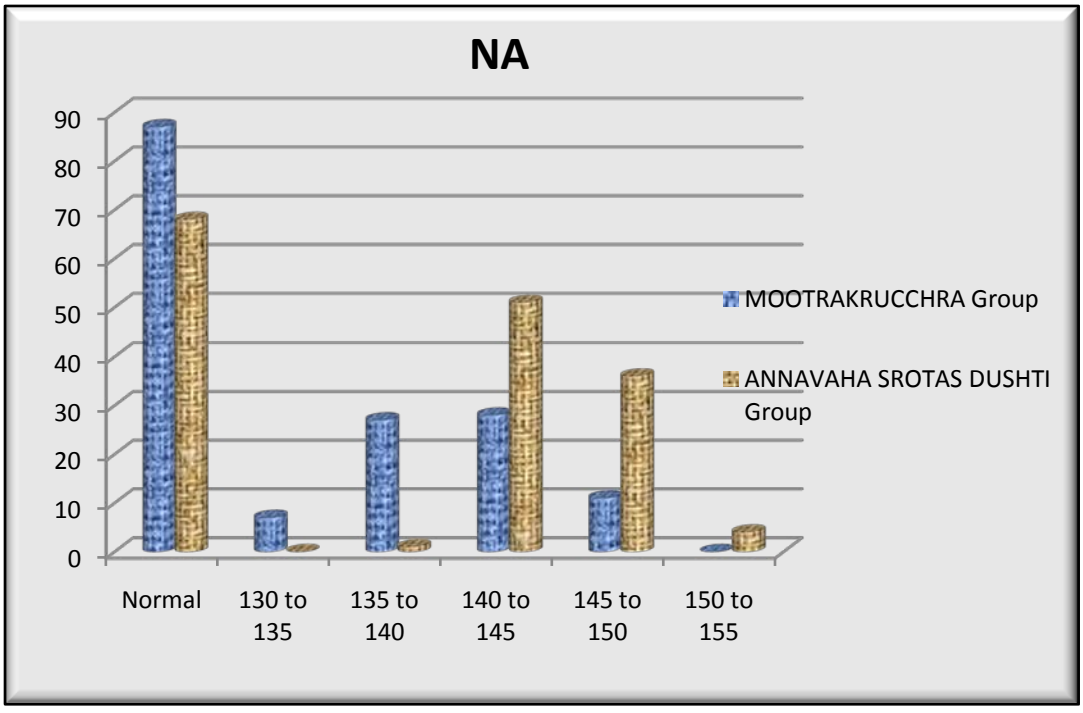

Table11: Frequency distribution of patients according to K

The frequency distribution of patients according to K is given below along with it's bar graph.

K	Groups			
	MOOTRAKRUCCHRA Group	**%**	**ANNAVAHA SROTAS DUSHTI Group**	**%**
Normal	111	69.38	130	81.25
3 to 3.5	3	1.88	2	1.25
3.5 to 4	38	23.75	28	17.50
4 to 4.5	8	5.00	0	0.00
Total	160	100	160	100

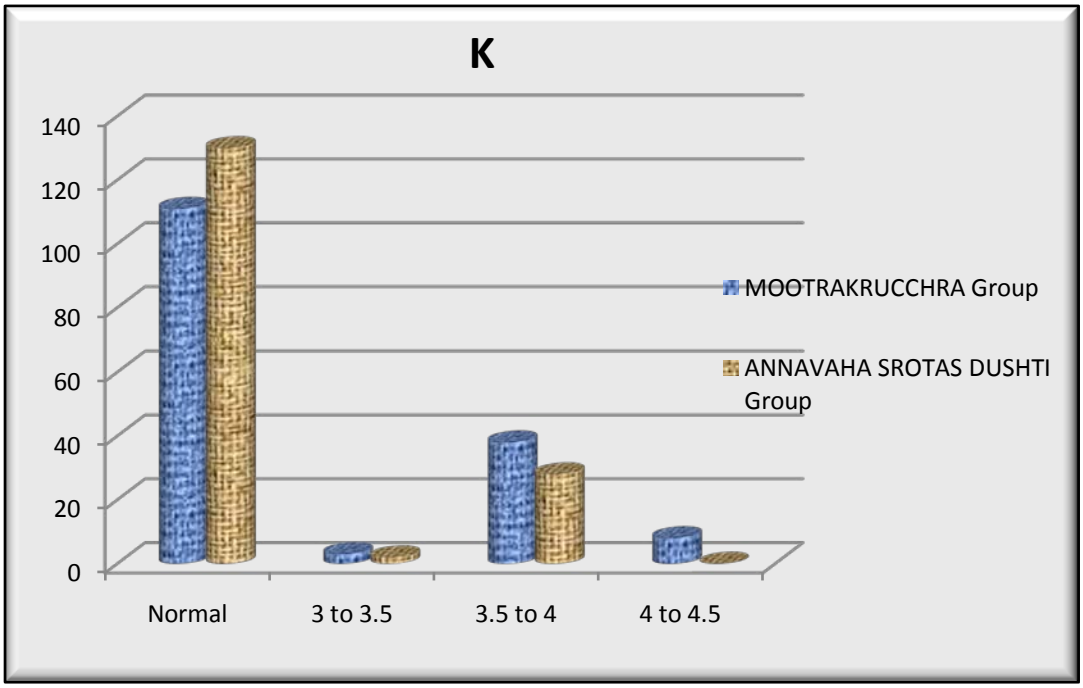

Table12: Frequency distribution of patients according to CL

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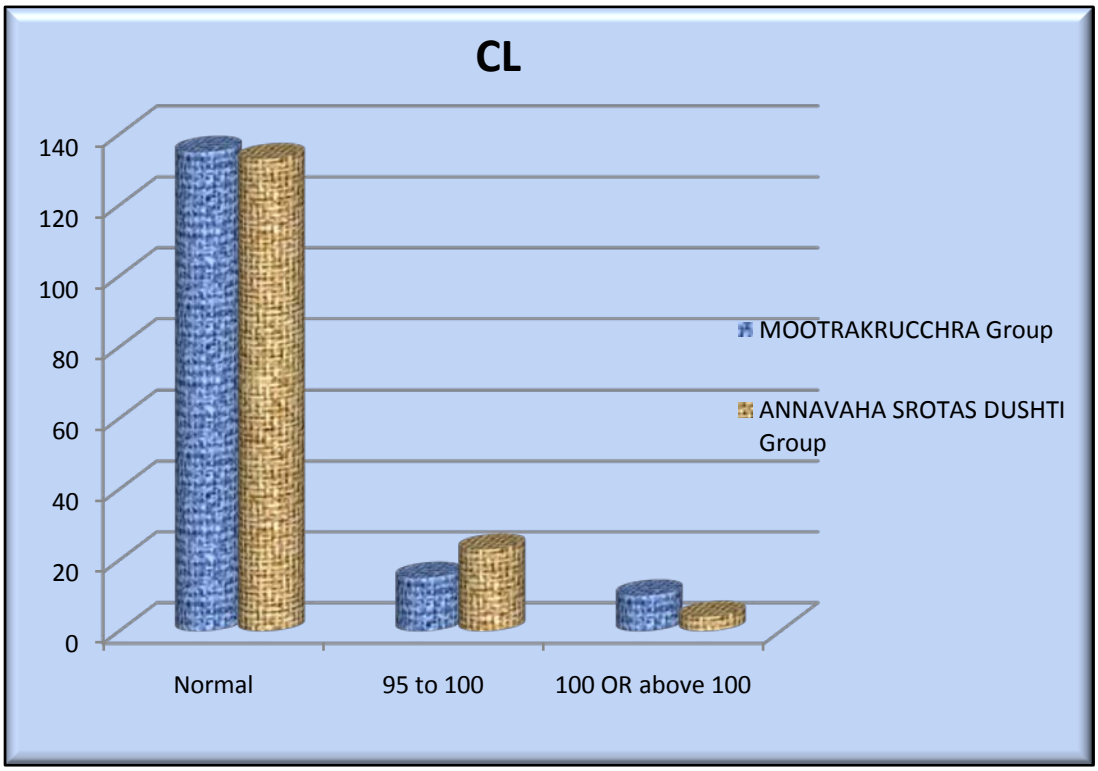
CL	Groups			
	MOOTRAKRUCCHRA Group	%	ANNAVAHA SROTAS DUSHTI Group	%
Normal	135	84.38	133	83.13
95 to 100	15	9.38	23	14.38
100 OR above 100	10	6.25	4	2.50
Total	160	100	160	100



Group: MOOTRAKRUCCHRA

Table1: Frequency distribution of patients according to TYPE

The frequency distribution of patients according to TYPE is given below along with it's bar graph.

TYPE	Frequency	%
K	35	21.9
P	52	32.5
V	73	45.6
Total	160	100.0

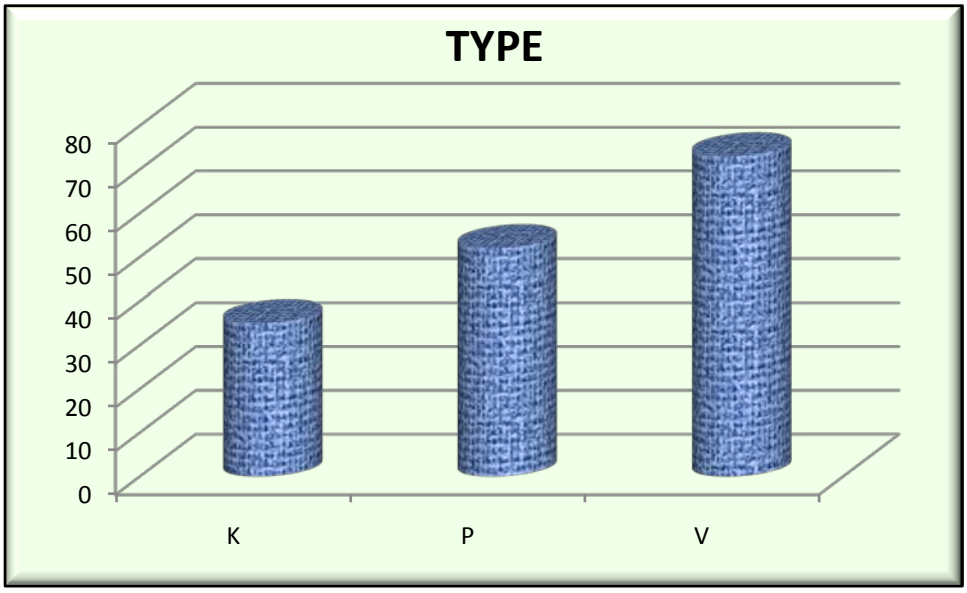


Table2: Frequency distribution of patients according to ASHMA

The frequency distribution of patients according to ASHMA is given below along with it's bar graph.

ASHMA	Frequency	%
None	82	51.3
S	1	0.6
Y	77	48.1
Total	160	100.0

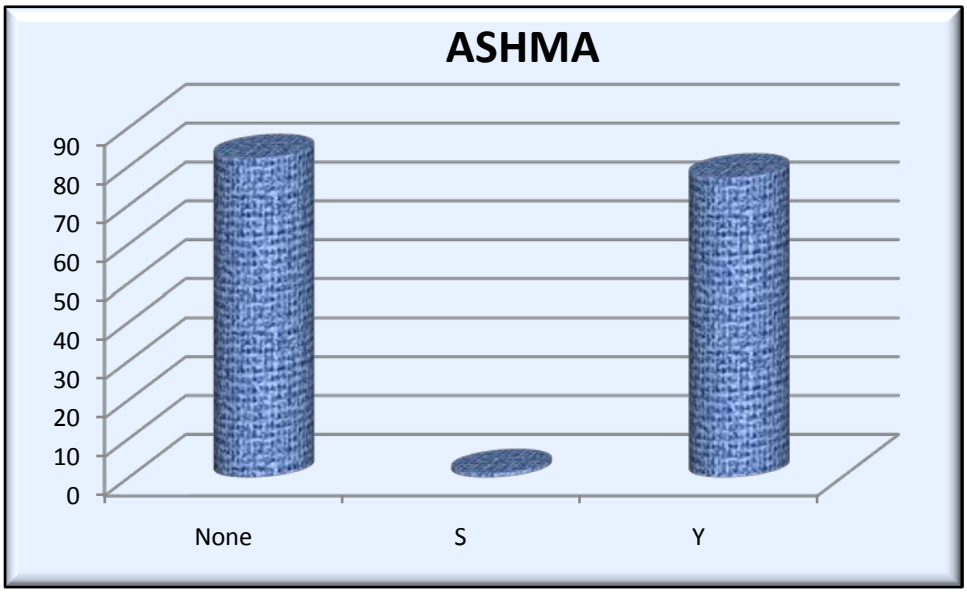


Table3: Frequency distribution of patients according to PLATELETS

The frequency distribution of patients according to PLATELETS is given below along with it's bar graph.

PLATELETS	Frequency	%
Below 1.5 lakhs	4	2.50
1.5 to 3.0 lakhs	112	70.00
3.0 lakhs & above	44	27.50

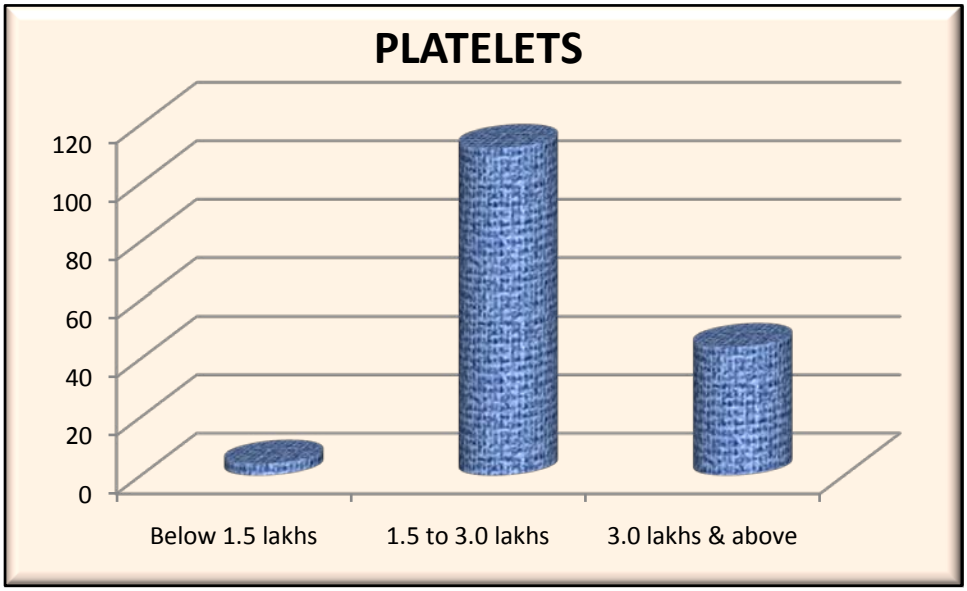


Table4: Frequency distribution of patients according to SHOOLA

The frequency distribution of patients according to SHOOLA is given below along with it's bar graph.

SHOOLA	Frequency	%
No	101	63.1
Yes	59	36.9
Total	160	100.0

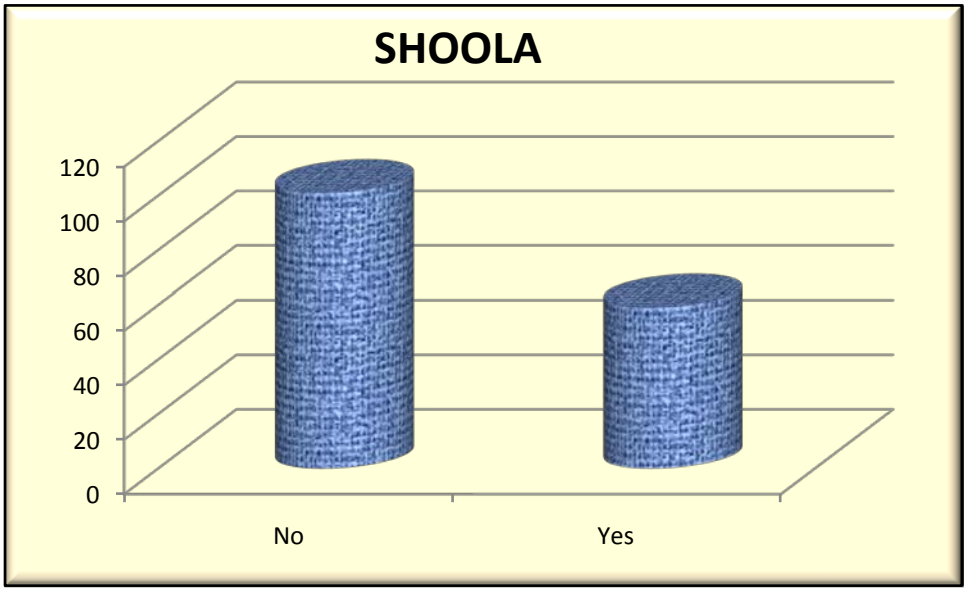
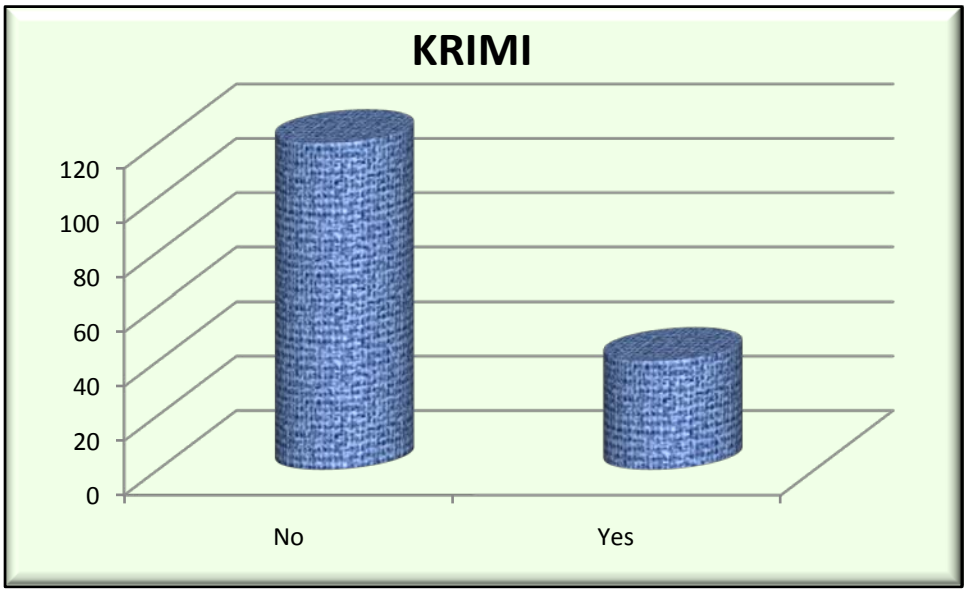


Table5: Frequency distribution of patients according to KRIMI

The frequency distribution of patients according to KRIMI is given below along with it's bar graph.

KRIMI	Frequency	%
No	120	75.0
Yes	40	25.0
Total	160	100.0



Group: MOOTRAKRUCCHRA

Table6: Frequency distribution of patients according to AJIRNA

The frequency distribution of patients according to AJIRNA is given below along with it's bar graph.

AJIRNA	Frequency	%
No	65	40.6
Yes	95	59.4
Total	160	100.0

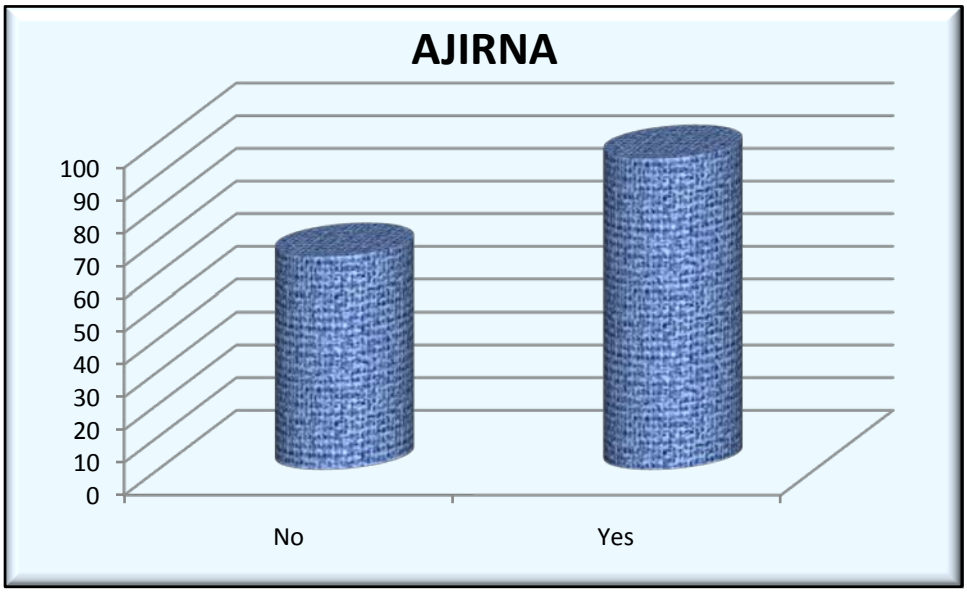


Table7: Frequency distribution of patients according to MALA

The frequency distribution of patients according to MALA is given below along with it's bar graph.

MALA	Frequency	%
No Constipation	62	38.8
Constipation	98	61.3
Total	160	100.0

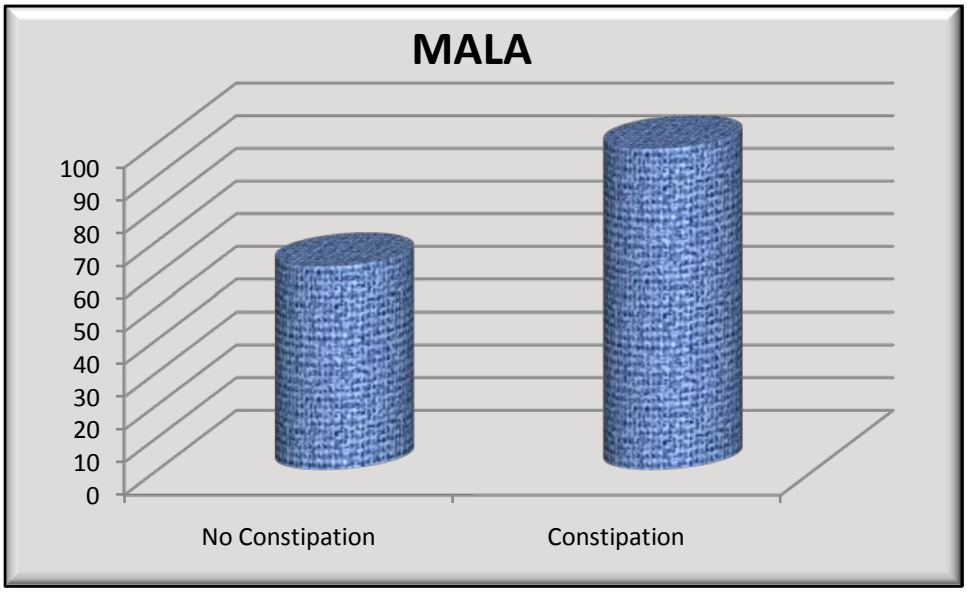


Table8: Frequency distribution of patients according to ESR

The frequency distribution of patients according to ESR is given below along with it's bar graph.

ESR	Frequency	%
Below 10	29	18.13
10 to 20	92	57.50
20 to 30	19	11.88
30 to 40	8	5.00
40 to 50	11	6.88
50 to 60	1	0.63
Total	160	100.0

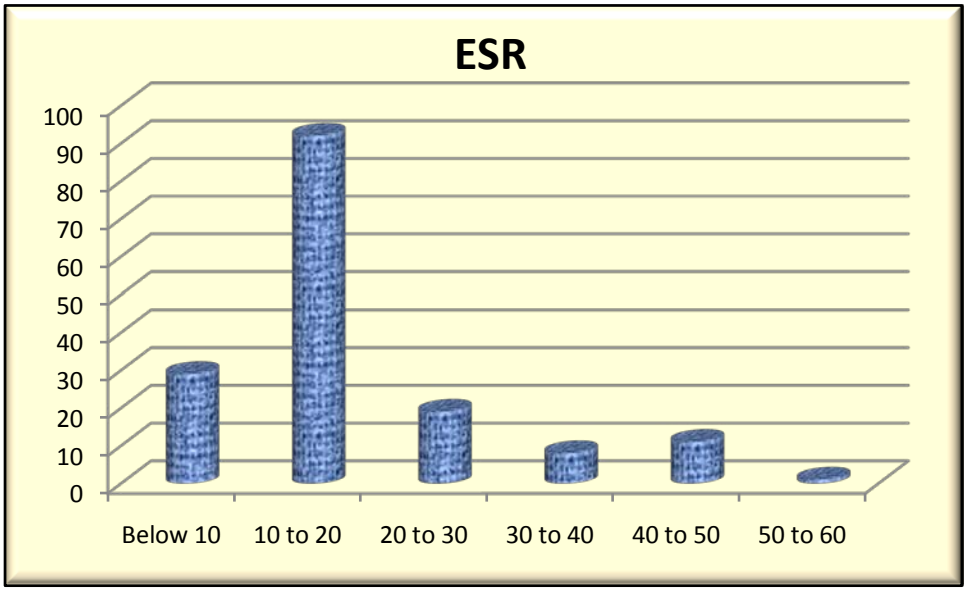
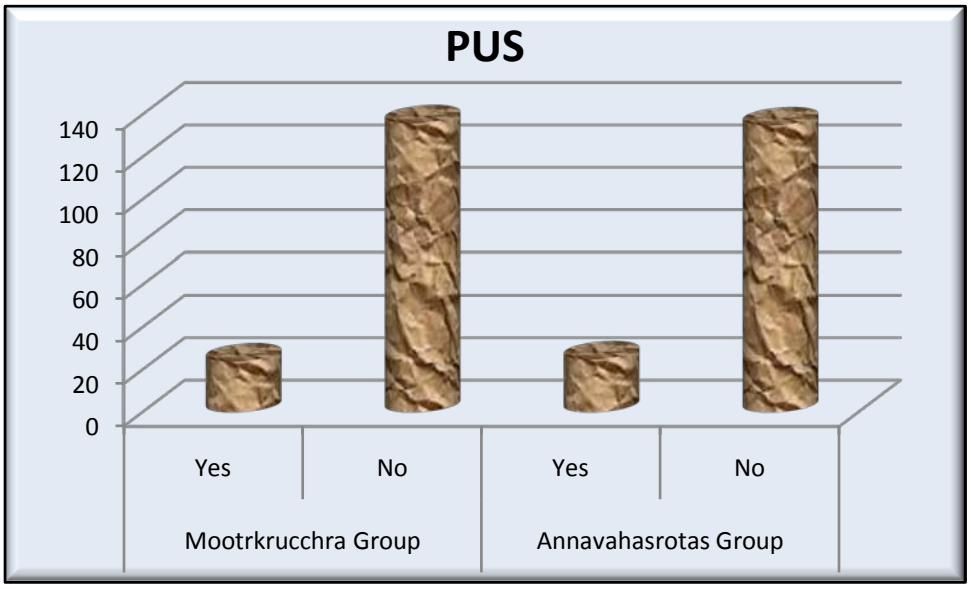


Table1: Frequency distribution of patients according to PUS

The frequency distribution of patients according to PUS in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
PUS	24	136	25	135
%	15.00	85.00	15.63	84.38



Summary:

The frequency of patients having PUS is approximately same in both the groups.

Test1: To test whether the proportion of patients with PUS is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with PUS in Mootrkrucchra Group is same as the proportion of patients with PUS in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with PUS in Mootrkrucchra Group is different than the proportion of patients with PUS in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
-0.16	0.88

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

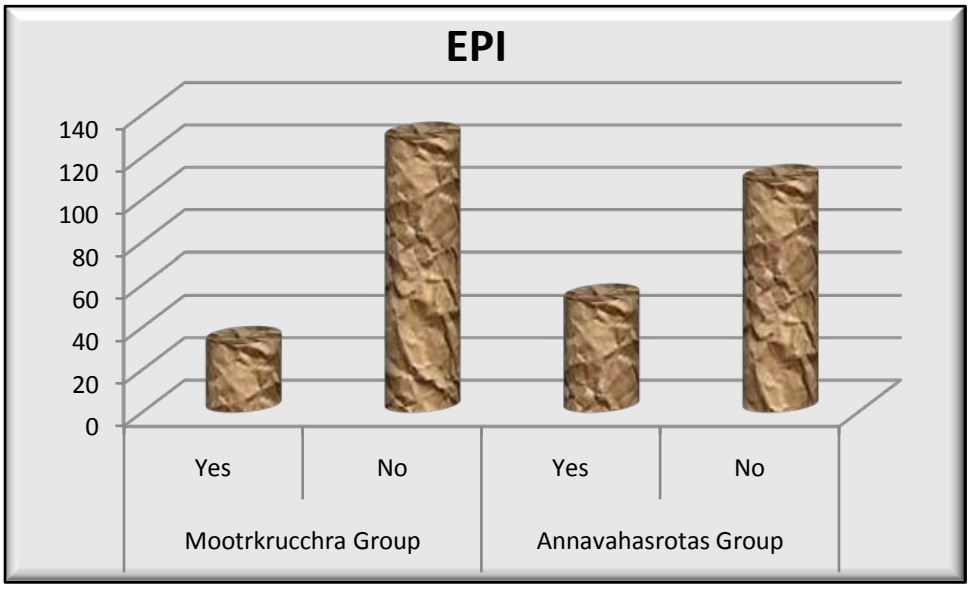
Conclusion:

The proportion of patients with PUS in Mootrkrucchra Group is same as the proportion of patients with PUS in Annavahasrotas Group.

Table2: Frequency distribution of patients according to EPI

The frequency distribution of patients according to EPI in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
EPI	32	128	52	108
%	20.00	80.00	32.50	67.50



Summary:

The frequency of patients having EPI is greater in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test2: To test whether the proportion of patients with EPI is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with EPI in Mootrkrucchra Group is same as the proportion of patients with EPI in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with EPI in Mootrkrucchra Group is different than the proportion of patients with EPI in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
-2.54	0.01

Since p value < 0.05, the level of significance; there is strong evidence to reject the null hypothesis.

Conclusion:

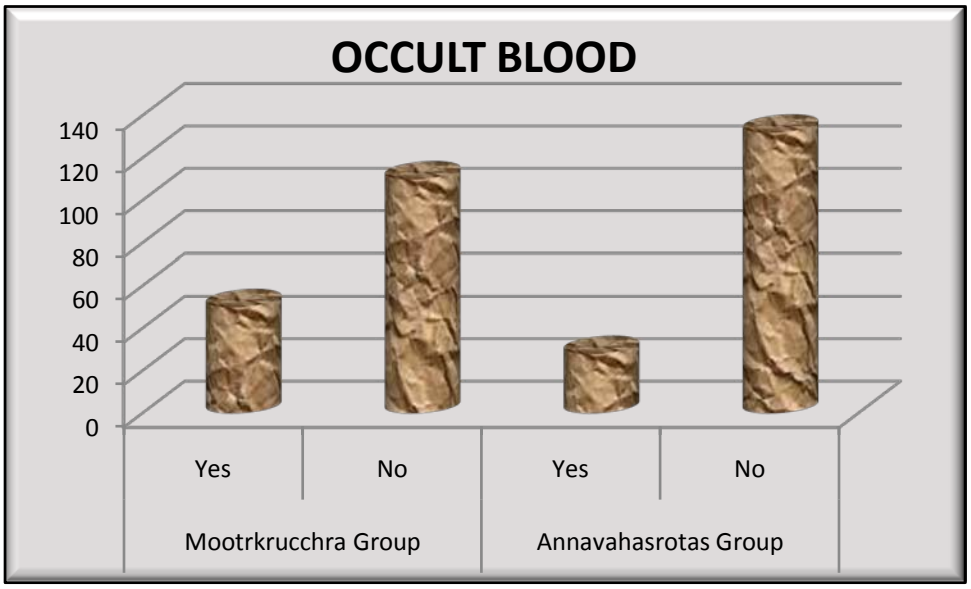
The proportion of patients with EPI in Mootrkrucchra Group is different than the proportion of patients with EPI in Annavahasrotas Group.

The proportion of patients with EPI in Mootrkrucchra Group is less than the proportion of patients with EPI in Annavahasrotas Group significantly.

Table3: Frequency distribution of patients according to OCCULT BLOOD

The frequency distribution of patients according to OCCULT BLOOD in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
OCCULT BLOOD	50	110	28	132
%	31.25	68.75	17.50	82.50



Summary:

The frequency of patients having OCCULT BLOOD is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test3: To test whether the proportion of patients with OCCULT BLOOD is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with OCCULT BLOOD in Mootrkrucchra Group is same as the proportion of patients with OCCULT BLOOD in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with OCCULT BLOOD in Mootrkrucchra Group is different than the proportion of patients with OCCULT BLOOD in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
2.86	0.00

Since p value < 0.05, the level of significance; there is strong evidence to reject the null hypothesis.

Conclusion:

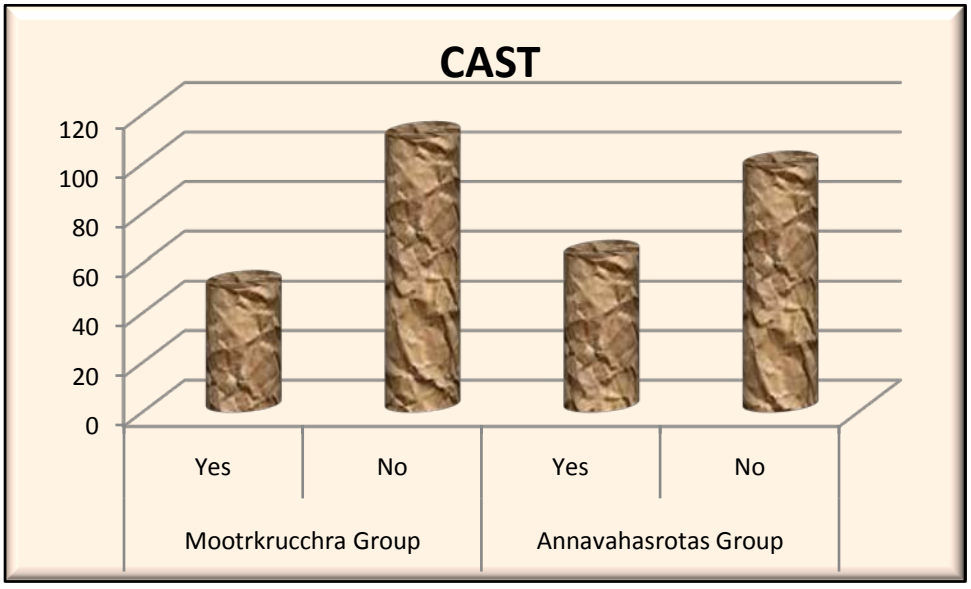
The proportion of patients with OCCULT BLOOD in Mootrkrucchra Group is different than the proportion of patients with OCCULT BLOOD in Annavahasrotas Group.

The proportion of patients with OCCULT BLOOD in Mootrkrucchra Group is greater than the proportion of patients with OCCULT BLOOD in Annavahasrotas Group significantly.

Table4: Frequency distribution of patients according to CAST

The frequency distribution of patients according to CAST in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
CAST	50	110	62	98
%	31.25	68.75	38.75	61.25



Summary:

The frequency of patients having CAST is greater in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test4: To test whether the proportion of patients with CAST is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with CAST in Mootrkrucchra Group is same as the proportion of patients with CAST in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with CAST in Mootrkrucchra Group is different than the proportion of patients with CAST in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
-1.41	0.16

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

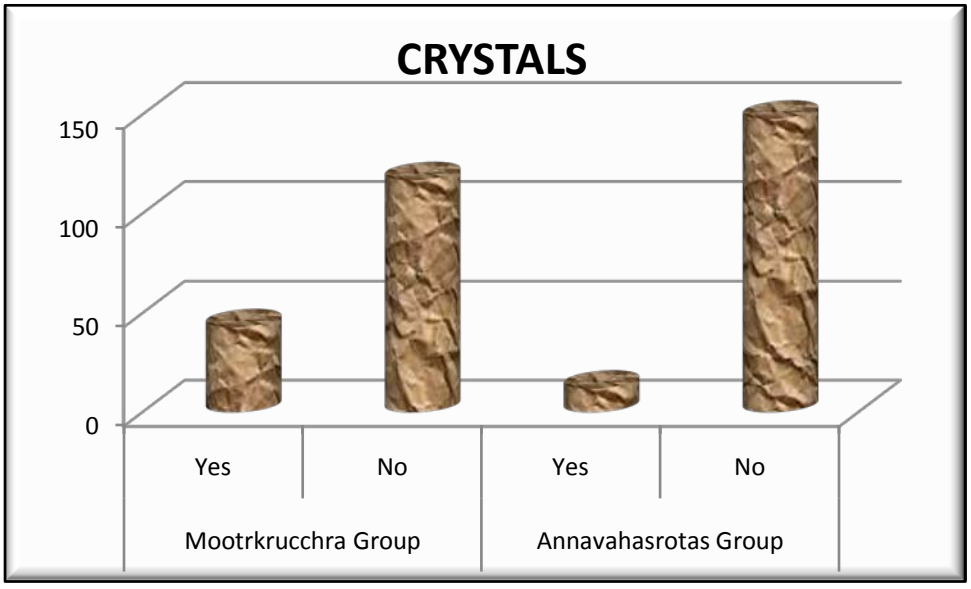
Conclusion:

The proportion of patients with CAST in Mootrkrucchra Group is same as the proportion of patients with CAST in Annavahasrotas Group.

Table5: Frequency distribution of patients according to CRYSTALS

The frequency distribution of patients according to CRYSTALS in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
CRYSTALS	43	117	12	148
%	26.88	73.13	7.50	92.50



Summary:

The frequency of patients having CRYSTALS is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test5: To test whether the proportion of patients with CRYSTALS is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with CRYSTALS in Mootrkrucchra Group is same as the proportion of patients with CRYSTALS in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with CRYSTALS in Mootrkrucchra Group is different than the proportion of patients with CRYSTALS in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
4.59	0.00

Since p value < 0.05, the level of significance; there is strong evidence to reject the null hypothesis.

Conclusion:

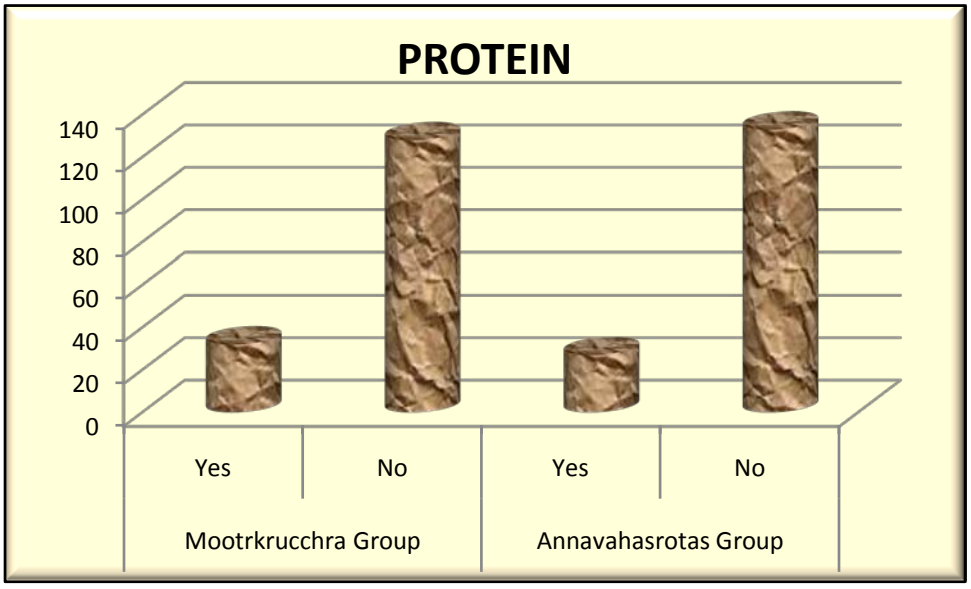
The proportion of patients with CRYSTALS in Mootrkrucchra Group is different than the proportion of patients with CRYSTALS in Annavahasrotas Group.

The proportion of patients with CRYSTALS in Mootrkrucchra Group is greater than the proportion of patients with CRYSTALS in Annavahasrotas Group significantly.

Table6: Frequency distribution of patients according to PROTEIN

The frequency distribution of patients according to PROTEIN in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
PROTEIN	32	128	27	133
%	20.00	80.00	16.88	83.13



Summary:

The frequency of patients having PROTEIN is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test6: To test whether the proportion of patients with PROTEIN is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with PROTEIN in Mootrkrucchra Group is same as the proportion of patients with PROTEIN in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with PROTEIN in Mootrkrucchra Group is different than the proportion of patients with PROTEIN in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
0.72	0.47

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

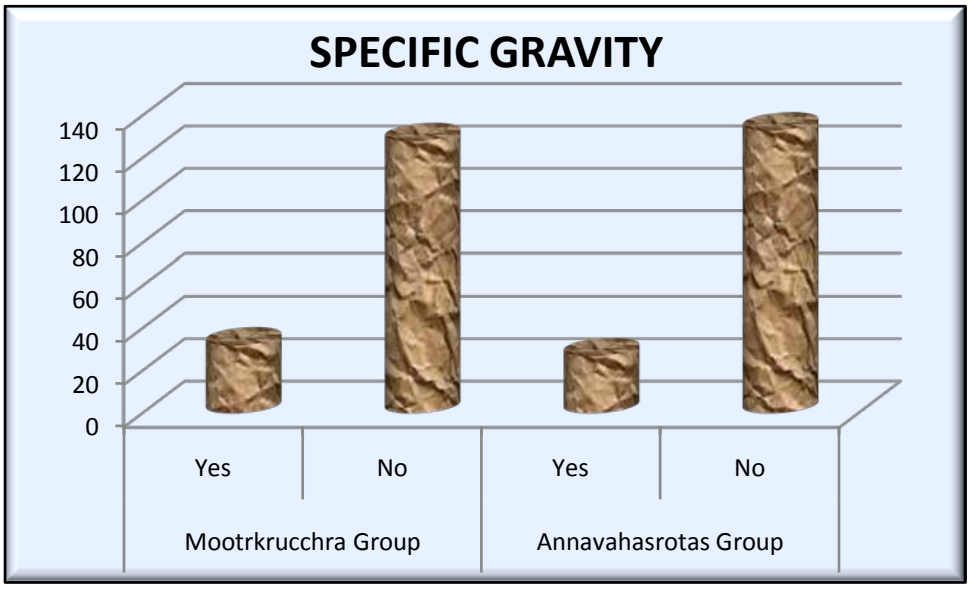
Conclusion:

The proportion of patients with PROTEIN in Mootrkrucchra Group is same as the proportion of patients with PROTEIN in Annavahasrotas Group.

Table7: Frequency distribution of patients according to SPECIFIC GRAVITY

The frequency distribution of patients according to SPECIFIC GRAVITY in both groups along with bar graph is as given below.

Parameters	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
SPECIFIC GRAVITY	32	128	27	133
%	20.00	80.00	16.88	83.13



Summary:

The frequency of patients having SPECIFIC GRAVITY is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test7: To test whether the proportion of patients with SPECIFIC GRAVITY is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with SPECIFIC GRAVITY in Mootrkrucchra Group is same as the proportion of patients with SPECIFIC GRAVITY in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with SPECIFIC GRAVITY in Mootrkrucchra Group is different than the proportion of patients with SPECIFIC GRAVITY in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
0.72	0.47

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

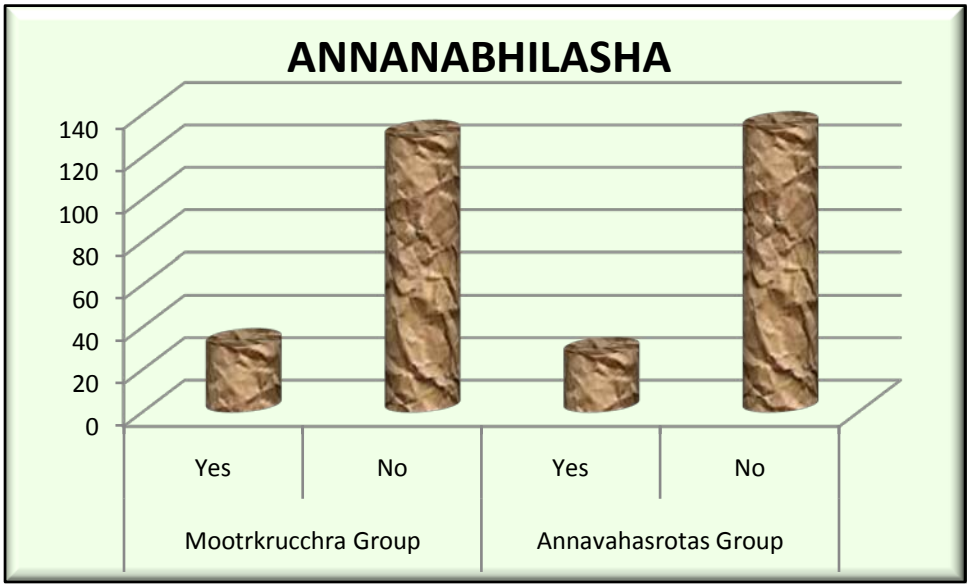
Conclusion:

The proportion of patients with SPECIFIC GRAVITY in Mootrkrucchra Group is same as the proportion of patients with SPECIFIC GRAVITY in Annavahasrotas Group.

Table8: Frequency distribution of patients according to ANNANABHILASHA

The frequency distribution of patients according to ANNANABHILASHA in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
ANNANABHILASHA	31	129	27	133
%	19.38	80.63	16.88	83.13



Summary:

The frequency of patients having ANNANABHILASHA is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test8: To test whether the proportion of patients with ANNANABHILASHA is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with ANNANABHILASHA in Mootrkrucchra Group is same as the proportion of patients with ANNANABHILASHA in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with ANNANABHILASHA in Mootrkrucchra Group is different than the proportion of patients with ANNANABHILASHA in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
0.58	0.56

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

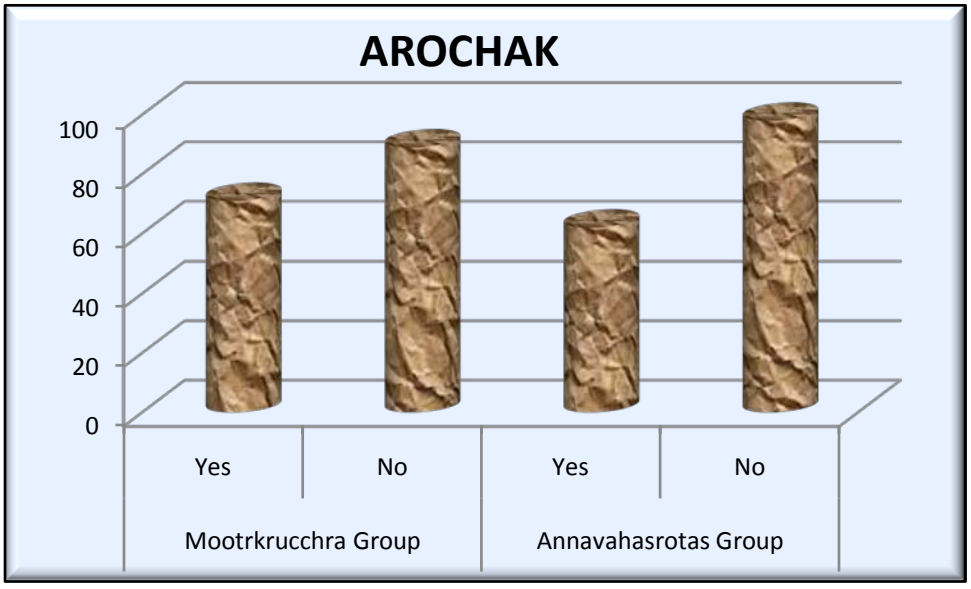
Conclusion:

The proportion of patients with ANNANABHILASHA in Mootrkrucchra Group is same as the proportion of patients with ANNANABHILASHA in Annavahasrotas Group.

Table9: Frequency distribution of patients according to AROCHAK

The frequency distribution of patients according to AROCHAK in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
AROCHAK	71	89	62	98
%	44.38	55.63	38.75	61.25



Summary:

The frequency of patients having AROCHAK is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test9: To test whether the proportion of patients with AROCHAK is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with AROCHAK in Mootrkrucchra Group is same as the proportion of patients with AROCHAK in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with AROCHAK in Mootrkrucchra Group is different than the proportion of patients with AROCHAK in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
1.02	0.31

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

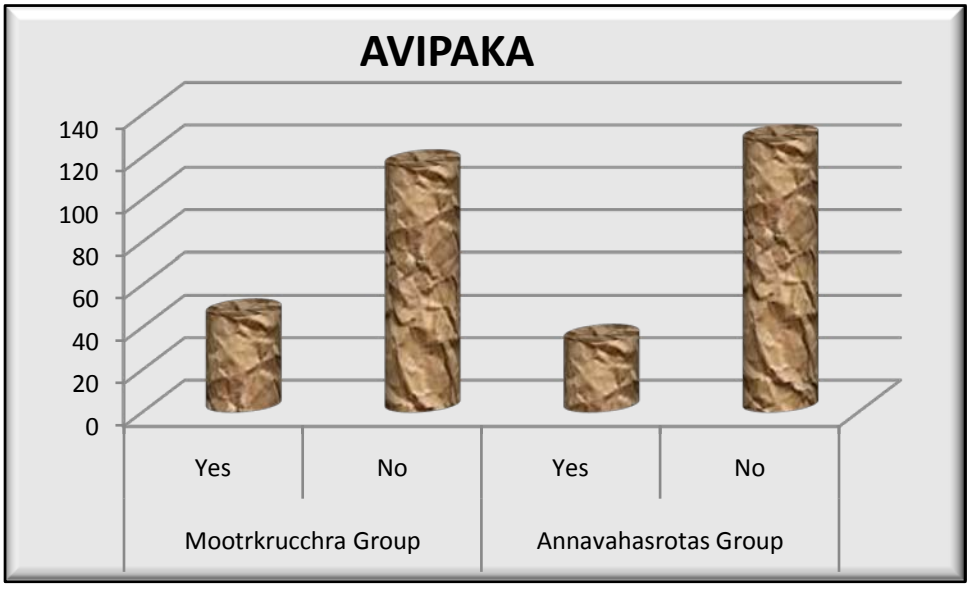
Conclusion:

The proportion of patients with AROCHAK in Mootrkrucchra Group is same as the proportion of patients with AROCHAK in Annavahasrotas Group.

Table10: Frequency distribution of patients according to AVIPAK

The frequency distribution of patients according to AVIPAK in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
AVIPAKA	45	115	33	127
%	28.13	71.88	20.63	79.38



Summary:

The frequency of patients having AVIPAK is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test10: To test whether the proportion of patients with AVIPAK is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with AVIPAK in Mootrkrucchra Group is same as the proportion of patients with AVIPAK in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with AVIPAK in Mootrkrucchra Group is different than the proportion of patients with AVIPAK in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
1.56	0.12

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

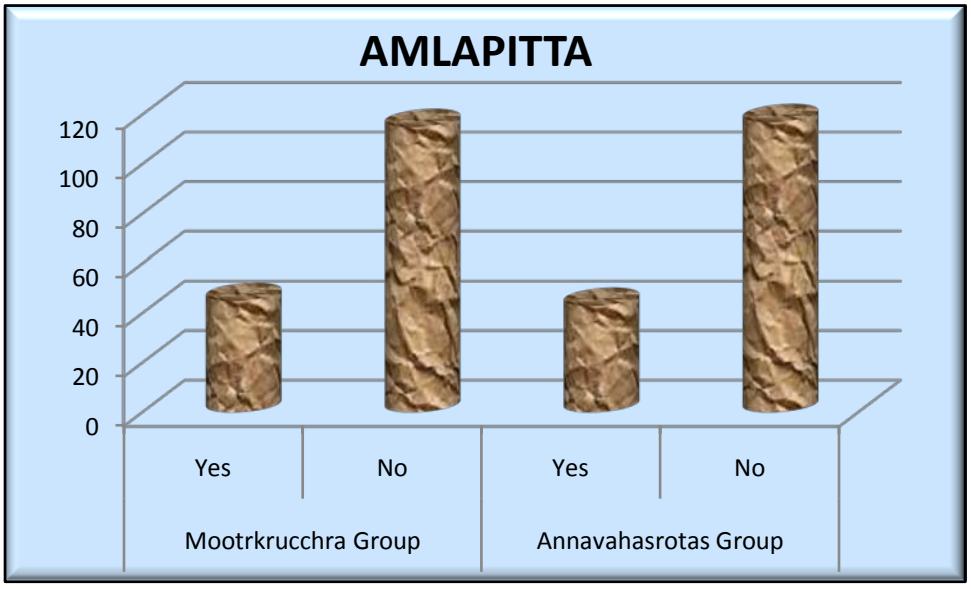
Conclusion:

The proportion of patients with AVIPAK in Mootrkrucchra Group is same as the proportion of patients with AVIPAK in Annavahasrotas Group.

Table11: Frequency distribution of patients according to AMLAPITTA

The frequency distribution of patients according to AMLAPITTA in both groups along with bar graph is as given below.

Parameter	Mootrkrucchra Group		Annavahasrotas Group	
	Yes	No	Yes	No
AMLAPITTA	45	115	43	117
%	28.13	71.88	26.88	73.13



Summary:

The frequency of patients having AMLAPITTA is less in Annavahasrotas Group than it is in Mootrkrucchra Group.

Test11: To test whether the proportion of patients with AMLAPITTA is same in both the groups.

To test the hypotheses,

The null hypothesis, H_0:

The proportion of patients with AMLAPITTA in Mootrkrucchra Group is same as the proportion of patients with AMLAPITTA in Annavahasrotas Group.

Vs.

The alternative hypothesis, H_a:

The proportion of patients with AMLAPITTA in Mootrkrucchra Group is different than the proportion of patients with AMLAPITTA in Annavahasrotas Group.

The test used is z test for proportions.

Calculation Table:

Z statistic	P value
0.25	0.80

Since p value > 0.05, the level of significance; there is no sufficient evidence to reject the null hypothesis.

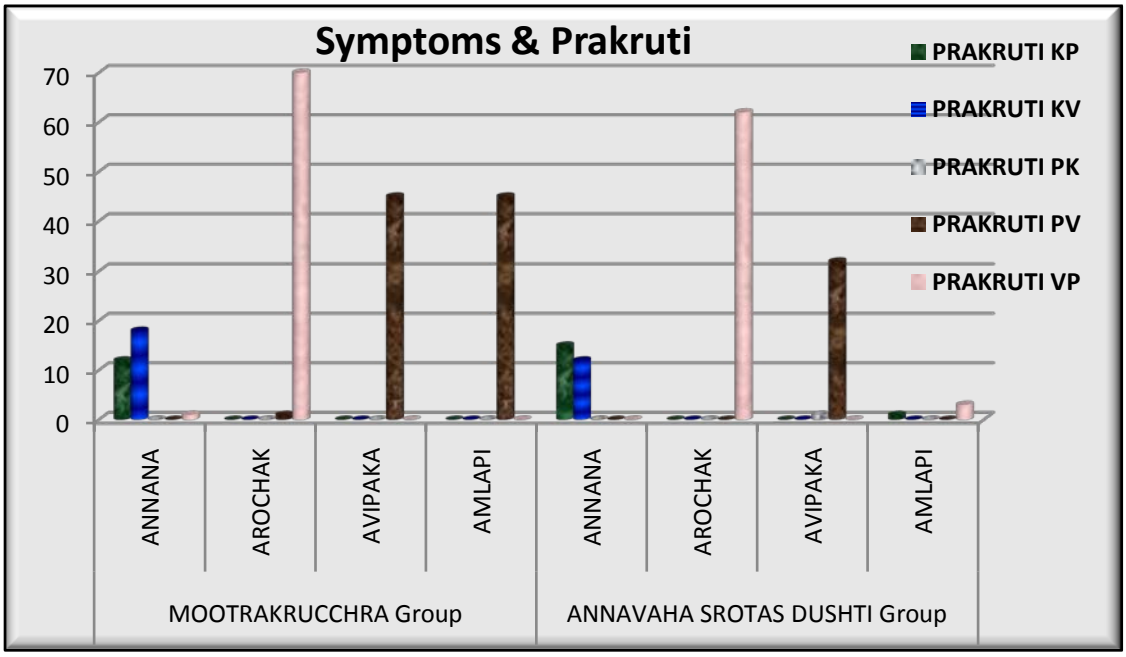
Conclusion:

The proportion of patients with AMLAPITTA in Mootrkrucchra Group is same as the proportion of patients with AMLAPITTA in Annavahasrotas Group.

Table1: Cross tabulation of patients according to PRAKRUTI & Symptoms

The frequency distribution of patients according to Prakruti & Symptoms is given below along with it's bar graph.

Groups	Symptoms	PRAKRUTI				
		KP	KV	PK	PV	VP
MOOTRAKRUCCHRA Group	ANNANA	12	18	0	0	1
	%	7.50	11.25	0.00	0.00	0.63
	AROCHAK	0	0	0	1	70
	%	0.00	0.00	0.00	0.63	43.75
	AVIPAKA	0	0	0	45	0
	%	0.00	0.00	0.00	28.13	0.00
	AMLAPI	0	0	0	45	0
	%	0.00	0.00	0.00	28.13	0.00
ANNAVAHA SROTAS DUSHTI Group	ANNANA	15	12	0	0	0
	%	9.38	7.50	0.00	0.00	0.00
	AROCHAK	0	0	0	0	62
	%	0.00	0.00	0.00	0.00	38.75
	AVIPAKA	0	0	1	32	0
	%	0.00	0.00	0.63	20.00	0.00
	AMLAPI	1	0	0	0	3
	%	0.63	0.00	0.00	0.00	1.88



Summary:

The table & graph shows clearly that

1) ANNANBHILASHA is prominent in K-P & K-V prakruti in both the groups.
2) AROCHAK is prominent in V-P prakruti in both the groups.
3) AVIPAKA is prominent in P-V prakruti in both the groups.
4) AMLAPITTA is prominent in P-V prakruti in MOOTRAKRUCCHRA Group.

Aim: To test that the NA content is high in patients having symptom AROCHAK.

To test the hypotheses,

The null hypothesis, H_0: There is no association between the presence of symptom AROCHAK & having NA content high.

Vs.

The alternative hypothesis, Ha: There is association between the presence of symptom AROCHAK & having NA content high.

The test used is chi square test.

Calculation table:

Groups	NA	AROCHAK		Chi square Value	P value
		No	Yes		
MOOTRAKRUCCHRA Group	Normal	85	2	136.77	0.000
	130 or above 130	4	69		
ANNAVAHA SROTAS DUSHTI Group	Normal	66	2	63.89	0.000
	130 or above 130	32	60		

Since p value < 0.05, the level of significance for both groups; there is strong evidence to reject the null hypothesis.

Conclusion:

There is association between the presence of symptom AROCHAK & having NA content high.

The cross tabulation shows very clearly that the frequency is concentrated at NA level normal & No AROCHAK as well as NA level high & Presence of AROCHAK in both the groups.

The NA content is high in patients having symptom AROCHAK.

CHAPTER

7

DISCUSSION

DISCUSSION

In last 5 years, the lifestyle management awareness in the society has increased noticeably. People are now much focused on balance diet, immunity, prevention and Ayurved science plays an important role in that.

Latest research in medicine field has made the life span of human being very comfortable and larger than before. But although this condition, in the case of kidney disease, numbers of dialysis centre in Mumbai and peripheral area are increasing noticeably.

In our day to day practice the patient complaining of difficulty in passing urine are increasing in numbers noticeably. It is very often that after so many precaution, number of patients of kidney patients are raising day by day. In such patients the journey from simple primary symptom of the disease to system failure becomes very fast.

Ayurved explains the prevention therapy well than any other sciences. Aahar vihar, pathyapathya, shatkriyakaal and its all alarming symptoms are well explained for every vyadhi in Ayurved which plays a key role in preventive measures. It has some specific diagnostic alarms for the diseases which are not dependent on pathology or radiological test as Ayurved takes these techniques for confirmation of diagnosis.

In the case of kidney disease the journey starts from preliminary stage i.e. mootrakrucchra vyadhi and if this vyadhi repeats with number of times in a patient then that patient may suffer from serious complications of that system. So it is time now to focus on the prevention of kidney diseases in society with the Ayurvedic help.

Ayurved has explained the involvement of annavaha srotas dushti in the onset of kidney diseases which can plays key role in prevention of the primary diseases like mootrakruchhra as well all diseases of the urinary system. Hence we have decided to assess the exact type of the annavaha srotasdushti in mootrakruchhra vyadhi as it is the primary disease of urinary system.

So the aim of study was the assessment of anavaha srotas dushti in mootrakrucchra vyadhi

We have studied 160 patients suffering from annavahas srotas dushti more than one year and the second group of 160 patients of mootrakrucchra vyadhi.

Some specific types of annavaha srotas dushti seen which have alarming readings of basic renal pathology that is mootrakrucchra vyadhi.

a) **Annanabhilasha**- Annanabhilasha on and off particularly kapha pradhan prakruti with annavaha srotas vikruti patients more than one year leads to consistent pus cells in the urine microscopy with traced albumin and high range specific gravity of urine seen.

   In kaphaja mootrakruchhra also kapha pradhan prakruti patient showed dominance and annanabhilasha, udargaurav, pus cells in urine and specific gravity of urine on higher side, traced albumine noted.

   So vitiated kapha dosha in kapha pradhan prakruti patients, enters in aanavaha srotas and disturbs the function of agni and forms the pattern of malfunction of agni in terms of annanabhilasha, angagaurav,aalasya .Kapha vitiates the cycle of kleda which lead to kaphaja mootrakruchhra in mootravaha srotas

b) **Arochaka-** Mainly seen in vatpradhan pittanubandhi prakruti patients which is alarming for vataja mootrakrucchra specially ashmari type .Epithelial cells and casts seen in urine microscopy, rasa, rakta, meda, majja , asthi vaha srotas vikruti seen in patients. As described by sushrut in uttarsthan adhyay 7(Tritaiya patalgata dosha), it is found that the long term arochak in vatapitta prakruti patients suffering from squint which is noted and developed during the annavaha srotas dushti period of more than 1 year. In these patients serum sodium which is extracellular electrolyte noted on higher side of normal range which leads to vataj mootrakrucchra as vataj mootrakrucchra is showing as arochaka, is the annavaha srotas vikruti ,morning sickness, puffiness on face and Na in serum at higher range

   In vatapradhan prakruti patients,vitiated vata dosha forms the excessive dryness in all types of secretion of the body and when it enters in annavaha srotas it shows arochaka,aadhmaan as the pattern of dushti and if this dudhti consist for more than one year than the journey of vitiated ruksha guna of vata passes through rasa,rakta,mansa,meda, mootravaha srotas and gives

rukshstva to kapha and pitta and form ashmarijanya mootrakrucchra,in which microscopy cast and epithelial cells are seen.

c) **Avipaka**- mainly seen in pittavata prakruti patients, RBC in urine microscopy and occult blood seen in urine chemistry. Mainly moves towards pittashmari janya mootrakrucchra after one year consistant avipaka. It seen seen that in avipaka more than 1 year in pittaja mootrakrucchra patients, the level of serum potassium seen on lower side in normal limits. Potassium is intracellular electrolyte which balances the internal pressure of the cell for the regular osmosis as well physiology of cell. Symptoms of potassium deficiency show the symptoms of vitiation of pitta by ushna and ruksha guna. Excessive dryness leads to wasting of muscles as well tachycardia which are symptoms of pitta vata prakopa. So it is noted that avipaka more than 1 year can be lead to serum potassium lower range

   The balance of drava, sara and tiksha guna pitta in agni plays important role in annavaha srotas dushti which vitiation shows avipaka and amlapitta significantly and when it goes to mootravaha srotas this gets convert into pittaja mootrakrucchra showing RBC and occult blood in urine

d) **Grahani**- is actually noted as organic so functional disease of the system which starts from agnimandya so this noted in all types of mootrakrucchra as history. Signs of grahani, its pattern of mala, aalasya, angagaurava , trishna and general pattern consist mixed symptoms of vitiation of tridosha specially seen in annavaha srotas. So agnimandya leads to grahani where pitta dushti plays key role among tridoshadushti. and that takes after more than one year to mootrakrucchra vyadhi

e) Asmarijanya mootrakrucchra seen in vatapradhan and pittapradhan prakruti patients which shown arochak and avipaka respectively as an alarm in the form of RBC and cast cells in urine as significant annavaha srotas dushti.

f) Shukraja, kshayaja, Kshataja, Purishaja types of mootrakrucchra were not found in this sample size as the age group included in this study not showing the active participation in samprapti of these disease.

g) Chardi is not seen significantly in mootrakrucchra vyadhi patient which is seen as annavaha srotas dushti in kapha pradhaan prakruti and predormal symptom of renal failure according modern sciences and which involves tridoshs dushti

h) So chardi by tridosha dushti leads to mootraghaat and no any significance in this age group for mootrakruchhra

i) **Amlapitta** – Amlapitta seen in pittapradhaan prakruti patients who are showing RBC in urine and that may lead to pittaja mootrakrucchra as pittaja mootrakruchra showing amlapitta as noticeable symptom of annavaha srotas vikruti and RBC in urine with daha as the specific symptom.

   Amlapitta consistently more than one year with mootrakrucchra hetu as adhoga amlapitta may disturb the normal pH and specific gravity of urine and it may lead to conversion in colour of mootra that is dark yellow to reddish

CHAPTER

8

CONCLUSION

CONCLUSION

After the study of 160 patients in each group we can conclude

1. In present scenario patients from vata pradhan prakruti having arochak, shoola as the symptoms of annavaha srotas dushti more than one year which may converts into microcytic hypochromic anemia with the compliant of vataja asmarija mootrakrucchra presence of epithelial cells as well occasional casts in the urine, after one year Rasa rakta medovaha udakavaha and asthivha srotas dushti seen in these patients. Serum sodium in blood in these patients noticeably seen on higher side of the normal range.

2. Patients having pittapradhan prakruti but more than one year avipaka amlapitta leads to raktavaha, majjavaha, medovaha and mootravaha srotas dushti and that converts into pittaja mootrakruchra which shows occult blood in urine chemistry and calcium oxalate and RBC in microscopy of urine serum potassium of these patients is noticeably seen on lower side of the normal range

3. Patients having annanabhilasha persistently more than one year may convert into kaphaja mootrakruchra which shows pus cells in urine microscopy and specific gravity of urine will be raised in them and protein will be detected in urine. serum chloride of these patients is noticeably seen on lower side of the normal range WBC are on higher side of the normal range and ESR in these patients increased noticeably

4. There is no any significance of serum urea and serum creatinine level with mootrakrucchra patients as well annavaha srotas dusthi diseases as the primary disease.

5. **Aalasya angagaurav trushna and irregular bowel movements** are noticeably seen in the history of all mootrakrucchra patients. **Chardi lakshan not seen** in any of the patient of **Mootrakrucchra in this age group**

CHAPTER

9

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CHAPTER

10

APPENDICES

MOOTRAKRUCCHRA

NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	TYPE	ASHMA	HB	WBC	PLT	INDI	UREA	CREAT	NA	K
1	20	M	STUDENT	VP	S	V	Y	14.2	4500	1.7	N	34.2	1.2	143	
2	34	F	HW	VP	S	V	Y	10.3	8900	1.35	MH	34.9	1.1	145	
3	33	M	PROF	PV	S	P	Y	12.9	6700	2.2		28.5	0.9		3.8
4	26	M	PROF	VP	S	V		10.3	6700	2.5	MH	33.2	0.6	140	
5	33	M	SERVICE	VP	S	V		11.2	7900	3.2		18.7	1.2	147	
6	34	F	HW	PV	S	P	Y	12.4	9900	3.1		23.9	0.7		3.7
7	36	M	PROF	PV	S	P		13.2	9000	1.7		22.6	0.6		3.4
8	45	M	PROF	VP	S	V	Y	12.1	8900	2.2		43.9	1.2	143	
9	43	F	HW	VP	S	V	Y	11.6	8700	3.2	MH	23.9	1.2		
10	23	F	STUDENT	VP	S	V		11.2	5600	3.2	MH	34.2	0.9	144	
11	33	M	SERVICE	VP	S	V	Y	10.9	7600	1.7	MH	28.4	0.8	143	
12	34	M	SERVICE	KP	S	K		13.8	9000	1.7		23.9	1.2		
13	35	M	SERVICE	KV	S	K		13.9	8000	3.2		34.8	0.7		
14	40	F	SERVICE	VP	S	V		11.2	8700	2.8	MH	24.9	0.8	144	
15	32	M	PROF	KP	S	K		13	5700	3.2		22.1	1.2		
16	23	M	STUDENT	VP	S	V		10.2	6400	1.7	MH	28.3	1.2	142	
17	22	F	STUDENT	KV	S	K		11.1	9000	3.2	MH	34.1	0.9		
18	34	F	HW	VP	S	V		11.9	8900	1.3		23.8	0.8	143	
19	35	M	SERVICE	VP	S	V		11.2	7600	1.7	MH	35.2	1.2	144	
20	33	F	SERVICE	VP	S	V	S	10.9	7800	1.7	MH	28.5	0.9	142	
21	34	F	HW	PV	S	P	Y	11.8	9800	1.7		23.9	0.7		3.7
22	33	F	HW	VP	S	K		11.2	9800	2.2	MH	43.9	0.6	138	
23	33	M	SERVICE	VP	S	V		10.2	5600	3.1	MH	34.8	0.5	142	
24	33	M	SERVICE	PV	S	P	Y	12.8	6800	2.2		28.5	1.2		3.6
25	34	M	PROF	PV	S	P	Y	11.6	7600	3.2		34.2	1.1		3.5
26	35	F	SERVICE	VP	S	V		11.9	6500	2.9	MH	23.9	1.3	142	
27	38	M	SERVICE	VP	S	V	Y	11.6	8600	3.2	MH	28.5	0.9	138	
28	37	F	SERVICE	VP	S	V	Y	10.6	9200	1.3	MH	43.9	1.2	137	
29	34	M	SERVICE	PV	S	P		13.1	5900	1.7		34.2	1.3		3.6
30	33	M	PROF	VP	S	V	Y	11.5	5400	2.3	MH	33.2	1.1	135	
31	33	M	PROF	PV	S	P	Y	13.8	8700	3.2		23.9	0.9		3.5
32	45	F	SERVICE	VP	S	V		12.1	6700	3.2		18.3	0.9	139	
33	44	F	SERVICE	PV	S	P	Y	12.4	6500	2.1	MH	19.6	1.1		3.7

MOOTRAKRUCCHRA

NO	CL	PUS	EPI	OCC BLOD	CAST	CRYSTALS	PROTEIN	SP GR	ANNANA	AROCHAK	AVIPAKA	AMLAPI	CHARDI	SHOOLA	KRIMI
1			**P**		**P**					**P**				**P**	**P**
2			P		P					P				P	
3				P		P					P	P			
4			P							P				P	P
5			P							P					
6				P		P					P	P			
7				P		P					P	P			
8			P		P					P					
9			P							P				P	
10			P		P					P					
11			P		P					P				P	P
12	97	P					T	H	P						
13	99	P													
14			P		P					P					
15	98	P					T	H	P						
16			P							P				P	P
17	99	P													
18			P		P					P				P	
19			P		P					P					
20			P							P				P	P
21				P		P					P	P			
22	96	P								P					
23										P					
24				P											
25				P		P					P	P			
26										P				P	
27										P				P	P
28										P					
29				P		P					P	P			
30										P				P	
31				P		P									
32										P				P	P
33															

MOOTRAKRUCCHRA

NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	TYPE	ASHMA	HB	WBC	PLT	INDI	UREA	CREAT	NA	K
34	44	M	SERVICE	VP	S	V	Y	12.2	9000	3.2	MH	34.2	1	135	
35	43	M	SERVICE	PV	S	P	Y	12.2	8700	2.9		33.2	1.2		3.5
36	44	F	HW	KP	S	K		13.2	8900	3.2		23.9	1.4		
37	40	M	PROF	VP	S	V	Y	11.9	9300	2.9	MH	34	1.2	135	
38	43	M	PROF	PV	S	P	Y	13.4	9000	2.2		32.2	1.3		3.5
39	34	F	HW	KP	S	K		13.5	9000	3.2		33.2	1.12		
40	33	M	PROF	VP	S	V	Y	11.9	8700	2.1	MH	23.9	1.23	145	
41	33	F	HW	KV	S	K		11.4	6800	2.9	MH	28.5	1.2		
42	33	M	PROF	PV	S	P	Y	11.9	7600	2.2	MH	23.9	1.2		3.7
43	34	F	HW	VP	S	V	Y	12.1	8700	2.7	MH	19.2	1.1	144	
44	33	M	SERVICE	KP	S	K		13.8	5700	1.7		43.9	0.98		
45	35	F	SERVICE	VP	S	V	Y	11.6	9000	2.8		15.9	1.2	145	
46	34	M	SERVICE	KP	S	K		13.9	7800	2.9		19.6	0.87		
47	23	F	STUDENT	KV	S	K		13.2	5600	1.7		28.5	0.79		
48	22	M	STUDENT	VP	S	V	Y	12.1	5700	2.7	MH	18.3	1.5	145	
49	20	M	STUDENT	KV	S	K		13.9	9000	1.9		27.9	1.2		
50	34	M	PROF	PV	S	P	Y	14	8700	3.2		28.5	1.45		3.5
51	34	M	PROF	KV	S	K		13.9	7800	1.9		34.2	1.23		
52	35	M	PROF	VP	S	V	Y	12.1	9800	2.2	MH	35.4	0.9	143	
53	37	F	HW	KV	S	K		11.2	7600	1.9	MH	33.2	1.25		
54	22	F	STUDENT	KP	S	K		11.6	9000	1.9	MH	23.9	1.2		
55	34	M	PROF	VP	S	V	Y	11.6	8700	3.2	MH	22.3	1.09	139	
56	33	M	SERVICE	PV	S	P	Y	13.4	5700	2.2		34.2	0.9		3.4
57	33	F	SERVICE	VP	S	V	Y	11.6	9000	1.3		24.8	1.2	143	
58	35	F	HW	PV	S	P	Y	9.8	8700	2.2	MH	21.8	1.04		3.7
59	35	M	PROF	PV	S	P	Y	13.2	8700	2.2		23.9	1.2		3.5
60	33	M	SERVICE	VP	S	V		11.6	4500	1.7	MH	33.2	1.1	139	
61	32	F	SERVICE	PV	S	P	Y	13.4	5700	3.2		23.8	1.2		3.5
62	33	M	PROF	VP	S	V	Y	12.7	9000	1.6		19.6	1.07	133	
63	31	M	SERVICE	VP	S	V		12.9	6500	2.25		23.9	1.45	139	
64	31	M	SERVICE	PV	S	P	Y	13.2	5700	3.2		28.5	1.34		3.5
65	32	M	SERVICE	PV	S	P		13.4	6700	1.6		32.9	1.2		3.4
66	30	M	SERVICE	VP	S	V	Y	12.7	8600	2.2		23.9	1.23	139	

MOOTRAKRUCCHRA

NO	CL	PUS	EPI	OCC BLOD	CAST	CRYSTALS	PROTEIN	SP GR	ANNANA	AROCHAK	AVIPAKA	AMLAPI	CHARDI	SHOOLA	KRIMI
34										P				P	
35				P		P					P	P			
36	100	P					T	H	P						
37										P					P
38				P		P					P	P			
39	100	P			P		T	H	P						
40			P		P					P				P	P
41	101	P					T	H	P						
42				P		P					P	P			
43			P		P					P				P	P
44	100	P													
45			P							P				P	
46	99	P					T	H	P						
47	100	P					T	H	P						
48			P		P					P				P	P
49	101	P					T	H	P						
50				P		P					P	P			
51	99	P					T	H	P						
52			P		P					P				P	
53	100	P					T	H	P						
54	99	P					T	H	P						
55			P		P					P				P	P
56				P		P					P	P			
57			P		P					P				P	
58				P	P	P					P	P			
59				P	P	P					P	P			
60										P				P	P
61				P	P	P					P	P			
62										P				P	
63										P				P	
64				P	P	P					P	P			
65				P	P	P					P	P			
66			P		P					P				P	P

MOOTRAKRUCCHRA

NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	TYPE	ASHMA	HB	WBC	PLT	INDI	UREA	CREAT	NA	K
68	22	F	STUDENT	VP	S	V	Y	12.1	8700	2.9	MH	18.3	1.32	148	
69	23	M	STUDENT	PV	S	P	Y	13.4	8900	2.2		22.8	1.1		3.7
70	33	M	PROF	VP	S	V		12.1	5700	1.6	MH	23.9	1.35	144	
71	33	F	SERVICE	PV	S	P	Y	11.2	4700	3.1	MH	34.2	1.2		3.7
72	34	F	SERVICE	VP	S	V	Y	10.9	9000	2.2	MH	29.7	1.09	143	
73	39	M	SERVICE	VP	S	V		11.7	9800	1.6		33.2	0.9	145	
74	20	F	STUDENT	PV	S	P	Y	10.1	7400	1.7	MH	23.9	1.2		3.9
75	23	F	STUDENT	PV	S	P		11.2	6500	3.2		34.2	1.086		3.7
76	36	F	SERVICE	VP	S	V		12.1	5700	3.2		23.8	1.67	145	
77	34	M	SERVICE	VP	S	V		11.9	9000	2.2		23.9	1.34	142	
78	34	M	SERVICE	VP	S	V	Y	10.9	8700	2	MH	29.1	1.2	145	
79	32	F	HW	KV	S	K		10.2	9800	1.7	MH	17.6	1.03		
80	28	F	STUDENT	KP	S	K		11.2	4800	3.2	MH	33.2	1.097		
81	35	F	HW	VP	S	V		11.9	9300	3.2	MH	23.9	1.24	142	
82	33	M	PROF	VP	S	V	Y	11.8	8700	3.1		19.6	1.07	143	
83	33	F	HW	KP	S	K		10.1	5700	1.6	MH	18.9	1.2		
84	35	M	PROF	KV	S	K		13.2	5400	3.2		18.3	0.9		
85	40	F	HW	VP	S	V		12.5	5700	2.9	MH	23.9	1.07	145	
86	19	M	STUDENT	VP	S	V	Y	11.8	7500	3.2		28.5	0.79	143	
87	22	M	STUDENT	VP	S	V		10.7	6900	1.6	MH	19.6	1.2	144	
88	23	F	STUDENT	KV	S	K		11.4	9200	2.8		17.9	1.07		
89	27	M	PROF	KV	S	K		10.9	9000	1.7	MH	23.9	1.2		
90	32	F	HW	VP	S	V	Y	9.8	5800	3.2	MH	17.5	1.07	145	
91	33	M	PROF	VP	S	V	Y	8.9	5700	2.8	MH	28.5	0.79	143	
92	33	M	PROF	VP	S	V	Y	10.2	7200	2.2		23.9	0.9	139	
93	34	M	PROF	KP	S	K		13.9	9800	2.8	MH	19.1	1.2		
94	40	M	SERVICE	KP	S	K		12.2	8700	2.2	MH	18.3	0.79		
95	45	F	SERVICE	KP	S	K		10.3	9900	2.8	MH	34.2	1.07		
96	44	M	SERVICE	VP	S	V	Y	12.1	7400	1.7	MH	33.2	0.6	139	
97	44	M	SERVICE	VP	S	V	Y	11.9	6500	2.7		23.9	1.2	139	
98	44	F	SERVICE	KP	S	K		11.3	9000	2.8		34.2	1.07		
99	43	F	SERVICE	KV	S	K		12.3	5900	3.2		23.9	0.6		
100	40	F	SERVICE	VP	S	V	Y	12.1	7300	3.1	MH	22.1	1.2	139	

MOOTRAKRUCCHRA

NO	CL	PUS	EPI	OCC BLOD	CAST	CRYSTALS	PROTEIN	SP GR	ANNANA	AROCHAK	AVIPAKA	AMLAPI	CHARDI	SHOOLA	KRIMI
68										P				P	
69				P		P					P	P			
70					P					P				P	P
71															
72					P					P				P	
73										P				P	P
74				P		P					P	P			
75				P		P					P	P			
76					P					P					
77			P		P					P				P	
78			P		P					P				P	P
79	100	P					T	H	P						
80	99	P					T	H	P						
81					P					P					
82					P					P				P	
83	98	P					T	H	P						
84	99	P					T	H	P						
85										P					
86			P		P					P				P	P
87			P		P					P					
88	99	P					T	H	P						
89	100	P					T	H	P						
90										P				P	P
91										P				P	P
92										P				P	P
93	98	P					T	H	P						P
94	99	P					T	H	P						
95	99	P					T	H	P						
96							T	H	P	P				P	P
97										P				P	P
98	100	P					T	H	P						
99			P		P		T	H	P						
100			P							P				P	

MOOTRAKRUCCHRA

NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	TYPE	ASHMA	HB	WBC	PLT	INDI	UREA	CREAT	NA	K
102	42	M	SERVICE	VP	S	V	Y	10.9	8000	3.2	MH	19.1	1.07	139	
103	40	M	SERVICE	VP	S	V	Y	11.9	7800	1.6		33.2	0.6	143	
104	32	F	HW	KV	S	K		11.2	9000	1.6		18.3	1.2		
105	30	F	HW	KV	S	K		11.3	6700	3.12		22.1	1.07		
106	43	M	SERVICE	KV	S	K		12.6	8700	3.2		23.9	0.79		
107	45	M	SERVICE	KV	S	K		12.9	7000	2.9		28.7	0.6		
108	34	F	HW	KV	S	K		10.2	6900	1.7	MH	28.5	1.2		
109	31	M	SERVICE	VP	S	V	Y	12.1	5700	2.7		18.3	1.09	144	
110	33	F	HW	KV	S	K		11.1	5400	3.2		19.6	0.79		
111	34	M	SERVICE	VP	S	V	Y	11.4	9000	2.7		23.7	0.9	143	
112	24	F	STUDENT	KV	S	K		10.8	9000	2.2	MH	19.6	1.2		
113	28	M	PROF	KV	S	K		12.9	9000	3.2		24.7	1.04		
114	28	M	PROF	KV	S	K		12.3	5700	1.7		28.5	0.6		
115	43	M	SERVICE	VP	S	V		11.9	9000	1.6		23.7	1.07	130	
116	32	M	SERVICE	PV	S	P	Y	12.9	7400	2.2		23.9	0.9		4.1
117	34	F	SERVICE	PV	S	P		11.1	6900	3.2		24.3	1.04		3.9
118	29	M	PROF	VP	S	V		11.9	7600	1.8		22.4	1.2	138	
119	30	M	SERVICE	PV	S	P	Y	12.9	9000	1.7		23.9	1.07		4.1
120	23	M	STUDENT	PV	S	P	Y	12.2	5600	1.6		34.2	0.79		4
121	25	M	STUDENT	VP	S	V	Y	10.8	7200	1.7	MH	33.2	1.2	139	
122	22	F	STUDENT	PV	S	P		10.3	8700	1.9	MH	33.2	1.04		3.9
123	38	M	SERVICE	PV	S	P		11.9	9000	2.8		34.2	1.07		3.8
124	33	M	SERVICE	VP	S	V	Y	11.2	9000	1.6		23.9	1.2	135	
125	34	F	SERVICE	PV	S	P	Y	12.1	7800	3.2		18.3	0.6		3.9
126	30	M	SERVICE	VP	S	V	Y	12.1	7600	2.8		28.5	1.2	133	
127	32	M	SERVICE	PV	S	P	Y	12	8700	1.7		19.6	0.79		3.9
128	23	F	STUDENT	PV	S	P	Y	11.3	7500	2.8		18.3	1.2		4.1
129	22	M	STUDENT	PV	S	P		13.1	9000	1.7		23.8	1.07		3.9
130	24	M	STUDENT	VP	S	V	Y	11.7	5600	3.2	MH	19.6	0.6	139	
131	30	M	SERVICE	PV	S	P		12.4	9000	2.8		33.2	1.3		4.1
132	22	M	STUDENT	PV	S	P		12.9	7400	1.7		18.9	1.2		
133	32	F	SERVICE	PV	S	P	Y	11.9	7800	2.2		32.7	1.28		
134	30	F	HW	PV	S	P	Y	11.4	8600	3.2		32.1	1.04		

MOOTRAKRUCCHRA

NO	CL	PUS	EPI	OCC BLOD	CAST	CRYSTALS	PROTEIN	SP GR	ANNANA	AROCHAK	AVIPAKA	AMLAPI	CHARDI	SHOOLA	KRIMI
102			P		P					P				P	P
103			P		P					P				P	
104							T	H	P						
105						P	T	H	P						
106							T	H	P						
107						P	T	H	P						
108						P		H							
109					P					P					
110							T	H	P					P	P
111					P					P					
112							T	H	P					P	
113							T	H	P						
114							T	H	P						
115			P		P									P	P
116				P		P					P	P			
117				P		P					P	P			
118														P	
119				P		P					P	P			
120				P		P					P	P			
121			P							P				P	P
122				P		P					P	P			
123				P		P					P	P			
124			P		P					P				P	P
125				P		P					P	P			
126										P				P	P
127				P	P	P					P	P			
128				P							P	P			
129				P							P	P			
130										P					
131				P	P	P					P	P			
132				P											
133				P											
134				P											

MOOTRAKRUCCHRA

NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	TYPE	ASHMA	HB	WBC	PLT	INDI	UREA	CREAT	NA	K
136	33	M	SERVICE	VP	S	V	Y	12.1	8900	3.2	MH	34.2	0.79	139	
137	33	F	SERVICE	PV	S	P		13.1	8700	1.9		23.8	0.6		3.7
138	36	F	HW	PV	S	P		12.8	7800	2.8		28.5	1.07		4.1
139	37	M	SERVICE	VP	S	V	Y	11.7	9000	3.2		34.2	1.2	137	
140	37	M	SERVICE	PV	S	P		12.8	6400	1.9		23.5	0.79		4.1
141	28	M	PROF	PV	S	P	Y	13.2	6500	1.6		31.9	1.04		3.7
142	28	F	HW	VP	S	V		10.9	9000	3.2	MH	33.2	0.6	133	
143	20	F	STUDENT	PV	S	P	Y	11.9	6500	1.6		34.2	1.2		3.7
144	19	M	STUDENT	PV	S	P	Y	13.5	5600	2.2		22.2	0.6		3.5
145	25	M	STUDENT	PV	S	P		13.9	8700	2.8		28.5	1.04		3.8
146	23	F	STUDENT	VP	S	V	Y	11.6	9000	2.2		23.7	0.79	139	
147	34	M	SERVICE	PV	S	P		13.4	5700	2.2		21.9	1.2		3.5
148	30	M	PROF	VP	S	V	Y	10.7	9000	1.9	MH	28.5	0.6	135	
149	33	M	PROF	VP	S	V	Y	11.6	5700	2.8	MH	33.2	1.2	133	
150	34	M	SERVICE	PV	S	P		12.9	9000	2.2	MH	28.5	1.04		3.5
151	33	M	PROF	PV	S	P	Y	13.2	9000	2.2		23.5	1.07		3.6
152	20	M	STUDENT	VP	S	V		12.1	9000	2.9	MH	33.2	1.04	138	
153	29	M	PROF	PV	S	P	Y	13.3	5700	1.9		34.1	1.07		3.5
154	39	F	HW	VP	S	V		10.8	8800	3.1	MH	34.2	0.79	139	
155	34	F	SERVICE	PV	S	P	Y	12.2	9900	2.8		28.5	0.6		3.7
156	38	M	SERVICE	PV	S	P		12.9	9800	1.9		19.6	1.04		3.8
157	33	M	SERVICE	VP	S	V	Y	10.5	4900	1.6	MH	33.2	1.2	133	
158	32	F	HW	VP	S	V		10.8	7500	1.7		25.6	0.9	136	
159	33	F	HW	VP	S	V		10.9	4900	1.9		32.8	1.1	142	
160	32	F	HW	VP	S	V		11.2	6700	2.3		30.5	1	133	

MOOTRAKRUCCHRA

NO	CL	PUS	EPI	OCC BLOD	CAST	CRYSTALS	PROTEIN	SP GR	ANNANA	AROCHAK	AVIPAKA	AMLAPI	CHARDI	SHOOLA	KRIMI
136										P				P	P
137				P		P					P	P			
138				P		P					P	P			
139					P					P				P	P
140				P							P	P			
141				P							P	P			
142														P	P
143				P		P					P	P			
144				P		P					P	P			
145				P		P					P	P			
146					P					P				P	P
147				P	p						P	P			
148					P					P				P	
149										P				P	P
150				P		P					P	P			
151				P		P					P	P			P
152										P				P	
153				P	P	P				P	P	P			
154															
155				P							P	P			
156				P		P					P	P			
157					P					P				P	P
158					P					P				P	P
159					P					P				P	P
160					p					P				P	P

ANNAVAHA SROTAS

SUB NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	AJIRNA	ANNANA	AROCHAK	AVIPAKA	CHARDHI	MALA	AMLAPITTA	NA	CL	K
1	35	F	PROF	VP	S	Y		Y			CONSTI		144		
2	33	M	SERVICE	PV	S				Y			Y			
3	29	F	HW	VP	N	Y		Y			CONSTI		145		
4	33	F	HW	PV	N				Y			Y			
5	34	F	PROF	VP	N			Y		Y	CONSTI		142		
6	24	F	HW	PV	N				Y			Y			
7	29	F	HW	VP	N	Y		Y			CONSTI		140		
8	33	M	SERVICE	KP	S		Y							95	
9	34	F	HW	VP	S	Y					CONSTI		145		
10	39	M	SERVICE	KV	S		Y							95	
11	34	M	SERVICE	KV	S		Y							97	
12	35	F	HW	VP	S	Y		Y					142		
13	35	M	SERVICE	KP	S					Y					
14	34	F	HW	KV	S		Y							95	
15	33	F	HW	VP	S	Y		Y			CONSTI		145		
16	34	M	SERVICE	KP	S		Y							97	
17	35	F	HW	PV	S				Y			Y			3.7
18	35	F	SERVICE	PV	N				Y			Y			3.9
19	33	F	SERVICE	PV	S				Y			Y			3.4
20	34	M	SERVICE	PV	N				Y			Y			3.7
21	33	F	SERVICE	VP	S	Y		Y			CONSTI		145		
22	36	F	SERVICE	PV	N				Y			Y			3.5
23	33	F	HW	VP	S	Y		Y			CONSTI		145		
24	29	M	SERVICE	PV	N				Y			Y			3.6
25	33	M	SERVICE	PV	S				Y			Y			3.5
26	39	F	HW	VP	N	Y		Y			CONSTI		145		
27	35	F	HW	PV	N				Y			Y			3.7
28	33	F	SERVICE	VP	N	Y		Y			CONSTI		144		
29	34	M	SERVICE	PV	S				Y			Y			3.7
30	37	M	SERVICE	VP	S	Y					CONSTI		150		
31	33	F	SERVICE	VP	S	Y					CONSTI		144		
32	39	F	SERVICE	VP	S	Y		Y			CONSTI		139		
33	33	M	SERVICE	VP	SN	Y					CONSTI		144		
34	23	F	HW	VP	S	Y					CONSTI		151		

ANNAVAHA SROTAS

SUB NO	UREA	CREAT	HB	WBC	INDICES	PUS	CRYSTAL	EPI	OCCULT	RBC URINE	proein	SP GR	cast
1	23.9	0.8	10.2	5600	MH			SEEN					Y
2	18.9	0.9	12.4	6700									
3	18.5	0.8	11.4	6300	MH			S					Y
4	17.7	0.6	11.2	9800									
5	23.7	0.7	11.9	7600									Y
6	22.3	0.8	11.2	7900									
7	18.43	1.2	11.5	8900	MH								Y
8	21.4	1.1	13.2	10000		2+					T	S	
9	19.2	0.7	10.6	6500	MH			S					
10	24.4	0.7	12.6	9800		2+					T	S	
11	22.3	0.6	13.5	8900		2+					T	S	
12	23.9	1.2	11.2	7300	MH								Y
13	21.9	1.1	11.9	4600									
14	19.8	1.01	13.2	9800		2+					T	S	
15	29.3	1.05	11.7	8700	MH			S					Y
16	32.1	1.1	13.1	9900		2+					T	S	
17	22.3	0.7	12.9	8600			cal ox			SEEN			
18	28.3	0.9	11.9	9400									
19	32.1	1.2	13.4	4500									
20	29.1	1.1	13.2	5600			cal ox		TRACE	SEEN			
21	22.7	1.2	10.8	6400	MH			S					Y
22	22.8	1.1	12.9	6700									
23	29.3	0.9	11.2	5900	MH								Y
24	32.3	0.9	13.2	7600									
25	34.8	0.7	13.7	7800			cal ox		T	SEEN			
26	28.4	1.2	10.8	9400	MH			S					Y
27	31.5	1.1	13.2	5800									
28	40.1	1.2	11	4700	MH			S					Y
29	28.4	1.1	13.3	6500			cal ox		T	SEEN			
30	28.9	1.1	12.2	7600	MH								
31	38.2	1.2	10.7	8700	MH			S					
32	33.5	1.3	10.8	8900	MH								Y
33	29.3	0.9	12.9	9800	MH			S					
34	34.4	0.9	11.2	6700	MH								

ANNAVAHA SROTAS

SUB NO	AGE	SEX	JIVHA	AJIRNA	ANNANA	AROCHAK	AVIPAKA	CHARDHI	MALA	AMLAPITTA	NA	CL	K
35	29	F	S				Y			Y			3.1
36	32	M	S				Y			Y			3.7
37	33	M	SS			Y		Y	CONSTI				
38	37	F	N	Y					CONSTI		142		
39	39	F	S				Y			Y			3.7
40	33	M	S	Y		Y			CONSTI		141		
41	39	F	S	Y		Y			CONSTI		143		
42	38	F	S	Y		Y			CONSTI		147		
43	29	M	S	Y					CONSTI		144		
44	23	F	S	Y					CONSTI		145		
45	28	F	S	Y		Y			CONSTI		143		
46	28	F	S				Y			Y			3.5
47	36	M	S	Y		Y			CONSTI		144		
48	33	F	S				Y			Y			3.6
49	38	M	S				Y			Y			3.7
50	33	F	N				Y			Y			3.8
51	39	M	S		Y							98	
52	32	F	N		Y							95	
53	32	M	N	Y					CONSTI		144		
54	33	F	N				Y			Y			3.9
55	30	M	N				Y			Y			3.5
56	25	M	S	Y		Y			CONSTI		147		
57	29	F	S	Y		Y			CONSTI		144		
58	33	F	N	Y		Y			CONSTI		147		
59	34	M	S	Y		Y			CONSTI		144		
60	33	F	S				Y			Y			3.6
61	38	F	S				Y			Y			3.5
62	39	M	N	Y					CONSTI				
63	38	F	S	Y					CONSTI				
64	35	M	S		Y							98	
65	34	M	S		Y							95	
66	33	F	S				Y			Y			3.7
67	33	F	N				Y			Y			3.5
68	32	M	N	Y		Y			CONSTI		144		
69	23	F	S	Y		Y			CONSTI		145		

ANNAVAHA SROTAS

SUB NO	UREA	CREAT	HB	WBC	INDICES	PUS	CRYSTAL	EPI	OCCULT	RBC URINE	proein	SP GR	cast
35	33.1	0.8	13.2	7800									
36	23.3	0.8	13.9	5700			cal ox		T	SEEN			
37	25.8	0.7	11.8	6700									Y
38	22.7	0.9	10.6	7500	MH			S					
39	32.7	1.1	13.4	7600									
40	35.4	0.7	11.2	6800	MH								Y
41	19.5	0.8	11.6	5600	MH								Y
42	23.8	1.2	11.1	6700	MH								Y
43	22.7	1.2	12.3	6900	MH			S					
44	27.9	1.1	10.8	8400	MH								
45	32.8	1.2	10.5	6700	MH			S					Y
46	36.9	1.1	13.5	8900			cal ox		T	SEEN			
47	31.9	0.8	13.2	5900	MH								Y
48	32.9	0.9	13.7	6300									
49	31.8	1	11.9	5900									
50	23.3	1.1	13.2	5600			cal ox		T	SEEN			
51	19.8	1.2	13.8	8900		3+					T	S	
52	19.7	1.1	10.2	8600							T	S	
53	29.1	0.9	10.8	8400	MH			S					
54	22.7	0.8	10.9	5800							T	S	
55	28.4	1.1	10	7400							T		
56	26.4	1	13	5000	MH								Y
57	23.3	0.9	11	9800	MH			S					Y
58	21.8	0.7	11.1	7600	MH								Y
59	23.4	0.8	13.1	8700	MH								Y
60	23.1	0.7	13.4	9000			cal ox		T	SEEN			
61	32.8	0.7	13.9	9800									
62	22.3	0.8	12.8	8500	MH			S					
63	27.3	0.7	11.1	4700	MH								
64	28.3	0.8	13.2	9800		3+					T	S	
65	22.3	1.1	13.5	9800		2+					T	S	
66	32.2	1.2	11	7800			cal ox		T	SEEN			
67	31.2	1.1	13.4	8700					T	SEEN			
68	23.8	1.4	13	8900	MH								Y
69	29.3	1.3	10.8	5700	MH			S					Y

ANNAVAHA SROTAS

SUB NO	AGE	SEX	PRAKRUTI	JIVHA	AJIRNA	ANNANA	AROCHAK	AVIPAKA	CHARDHI	MALA	AMLAPITTA	NA	CL	K
72	22	M	VP	S	Y					CONSTI				
73	20	F	VP	S	Y		Y			CONSTI		144		
74	22	F	VP	N	Y		Y			CONSTI		148		
75	28	F	VP	S	Y		Y			CONSTI		145		
76	32	M	VP	S	Y					CONSTI		146		
77	29	F	KV	S		Y							96	
78	33	F	VP	S	Y		Y			CONSTI		147		
79	26	M	KV	S		Y							98	
80	36	M	VP	S	Y		Y			CONSTI		144		
81	33	F	VP	S	Y		Y			CONSTI		145		
82	39	M	VP	S	Y		Y			CONSTI		146		
83	39	F	VP	S	Y		Y			CONSTI		146		
84	32	F	VP	S	Y					CONSTI		148		
85	28	M	VP	S	Y		Y			CONSTI		145		
86	22	F	VP	S	Y					CONSTI		145		
87	25	M	VP	S	Y					CONSTI		144		
88	26	M	KP	S		Y				CONSTI			98	
89	22	F	VP	S	Y					CONSTI		143		
90	27	M	VP	S	Y		Y			CONSTI		143		
91	37	F	VP	N	Y		Y			CONSTI		145		
92	23	F	PV	S				Y		CONSTI	Y			3.8
93	24	M	VP	S	Y					CONSTI		145		
94	29	F	VP	S	Y		Y			CONSTI		145		
95	32	F	VP	S	Y		Y			CONSTI		152		
96	32	M	VP	S	Y		Y			CONSTI		145		
97	22	M	VP	S	Y		Y			CONSTI		155		
98	27	F	VP	N	Y		Y			CONSTI		145		
99	19	M	VP	N	Y					CONSTI		145		
100	37	F	VP	S	Y					CONSTI		147	F	
101	35	M	VP	S	Y		Y			CONSTI		148		
102	34	F	VP	S	Y		Y			CONSTI		145		
103	28	F	VP	S	Y					CONSTI		144		
104	22	M	VP	S	Y					CONSTI		145		
105	21	F	VP	S	Y					CONSTI		144		
106	31	F	VP	S	Y		Y			CONSTI		143		

ANNAVAHA SROTAS

SUB NO	**UREA**	CREAT	HB	ESR	WBC	INDICES	PUS	CRYSTAL	EPI	OCCULT	RBC URINE	proein	SP GR
72	23.9	0.8	13.1	10	7900	MH			S				
73	27.4	0.9	11.1	9	7600	MH							
74	26.3	1.1	10.1	12	7800	MH							
75	22.3	1.2	10	11	5600	MH			S				
76	27.4	1.2	9.8	12	6500	MH							
77	28.5	1.1	9.7	24	9800		2+						
78	23.5	0.9	11.1	12	7900	MH							
79	28.4	0.8	13.6	31	8900		3+					T	S
80	34.2	1.1	13.1	10	9800	MH			S				
81	33.1	1.2	12	9	8800	MH							
82	35.2	1.1	13	11	5600	MH			S				
83	21.3	1	12.1	12	7800	MH			S				
84	22.5	0.9	11	11	7600	MH			S				
85	23.4	0.8	13	7	7800	MH							
86	21.9	0.9	10	10	8900	MH							
87	34.6	0.9	12.9	12	8800	MH							
88	34.5	0.8	13.6	31	9800		3+					T	S
89	33.4	0.9	11	10	9700	MH							
90	32.1	0.8	11.9	12	9700	MH			S				
91	21.4	0.9	11.2	11	8700	MH							
92	22.3	0.7	11.9	10	6700			cal ox		T	SEEN		
93	27.4	0.9	12.9	12	6800	MH							
94	28.3	0.95	10.8	11	7600	MH							
95	23.9	1.1	12.9	7	7700	MH			S				
96	22.1	1.1	12.9	10	7800	MH			S				
97	26.4	1.3	13	11	9700	MH			S				
98	32.9	1.1	10.8	12	5600	MH							
99	34.1	1.02	11.5	11	6700	MH			S				
100	40	1.09	12.1	11	6800	MH			S				
101	34	1.03	13.7	10	7800	MH							
102	33.9	1.09	8.9	12	5600	MH							
103	38.7	1.12	9	21	7600	MH			S				
104	32.8	1.2	13.1	11	7800	MH			S				
105	34.7	1.23	9.1	9	7500	MH							
106	29.4	1.21	9.9	10	7700	MH			S				

ANNAVAHA SROTAS

SUB NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	AJIRNA	ANNANA	AROCHAK	AVIPAKA	CHARDHI	MALA	AMLAPITTA	144		
107	33	M	STUDENT	VP	S	Y		Y			CONSTI		143		
108	33	F	HW	VP	S	Y					CONSTI		**NA**	**CL**	**K**
109	32	M	STUDENT	VP	N	Y					CONSTI		145		
110	29	M	STUDENT	KP	N		Y							99	
111	20	M	STUDENT	VP	N			Y		Y					
112	20	M	STUDENT	PV	N				Y			Y			3.8
113	22	M	STUDENT	VP	S	Y		Y			CONSTI		144		
114	32	F	HW	VP	S	Y		Y			CONSTI		145		
115	37	M	SERVICE	VP	S	Y		Y			CONSTI		144		
116	34	F	HW	VP	S	Y		y			CONSTI		145		
117	33	M	SERVICE	KP	S		Y							101	
118	32	F	HW	KV	N		Y							100	
119	27	M	STUDENT	KP	N		Y							99	
120	25	M	STUDENT	VP	N	Y		Y			CONSTI		145		
121	29	F	HW	KP	S		Y							99	
122	22	M	STUDENT	VP	S	Y					CONSTI		143		
123	28	F	STUDENT	VP	S	Y		Y			CONSTI		141		
124	22	M	STUDENT	KP	S										
125	32	F	SERVICE	VP	S	Y		Y			CONSTI		140		
126	33	M	SERVICE	KP	S		Y							98	
127	26	M	SERVICE	VP	S	Y		Y			CONSTI		141		
128	29	F	HW	KV	S		Y							99	
129	39	M	SERVICE	KV	S		Y							99	
130	35	F	HW	KV	S		Y							100	
131	34	M	SERVICE	KP	S		Y							99	
132	34	M	SERVICE	VP	S	Y					CONSTI		140		
133	37	M	SERVICE	VP	S	Y					CONSTI		141		
134	34	M	SERVICE	KP	S		Y							98	
135	24	M	STUDENT	VP	S	Y					CONSTI		144		
136	27	M	STUDENT	KP	S		Y							100	

ANNAVAHA SROTAS

SUB NO	UREA	CREAT	HB		ESR	WBC	INDICES	PUS	CRYSTAL	EPI	OCCULT	RBC URINE	proein	SP GR
107	22.4	1.22	11.7		11	7400	MH							
108	25.3	1.29	10.6		21	5700	MH			S				
109	23.3	1.1	11.9		10	6700	MH			S				
110	27.5	1.12	13.2		31	9000		3+					T	S
111	21.1	0.93	12.3		10	7500								
112	32	0.87	12.1		22	8300								
113	22.7	0.89	13.1		24	7900	MH			S				
114	33.4	0.93	10.2		12	8900	MH			S				
115	32.1	0.98	13.3		10	8700	MH			SS				
116	31.2	0.99	10.7		32	5600	MH			S				
117	30.1	0.87	13.2		21	8900		3+					T	S
118	29.8	0.89	12.9		41	9800		2+					T	S
119	22.3	0.8	12.9		25	9000		2+					T	S
120	28.7	1.1	12.6		10	6700	MH							
121	24.3	1.2	13.9		29	9000							T	S
122	28.2	1.1	12.7		11	7600	MH							
123	22.4	1.1	10.2		10	5700	MH							
124	27.3	1.02	13.6		9	6800							T	S
125	25.3	1.02	10.5		8	7800	MH			S				
126	21.4	1.03	13.6		40	8400		2+					T	S
127	32.4	1.1	11.9		5	8900	MH			S				
128	31.5	1.2	10.3		45	9800		2+					T	S
129	30.3	1.1	13.2		35	9700		2+					T	S
130	34.3	1.23	11.2		43	9300		3+					T	S
131	32.3	1.29	3.1		27	9000		2+					T	S
132	34.5	1.1	13.1		9	6700	MH			S				
133	31.2	0.98	12.2		12	6500	MH			S				
134	30.3	0.93	13.2		41	8000		3+					T	S
135	21.2	0.8	12.1		10	7000	MH			S				
136	23.2	0.88	13.6		29	8000		2+					T	S

ANNAVAHA SROTAS

SUB NO	AGE	SEX	OCCU	PRAKRUTI	JIVHA	AJIRNA	ANNANA	AROCHAK	AVIPAKA	CHARDHI	MALA	AMLAPITTA	NA	CL	K
137	29	F	STUDENT	VP	N	Y		Y			CONSTI		143		
138	21	M	STUDENT	KP	N		Y							98	
139	22	M	STUDENT	KV	S		Y							99	
140	22	M	STUDENT	VP	S	Y		Y			CONSTI		143		
141	36	M	SERVICE	VP	N	Y		Y			CONSTI		144		
142	29	M	SERVICE	VP	N	Y		Y			CONSTI		143		
143	23	M	STUDENT	VP	N	Y		Y			CONSTI		142		
144	38	M	SERVICE	VP	S	Y		Y			CONSTI		144		
145	38	M	SERVICE	VP	S	Y					CONSTI		143		
146	33	M	SERVICE	KP	N		Y							99	
147	27	M	STUDENT	VP	S	Y					CONSTI		142		
148	28	M	STUDENT	VP	S	Y					CONSTI		140		
149	32	F	HW	VP	N	Y		Y			CONSTI		142		
150	31	M	SERVICE	PV	S				Y			Y			3.7
151	25	M	STUDENT	PV	S				Y			Y			3.8
152	32	M	SERVICE	PV	S				Y			Y			3.9
153	24	F	STUDENT	VP	S	Y		Y			CONSTI		142		
154	32	M	SERVICE	VP	N	Y		Y			CONSTI		144		
155	30	M	SERVICE	VP	N	Y		Y			CONSTI		143		
156	33	M	SERVICE	VP	S	Y					CONSTI		145		
157	33	M	SERVICE	VP	S	Y					CONSTI		144		
158	30	M	SERVICE	VP	S	Y					CONSTI		143		
159	34	F	HW	PV	S				Y			Y			3.7
160	35	F	HW	PV	S				Y			Y			3.9

ANNAVAHA SROTAS

SUB NO	UREA	CREAT	HB	ESR	WBC	INDICES	PUS	CRYSTAL	EPI	OCCULT	RBC URINE	proein	SP GR	cast	SUB NO
137	29.1	0.92	10.8	10	8700	MH								Y	137
138	32.3	0.91	13.6	28	7900		2+					T	S		138
139	36.3	0.99	13.2	27	9000		3+					T	S		139
140	32.1	0.98	12.7	11	8500	MH			S					Y	140
141	36.4	0.99	12.1	12	8000	MH								Y	141
142	41	0.91	11.6	10	6000	MH								Y	142
143	42.1	0.93	13.2	9	7500	MH			S					Y	143
144	26.9	0.89	13.2	12	7600	MH								Y	144
145	23.4	1.1	12.7	10	6900	MH									145
146	32.3	1.23	13.2	42	8000		2+					T	S		146
147	32.1	1.11	12.6	9	5900	MH			S						147
148	29.9	1.24	12.8	10	7500	MH			S						148
149	23.3	1.22	11.1	11	6700	MH			S					Y	149
150	25.4	1.25	13.2	9	6500			cal ox		T	SEEN				150
151	23.4	0.9	13.2	12	9800					T	SEEN				151
152	20.9	0.98	13.6	10	9600			cal ox		T	SEEN				152
153	31.9	0.97	10.2	8	4800	MH			S					Y	153
154	22.9	0.87	13.1	9	6800	MH			S					Y	154
155	32.9	0.79	11.3	11	4800	MH								Y	155
156	33.4	0.99	11.5	12	7500	MH			S						156
157	32.3	0.79	11.9	10	7600	MH									157
158	42.1	1.1	12.9	11	6900	MH			S						158
159	22.3	0.98	11	12	6200	MH				T	SEEN				159
160	25.80	0.78	10.2	14	6000	MH				T	SEEN				160

ASSESSMENT OF ANNAVAHA SROTAS DUSHTI IN MOOTRAKRUCCHRA VYADHI

CONSENT STATEMENT

- My signature on this form signifies that I have read and understood this subject information and consent form and that I freely give my consent to participate in this study.
- I have had sufficient time to consider the information provided and to ask for advice if necessary.
- I have had the opportunity to ask questions and have received satisfactory responses to my questions.
- I understand that all of the information collected will be kept confidential and that the results will only be used for scientific objectives.
- I understand that my participation in this study is voluntary and that I am completely free to refuse to participate or to withdraw at any time without changing in any way the quality of care that I receive.
- I understand that I am not waiving any of my legal rights as a result of signing this consent form.
- I understand that there is no guarantee that this study will provide any benefits to me (if applicable).
- I have read this form and I freely consent to participate in this study.
- I have been told that I will receive a dated and signed copy of this form.

_____ _____ _____

Name (printed) Signature Date

---------- ---------- ----------

Name (Witness) Signature Date

_____ _____ _____

Principal Investigator/designate Signature Date

CASE RECORD PROFORMA

Name - _____Date- _____

Address -

_____

_____

_____

OPD No - _____ IPD No - _____

Occupation - _____

***Desh* - _____ Cast - _____**

***Kaal* - _____ Age - _____ Sex -**

_____

Chief complaints with duration-

Vedana vishesh **Duration**

History of present illness-origin-duration-progress

(*Vartaman Vyadhivritta*)

History of past illness-

(*Purvotpanna Vyadhi*)

Family History-

(Kulaj Itihaas)

Samanya Parikshan-

Nadi-

Jivha-

Shabda-

Sparsha-

Druka-

Aakruti-

Mala-

Prakruti parikshan-

Srotas parikshan-

Annavaha srotas-

Hetu

1. *Ahita bhojan*
2. *Atimatra bhojan*
3. *Akaal bhojan*
4. *Pavakasya vaigunyaat*

Dushti lakshan

1. *Annanabhilashana-*
2. *Arochak-*
3. *Avipaka-*
4. *Chhardi-*

Jaranshakti-

Abhyavaran shakti-

Kshudbodha-

Agni-

Agnimandya-

Vyadhi	Hetu-period	Lakshan	Prakaar
Agnimaandya	Daurbalya Chinta Jaagran Shrama Avyaam Aahaar Niyambhanga Apatarpana	Gaurava Aalasya Kshudhamaandya Shoola Daurbalya	Vaataj Pittaj Kaphaj
Ajirna	Guru Snighdha Madhur Viruddha Anna Adhyashana Prabhutashana	Gaurav Aadhmaan Aatop Shoola Trishna Hrallas Utklesh Chardi Jwara Dravamala Pravrutti	Aama Vidagdha Vishtabdha Rasashesha Dinpaaki
Alasak	Viruddha Anna Garavisha Adhyashana Vegavidhaaran	Mala and Vata Apravrutti Aadhmaan Udgaarnirodh Shoola Arati	Vataj Pittaj Kaphaj
Chardi	Atidrava Asaatmya Tikshna Sevan Ashuchi Mrudbhakshan Krimi Ajirna	Hrallas Prasek Utklesh Chhardi Daha Trushna Shoola	Vataj Pittaj Kaphaj Sannipatik Dwishtaarthaja

Amlapitta	Katu Amla Lavan Ushna Abhishyandi Oily Viruddha Vishamasevan Shile sprout	Prasek Amlaudgaar Tiktaudgaar Katuudgaar Urovidaha Amlika Shoola Chardi Shirashoola Bhrama	Urdhwaga Adhoga
Shoola	Viruddha Vishamaseva Adhyashana Sproutes Ajeerna Vegavarodha	Aruchi Malavashtambha Aaadhmaan Aatop Shoola	Vataj Pittaja Kaphaja Vatapittaj Pittakaphaja Vatakaphaja Sannipatik Aamaj
Grahani	Vishamaashana	Avipaaak Aarochaka Aalasya Chaardi Daurbalya Malapravrutti- Dravya and aniyamit Aantrakoojan Shoola Mukhapaak Trishna Arati	Vataj Pittaj Kaphaj Sannipaatik

Krimi	Snigdha Madhur Vishama Ashuchi sevan	Udaraada Aantraada Hridayachara Churava Darbhapushpa Saugndhik Mahaguda	Atisar Jwara Udarvrana Shotha Kamala Gulma Baddhodar Vidradhi

Dosha-

Dushya-

Differential diagnosis (*Vyadhi pratyatma lakshan*)-

Disease-

Type-

Pathological investigations-

Urine-

Routine- Microscopic-

Blood -

Haemogram -

ESR-

Serological-

Serum urea-

Serum creatinine-

Serum electrolyte-

Upashaya/Anupashaya-

Upadrava-

Udarka-

Sadhyasadhyatva-

Arishta Lakshan-

CASE RECORD PROFORMA

Name - _____ Date- _____

Address -

_____

_____

_____

OPD No - _____ IPD No - _____

Occupation - _____

***Desh* - _____ Cast - _____**

***Kaal* - _____ Age - _____ Sex - _____**

Chief complaints with duration-

Vedana vishesh **Duration**

History of present illness-origin-duration-progress

(*Vartaman vyadhivritta*)

History of past illness-

(*Purvotpanna vyadhi*)

Family History-

(Kulaj itihaas)

Samanya parikshan-

Nadi- ***Jivha-***

Shabda- ***Sparsha-***

Druka- ***Aakruti-***

Mala-

Prakruti Parikshan-

Srotas parikshan-

Sign & Symptoms-

Pain-Mild / Moderate / Burning / Pricking / Radiating

Site-Adhodar/Vankshana/Basti/Paarshwa/Naabhi/penis

Heaviness - Mild / Moderate

Site-Adhodar/Vankshana/Basti/Paarshwa/Naabhi

Sign & Symptoms - **Duration-**

Dribbing micturation -

Burning micturition -

Hematuria -

Frequency - **Quantity-**

Color- **Watery/Pale yellow/Dark yellow/other**

Sticky urine-

Seminisation of urine -

Associated symptoms -

Loss of appetite -

Indigestion -

Constipation -

Fissure -

Distention of abdomen -

Pain at heart region -

Vertigo -

Nausea-

Others-

Dosha- ***Dushya-***

Differential diagnosis (*Vyadhi pratyatma lakshan*)-

Disease-

Type –

Hetu-

Tikshna

Ruksha

Adhyashana

Ajirna

Madyapaana

Ativyayaam

Maithun

Jerking travelling

Pathological investigations-

Urine-

Routine- **Microscopic-**

Blood -

Haemogram -

ESR-

Serological-

Serum urea-

Serum creatinine-

Serum electrolyte-

Upashaya/Anupashaya-

Upadrava-

Udarka-

Sadhyasadhyatva-

Arishta Lakshane

CHAPTER

12

NORMAL RANGE OF PATHOLOGICAL INVESTIGATIONS

Normal Range of pathological investigations

1. Haemoglobin (gm per 100ml)	Males - 13 to 18 gm %	
	Females - 12 to 14.5 gm %	
2. Erythrocytes (ml per c. mm)	Males - 4.5 to 6 mil/c. mm	
	Females - 4.0 to 5.0 mil/c. mm	
3. Leucocytes (per c. mm)	- 4000 to 10000 per c. mm	
4. Differential count		
a. Neutrophils	40 to 70 %	
b. Lymphocytes	20 to 30 %	
c. Monocytes	2 to 6 %	
d. Eosinophils	0 to 6 %	
e. Basophils	0 to 1 %	
f. Platelets	1.5 – 5 lacks / c. mm	
g. ESR	Male – 0 to 10 mm / hr	
	Female – 0 to 20 mm / hr	
5. Serum Urea	10 to 40 mg / dl	
6. Serum Creatine	0.5 to 1.5 mg / dl	
7. Serum sodium	136-145mEq/l	
8. Serum potassium	3.5-5.1 mEq/l	
9. Serum chloride	97-111m Eq/l	
10. Urine examination		

Routine –

Colour	-	Pale yellow
Appearance	-	Clear
Deposit	-	Nil
Albumin	-	Nil
Sugar	-	Nil
Occult blood	-	Nil
Ketone bodies	-	Nil
Bile salts	-	Nil
Bile Pigments	-	Nil

Microscopic-

Pus cells	-	1-2/hpf
Epitilial cells	-	occasionally
Amorphous material	-	Not seen
RBC	-	Not seen
Casts	-	Not seen
Crystals	-	Absent
Bacterial	-	Absent

CHAPTER

13

LIST OF ABBREVATIONS

LIST OF ABBREVATIONS

Charak-	Ch.
Sushrut-	Su.
Vagbhat-	Vag.
Sootrasthan-	Su.
Nidansthan-	Ni.
Viman sthan-	Vi.
Chiktsasthan-	Chiki.
Uttarshtran-	utt.
Ashtang Hridaya-	Ash.Hr
Amlapitta-	Amla.