CLINICAL MEDICINE

A SYSTEM OF DIAGNOSIS IN OUTLINE

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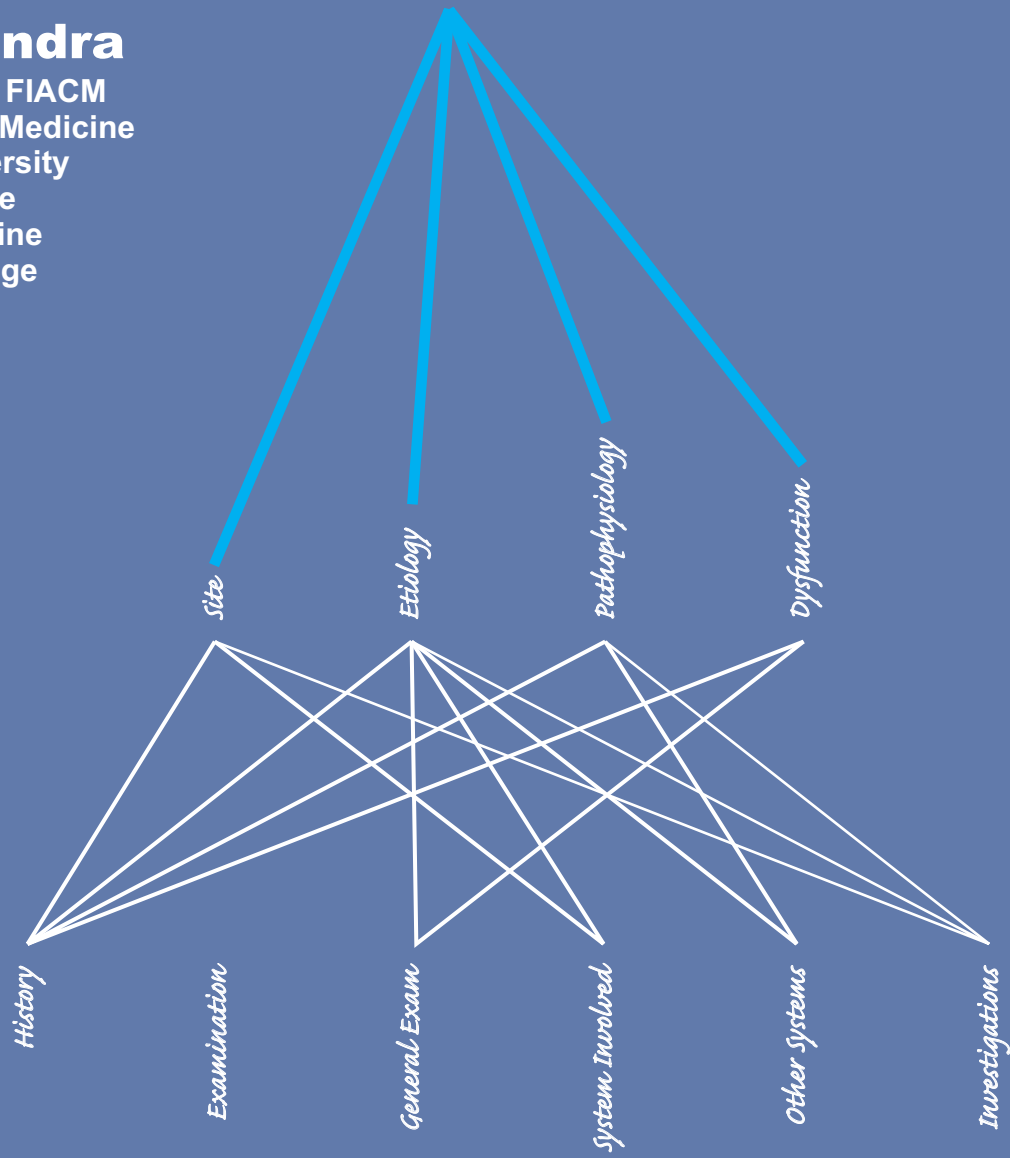

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DEDICATION

This book is dedicated to all my teachers and especially Prof. K.B. Kunwar, Professor and Head, Department of Medicine, King George Medical College (1960-1977), Lucknow who as a medical philosopher par excellence and a teacher to the core laid in me the seeds of this book.

It is dedicated to My Family who kept egging me on to complete this book.

As happens with a first book, and one with a new approach, it gets modified on each reading and with progressive teaching of its contents to successive generations of medical students.

Over the years this book has been in a continuous state of revision and so is also dedicated to My Students.

And perhaps continue to be improved upon.

CLINICAL MEDICINE
A SYSTEM OF DIAGNOSIS

INTRODUCTION

Clinical medicine encompasses two important facets:

- Reaching a complete diagnosis, and
- Getting the patient to comply with the management strategy planned

One being skill and the other an art, both of these have to take into account practicalities, and have to be cost-effective and within available resources. A good complete diagnosis reduces investigation costs and avoidance of polypharmacy further reduces the costs incurred.

This short work deals with a uniform format for diagnostic workup of a patient presenting with any symptom or sign due to involvement of any system. An obvious and useful bias will be there towards the common diseases and presentations but the format is adaptable to all branches, disciplines and specialties.

DIAGNOSIS includes-

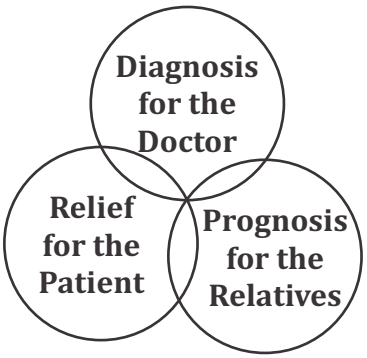
- Site of Disease (Anatomical)
- Nature of Disease (Etiological)
- Pathophysiology (Complications)
- Functional Disturbance (Dysfunction)

Books on clinical medicine tell us how to elicit the history and physical signs whereas standard text books discuss diseases one by one but there is no good book linking the former with the latter and this present work does just that.

This short treatise aims to train the clinician to reach a diagnosis by a rational step-by-step analysis of data obtained by the history and physical examination of patient, supplemented by some well reasoned investigations. Then one can read about the disease at length from any standard text book.

The art and science of clinical medicine does not undergo much change with time. Though newer tests, drugs and even diseases are discovered, basic clinical approach remains the same and is applicable to all branches, disciplines and specialties of medicine- it is all encompassing.

A good rapport with the patient is essential while taking history of illness and examining him. Well directed tests are also needed in many cases.



DIAGNOSIS can be made in many ways and one way may be more suited than the other in an individual case. Before we go to the COMMON PLAN a word about these various diagnostic methods is in order.

WAYS IN WHICH A DIAGNOSIS CAN BE MADE

1. SPOT diagnosis: Pertains to advanced disease, skin diseases, endocrinal system involvement, some collagen disorders and many rare congenital disorders.
2. Analyzing the main PRESENTING SYMPTOM
3. Analyzing the main PHYSICAL SIGN
4. Analyzing the SYNDROME - Symptom-sign complex
5. Analyzing the presenting COMPLICATION of an underlying undiagnosed disease
6. Investigations:
    - Routine
    - Random
    - Specific
    - Screening Programmes

The next section starts with a Common Plan about compiling the data from the various parts of the History, General Examination, Systemic Examination and the Other Systems and how to analyze this data in small comprehensive and comprehensible segments.

The sections following take up this Common Plan as applied to the various systems of the body; Cardiology, Respiratory System, Neurology, Liver and Kidney diseases. Each of these systems has its own peculiar anatomic configuration and sub-sites, common types of diseases and also their own pathophysiology.

Thereafter, there is a very short section which summates the information this book provides and finally an epilogue which seeks to put things into a practical perspective and re-emphasizes the importance of a wholesome diagnosis and how therapy is geared to this knowledge.

THE COMMON PLAN – AN OUTLINE

ANALYSIS OF PATIENT'S HISTORY / PHYSICAL EXAMINATION

The complete diagnosis including all four parts mentioned earlier can be deciphered by asking a set series of questions after having elicited the history and physical signs.

(Note: Pathophysiology is based on our understanding of how a disease affects a particular organ to produce dysfunction. It depends on our PREVIOUS knowledge and studies of Physiology and Pathology of similar patients. Some tests can help us in proving the pathophysiology operating in a particular case.)

A. HISTORY
A carefully elicited history should be able to answer the following FIVE questions—A1, A2, A3, A4 and A5

A.1 Which **ORGAN SYSTEM** is involved?
This is based on the conglomeration of symptoms--cardinal symptoms of a particular system.

Is there any particular SITE involved?
This is suggested by some pathognomic symptoms and other details.

e.g. -- Lateral chest pain associated with dry cough, and the pain increasing on coughing and deep breathing suggests pleural pain.

A.2 What **PATHOPHYSIOLOGY** accounts for some of the consequential symptoms?
These symptoms explain the changes occurring after the disease progresses.

E.g. A patient of known heart disease when develops pedal edema, one can suspect that congestive heart failure has occurred.

A.3 CAUSE OF DISEASE

What is the possible/probable **Nature (Etiology)** of the disease?

This is based on --
ONSET -- sudden / acute / sub-acute/ insidious

COURSE -- Progressive (slow/rapid)
-- Regressive
-- Stationary
-- Episodic

DURATION -- hours, days, weeks, months, years

TREATMENT taken and its response

NOTE: The natural history of any disease falls into one of the four patterns which have to be recognized and each of these patterns has one or more etiologies each (rarely more).

Pattern ONE:

The diseases in this have a **SUDDEN ONSET** (all symptoms occurring within a few hours) and this pattern has only two causes: -

Trauma and Vascular disease.

Pattern TWO:

These are diseases of **ACUTE ONSET** (all primary symptoms occurring within one or two days) and having a **PROGRESSIVE COURSE**. They are often self limiting or show a good response to treatment. This pattern is seen in: -

Acute Infections

Pattern THREE:

This pattern is characterized by a **SUB-ACUTE ONSET.** All primary symptoms develop within a week or so and have a **PROGRESSIVE COURSE** with duration of usually less than six months at diagnosis. This pattern is seen in: -

Chronic Infections and Malignancies

Pattern FOUR:

These diseases have an **INSIDIOUS ONSET** (All primary symptoms occur over a time period of more than two/three weeks) and a **LONG DURATION** (often more than six months). This is found in: -

Chronic diseases often Hereditary/ Genetic/ Metabolic/ Degenerative in Nature

A subtype of this pattern is one with an **EPISODIC COURSE** and then the etiology is an Electrical dysrhythmia (in the Heart or Brain) as in Epilepsy, Cardiac arrhythmias, Autoimmune as a recurrent autoimmune phenomenon and Reticuloendothelial diseases. Mechanical problems like calculi are also recurrent.

It must be stressed that this pattern recognition needs to take an overview of the Onset, Course, Duration and Response to Treatment and any one of these four is not paramount.

A.4 BACKGROUND HISTORY

Relates to the etiology of disease

This includes: -- Past history
-- Family history
-- Personal history (this can be gone into as great details as needed)

Included in this heading are habits, occupational, obstetric, sexual and socio-economic histories.

A.5 PATHOPHYSIOLOGICAL ABNORMALITY AND DYSFUNCTION MEASUREMENT

What is the loss of normal working of the patient?
This must be assessed in relation to:

-- Daily activities
-- Occupational needs
-- Recreational requirements

Does the patient have any of the known pathophysiological syndrome of the system?

NOTE: Assessment of points A.4. (Background history) and A.5. (Dysfunction) also helps in deciding the modes of therapy, how much the patient needs to be told about his illness and in establishing rapport.

B. GENERAL EXAMINATION

A carefully directed general examination should be aimed at providing the answer to the following THREE questions: B1, B2 and B3

B.1 VITAL SIGNS AND ANY OBVIOUS ABNORMALITIES

This would include -- Pulse
-- Blood Pressure
-- Respiration
-- Consciousness
-- Any other striking features, including facies

NOTE: Abnormalities of Vital Signs signify advanced disease. This may be due to a Primary disease in any system but is related pathophysiologically to the Heart, Lungs or Nervous System which are affected currently.

B.2 ETIOLOGICAL CONFIRMATION

Is there any finding which corroborates the etiology? e.g,
-finding hard fixed lymph glands in a case of malignancy, or
-symmetrical, large joint swellings in Rheumatic heart disease, or
-Cafe-au-lait spots in spinal cord compression

B.3 PATHOPHYSIOLOGICAL DISTURBANCE PRESENT AND MEASUREMENT OF DYSFUNCTION

What is the result of objective assessment of dysfunction?

Are there any specific pathophysiologic syndromes of the involved system (e.g., Respiratory failure in chronic bronchitis, or Congestive Cardiac Failure in mitral stenosis) present?

C. SYSTEMIC EXAMINATION

Of systems other than the one primarily affected.
This should tell us:

C.1 Has the same disease affected other systems earlier and then caused the present problem?

E.g. In Congenital heart disease, there may be other congenital anomalies, or
Pulmonary tuberculosis may precede the involvement of kidneys by the mycobacteria.

C.2 Whether other systems are affected by the disease, such as,
Polycythemia in Chronic Lung Diseases, or Thrombo-embolic episode in Mitral Stenosis with atrial fibrillation, or Gastric hemorrhage in cerebro-vascular stroke.

D. SYSTEMIC EXAMINATION OF THE AFFECTED SYSTEM

This should provide the answer to the following two questions, D1 and D2.

D.1 Which site and sub-site is/are involved?

Each system has different sites and sub-sites which can be involved in any disease process and these will be enumerated with each system.

D.2 Can the site, sub-site involved or the permutations and combinations of the aforementioned tell us the possible disease responsible?
E.g, Anterior horn cell is only affected by Polio and Motor Neurone Disease whereas pure motor spastic paraplegia has only a few well known causes.

E. INVESTIGATIONS

These should be able to answer the following queries. E1, E2, E3 and E4.

E.1 What **SITE/SUB-SITE** is affected?

E.2 What is the **CAUSE (NATURE)** of this process?

E.3 Presence and measurement of any **PATHOPHYSIOLOGICAL SYNDROMES** such as measuring CVP **in** a doubtful CHF, Pulmonary Wedge pressure in left heart disease.

E.4 What is the **FUNCTIONAL DISABILITY** produced? Can we quantify this disability and decipher its pattern? E.g. Spirometry in COPD

NOTE: The investigations should be mainly directed towards delineation of Site, Nature, measurement of pathophysiological alteration and quantification of dysfunction.

Some of these 4 points may become clear from a thorough clinical examination and only need confirmation by investigations, whereas the rest may need to be investigated as they could not be determined by Clinical Analysis. This could determine the selection of tests to be done.

CARDIOLOGY

The common anatomical site/s where diseases occur are listed in a box and so are the common etiologies of disease and the pathophysiological syndromes encountered.

SITE OF DISEASE

1. Pericardium

2. Myocardium

3. Endocardium: Valvular

4. Pancardium: Rheumatic Fever, Trauma

5. Vascular : *Artery*
: *Vein*
: *Lymphatic*

6. Electrical Pathways

SYNDROMES OF DYSFUNCTION

When the heart is not working properly it can result in the following pathophysiological syndromes:

1. Congestive Heart Failure
2. Cardiac Asthma
3. Low output Syndrome or Shock
4. Arrhythmia
5. Bacterial Endocarditis supervening on diseased Valves/ Shunt/ Artificial valves

ETIOLOGY OF CARDIAC DISEASE

(Note must be made of the common ones linked to a sub-site listed in the first box)

COMMON

1. Congenital
2. Rheumatic
3. Hypertensive
4. Infectious
: *Pericardial ~ Tuberculosis, Viral*
: *Myocardial ~ Virus, Rickettsia*
: *Endocardial ~ Subacute bacterial endocarditis*
5. Atherosclerotic

UNCOMMON

1. Collagen
2. Endocrinal
3. Immune Disease
4. Others

CARDIOLOGY – THE COMMON PLAN

A. HISTORY: As in the common, this part will answer questions A1-5.

A.1 SYMPTOMS SUGGESTING CARDIO VASCULAR INVOLVEMENT

-- Central chest pain esp. on exertion
-- Dyspnea on exertion
-- Nocturnal Dyspnea
-- Palpitation
-- Swelling over dependent parts

A.2 Symptoms suggesting PATHOPHYSIOLOGICAL EFFECTS

Most of the above symptoms occur because of the effects of primary disease on circulation. The symptoms of the disease per se may be few or none and the symptoms due to the consequent pathophysiological changes predominate or even predate.

A.3 CAUSE OF DISEASE

One has to consider the onset, course duration and the response to treatment in totality to reach the probable cause of disease. A chart depicting these will help as a guideline in the history. It needs to be re-emphasized that all these points have to be taken together to arrive at a decision.

CAUSE	ONSET	COURSE	DURATION	OUTCOME
VASCULAR	*Sudden*	*Rapidly Progressive or regressive*	*Few days*	*Good/ Bad*
RHEUMATIC	*Acute*	*Episodes of fever + Prophylaxis*	*Years*	*Variable*
TUBERCULAR	*Sub- Acute*	*Progressive*	*Months*	*Good with treatment*
CARDIOMYOPA - THY	*Sub- Acute*	*Slowly Progressive*	*Months/ years*	*Bad*
CONGENITAL	*Since birth*	*Progressive*	*Variable*	*Good in milder forms*
ARRYTHMIA	*Acute*	*Episodic*	*Years*	*Variable*
JOINT DISEASE	*Sub- Acute*	*Slowly Progressive*	*Years*	*Acute exacerbation problematic*

A.4BACKGROUND HISTORY: NATURE OF DISEASE CAN BE ELUCIDATED

- Congenital disease : There may be an antenatal history of
  - -- Disease in the mother like Rubella
  - -- Birth trauma
  - -- Delayed milestones
  - -- Squatting, crying spells
  - -- History of other anomalies

- Rheumatic disease
  - -- Chorea
  - -- Fleeting arthritis

- Atherosclerosis
  - -- Family history of diabetes, premature deaths in the family
  - -- Past history of other organ involvement by atherosclerosis
  - -- Diabetes mellitus
  - -- Smoking
  - -- Blood pressure

- Syphilis
  - -- Past, family or personal history pointers to syphilis
  - -- Present history of primary chancre
  - -- Past history of primary chancre or signs of secondary syphilis
  - -- Signs of Syphilis in the sexual partner and in congenital syphilishistory of still births/abortions of siblings
- Tuberculosis
  - -- Family history of Tuberculosis

- Hypertension
  - -- Past history of other organ involvement
  - -- Family history
  - -- History of renal diseases
  - -- History of Endocrinal diseases

A.5 PATHOPHYSIOLOGICAL ABNORMALITIES AND DYSFUNCTION MEASUREMENT

~ Edema may suggest CHF
~ Nocturnal dyspnea suggests cardiac asthma
~ Episodes of unconsciousness may suggest arrythymias
~ Prolonged fever may indicate bacterial endocarditis
~ Cold extremities and muscle fatigue may suggest low output syndrome

In assessing dysfunction, the degree of breathlessness is to be noted, keeping in mind his job requirement and personal situation which includes details of his residence,how many stairs he has to climb, what are his hobbies & other recreational activities and the disturbance of function should be measured against these parameters.
The NYHA grade of dyspnoea must be recorded.

B. GENERAL EXAMINATION: This part will provide answers to B1-3.

B.1 VITAL SIGNS AND OBVIOUS ABNORMALITY

A measure of the vital signs including pulse, blood pressure, respiration and temperature has to be made.

Any remarkable abnormal facies recorded. This may include mitral facies or Pixie like facies in pulmonary stenosis etc.

It should be highlighted that the arterial pulse and blood pressure reflect hemodynamic changes of the left side of the heart especially aortic valve, left ventricle and to lesser extent the mitral valve.

The neck veins reflect right sided heart hemodynamics including tricuspid valve, right ventricle and to the lesser extent the pulmonary valve and pulmonary artery pressures.

B.2 ETIOLOGICAL SIGNS

~Congenital anomalies of the fingers, eyes, cleft lip, webbing of the neck etc.

~Signs of Rheumatic fever

~Evidence of other joint diseases like rheumatoid arthritis, ankylosing spondylitis, syphlis

~Pre-mature atherosclerosis in the form of thickened arteries and other ophthalmic fundus abnormalities
~Renal and endocrinal diseases

B.3 PATHOPHYSIOLOGICAL ABNORMALTIES AND MEASURING DYSFUNCTION

-- The grade of dyspnoea should be measured on an NYHA scale of 1 to 4.
-- CHF (including tricuspid regurgitation)
-- Left ventricle failure
-- Low output syndrome/shock
-- Arrhythmia and fainting
-- Bacterial endocarditis

C. SYSTEMIC EXAMINATION OF OTHER SYSTEMS (EXTRA-CARDIAC)
Should provide information regarding the Cause (etiology C1) and Effect (pathophysiology C2)

C.1 CAUSE OF DISEASE

Cardiac diseases may develop after diseases of other organs and as an example, efforts should be made to look for tuberculosis in the other systems, as well as Rheumatic fever and Rheumatoid arthritis have to be looked for.

C.2 EFFECTS ON OTHER SYSTEMS

-- Lung: basal crepts in heart failure
-- Kidneys: microscopic haematuria
-- Spleen: enlargement in CHF and SBE

D. SYSTEM EXAMINATION – CARDIAC: Should give us information as to what anatomical structures (Site and Sub-sites) are affected by the disease.

D.1 SITE OF INCOLVEMENT IN CARDIO-VASCULAR SYSTEM

(a) Pericardial involvement:
-- Rub
-- Signs of effusion
(b) Myocardial diseases
-- Usually silent
-- Arrhythmia
-- CHF/LVF
-- Mitral/Tricuspid Regurgitation
(c) Endocardial diseases
-- Murmurs
-- Stenosis/regurgitation
-- Congenital shunt
-- Cyanotic/acyanotic
(d) Peripheral vascular diseases

D.2 NATURE INDICATORS

The site of involvement sometimes guides us to the cause of diseases.

~Pure M.S. is rheumatic
~Multiple valve involvements usually rheumatic
~Pure A.R. is often due to connective tissue disorder

E. INVESTIGATIONS

An investigation may give us structural as well as functional information however radiology usually gives us structural information (site).

Functional information is often obtained from dynamic studies including Echocardiography, Radioactive scanning and Angiography.

E.1 CONFIRMATION OF SITE OF DISEASE

(a) Pericardial diseases:
--Skiagram may show calcification
-- Catheterization can show thickness of walls
-- Echocardiography helps in diagnosis of effusion

-- Biopsy and aspiration give us etiological information

(b) Myocardial disease

-- EKG shows us the pattern of myocardial ischemia
-- Coronary angiography rules out Ischemic narrowing
-- Endocardial biopsy is helpful in etiology

(c) Endocardial disease (valves)

-- Chest skiagram
-- Barium swallow
-- Echocardiography
-- Catheterization and angiography
-- EKG for various chamber hypertrophies

(d) Blood vessels

-- Doppler studies
-- Angiography
-- Venography
-- Carotid Intima-Media thickness- this quantifies risk of both ischemic heart events and cerebral ischemic events
-- Testing for Endothelial Dysfunction

E.2 ETIOLOGICAL CONFIRMATION

- Rheumatic -- ASO titres
  -- C-reacting proteins
  -- Throat swab and culture

- Tuberculosis -- Pericardial biopsy and fluid testing
  -- Tuberculosis elsewhere

- Joint diseases -- Rheumatoid factor
  -- Anti nuclear antibodies
  -- Other antibodies
  -- Skiagram of joints

- Congenital diseases -- Chromosomes studies
  -- Biochemical enzyme estimation
  -- Skiagram of joints

E.3 PATHOPHYSIOLOGY AND DYSFUNCTION MEASUREMENT

-- Treadmill exercise testing
-- Systolic time intervals
-- Echo cardiography

-- Catheterisation
-- Radio nuclide assessment of ventricle function

These can be done before and after exercise or certain drugs.

E.4 MISCELLANEOUS

Risk factors assessment for pre-mature Coronary Artery Heart Disease

-- Lipids
-- Blood sugar
-- Serum uric acid
-- Lipoprotein A levels
-- Homocysteine levels
-- hs CRP levels
-- Carotid Intima-Media thickness (CIMT)
-- Endothelial Dysfunction studies
-- Ankle Brachial Index
-- Coronary Artery calcium Scores
-- Intravascular Ultrasonography

-- Catheterisation
-- Radio nuclide assessment of ventricle function

These can be done before and after exercise or certain drugs.

E.4 MISCELLANEOUS

Risk factors assessment for pre-mature Coronary Artery Heart Disease

-- Lipids
-- Blood sugar
-- Serum uric acid
-- Lipoprotein A levels
-- Homocysteine levels
-- hCRP levels

SITE OF DISEASE

1. Upper Respiratory tract
2. Trachea
3. Bronchi
- Large
- Small
4. Alveoli
5. Interstitial Tissue
- Collagen
- Vessels
6.Pleura

SYNDROMES OF DYSFUNCTION

1. Respiratory Failure
- Hypoxemic
- Hypercapnic
2. Pulmonary hypertension and CHF
3. Cardiac Arrhythmias

ETIOLOGY OF RESPIRATORY DISEASES

1. Infections
2. Allergy
3. Malignancy
4. Occupational Diseases
5. Collagen diseases
6. Degenerative diseases
7. Congenital

DIAGNOSTIC WORKUP OF RESPIRATORY SYSTEM CASE ACCORDING TO THE COMMON PLAN

A. HISTORY

A.1 Symptoms suggesting respiratory involvement

1. Cough with expectoration
2. Breathlessness at rest (may increase on exercise)
3. Lateral chest pain
4. Hemoptysis
5. Audible wheeze

SYMPTOM ANALYSIS

1. COUGH:

~ In Upper respiratory tract involvement, cough is generally dry or with very sticky sputum which is difficult to bring out along with soreness and throat irritation.

~ Laryngeal paralysis if it is "Bovine" type and ineffectual
~ Tracheal cough is loud and brassy

~ Bronchial cough is a deep cough with expectoration (mucoid type)

~ Alveolar coughing is commonly associated with dyspnoea and has frothy sputum

~ Pleural cough is painful and suppressed without expectoration unless there is a bronchopleural fistula

TIMING OF COUGH:

~ Nocturnal Cough can be due to
- Post-nasal drip
- Gastro-esophageal Reflux Disease
- Bronchiectasis
- Cardiac failure

~ Morning cough is seen in
- Chronic bronchitis
- Bronchiectasis

~ Post-meal cough may be due to
- Tracheo-esophageal fistula
- Hiatus hernia
- Aspiration
- Esophageal diverticulum

~ Precipitated by change in posture in
- Lung abscess
- Bronchiectasis

EXPECTORATION:

- Mucoid in Upper respiratory tract or tracheo-bronchial tree

- Frothy in Alveolar disease, especially in cardiac asthma

- Dry cough in Pleural diseases, early disease, upper respiratory tract disease and people who swallow sputum (like children)

- *Purulent* in Lung abscess, bronchiectasis and bronchopleural fistula
- *Rusty* as in Pneumonia
- *Foul odour* in Anaerobic infection
- *Bronchial casts* can be seen in Chronic bronchitis and bronchial asthma

2. DYSPNOEA

This can be respiratory, cardiac or psychological. Respiratory mechanisms can be:-

~ Inspiratory obstruction
~ Bronchospasm
~ Consolidation
~ Emphysema
~ Pleural effusion
~ Pneumothorax

The site of disease in respiratory system can often be told by associated symptoms/signs.

- Dyspnoea with inspiratory stridor occurs in Foreign body
- Wheeze is audible in Bronchitis and Asthma
- Nocturnal increase in dyspnoea is Cardiac (due to alveolar congestion)
- Shallow breathing is seen in Neuromuscular paralysis

3. LATERAL CHEST PAIN

This is the hallmark of pleural disease.
It has to be differentiated from musculoskeletal pain by the absence of other respiratory symptoms in the latter. Diaphragmatic pleurisy may be referred to the tip of shoulder and maybe associated with an increase during deep breathing and coughing.
Tracheitis may also be painful but the pain is in the front of neck and retrosternal.

4. HEMOPTYSIS

This symptom gives a lot of information about the site of involvement and sometimes helps in the etiological diagnosis as the causes of hemoptysis at each sub-site of the respiratory system are few and many diseases have their distinctive characteristics.

~ Upper Respiratory Tract often gives a Streaky hemoptysis

~ Alveolar origin of hemoptysis is often Frothy and is a hallmark of pulmonary edema

~ *Frank* blood can be seen in tuberculosis, mitral stenosis and bronchial adenoma

~ *Mucopurulent* hemoptysis is seen in bronchiectasis and lung abscess

~ *Rusty* hemoptysis is seen in early pneumonia

~ *Sudden* onset suggests pulmonary embolism and infarction

~ *Recurrent* hemoptysis occurs in hemosiderosis, Goodpasture's syndrome and bronchial adenoma

~ *Continuous* bleeding can be seen in malignancy

A.2 Are there any symptoms suggestive of pathophysiological effects of the disease?

- Tremulousness, drowsiness and coma in Respiratory Failure
- Pitting edema, right upper abdominal discomfort in CHF
- Palpitations in arrhythmias

A.3 Cause of Respiratory Diseases

The common causes are:
- Acute infections
- Chronic infections
- Malignancy
- Degenerative diseases (like Emphysema)
- Immunological diseases, common being asthma

Less common ones being
- Trauma
- Congenital
- Occupational and dust diseases-
- Vascular diseases (pulmonary embolism)

DISEASE	ONSET	COURSE	DURATION	TREATMENT RESPONSE
ASTHMA	*Acute / Chronic*	*Episodic*	*Years*	*Good for acute attack*
ACUTE INFECTION	*Acute*	*Progressive then Regressive*	*Days/ Weeks*	*Good*
CHRONIC INFECTION	*Sub-Acute*	*Slowly progressive*	*Months/ Years*	*Fair*
MALIGNANCY	*Sub-Acute*	*Rapidly Progressive*	*Months*	*Bad*
DEGENERATI - VE	*Insidious*	*Very Slowly Progressive*	*Years*	*Poor in long term*

As in all systems differentiation between these possibilities lies in analyzing the mode of onset, course, duration and response to treatment, if any. The table below highlights these for the common ones:

A.4 BACKGROUND HISTORY

~ Acute infections
- Present in others in family
- Endemic/epidemic in community

~ Tuberculosis
- Family history + debilitating disease
- Overcrowding
- Undernutrition

~ Malignancy
- Personal history of smoking
- Occupational history of exposure to asbestos or Polyvinyl chloride

~ Degenerative
- Family history of similar illness

~ Bronchial asthma
- Past history of atopy, eczema, rhinitis
- Family history of atopy, eczema, rhinitis, allergic pharyngitis, hay fever

A.5 What is the disturbance of function?

Once again this is considered in context of a patient's daily activities, occupation and recreational pursuits.
- Coughing can interfere with these
- Breathlessness can cause problems and depending on its severity can cause variable degree of problems.
- Hoarseness may cause untold misery in a professional singer or teacher

B. GENERAL EXAMINATION

B.1 What is the grade of dyspnoea 1-4 (MMRC grade)

- Is there Respiratory Failure?
- Presence of Cyanosis, clubbing, flapping tremors, drowsiness or papilledema
- Is there Cor pulmonale?
- Chest expansion must be measured

B.2 Any evidence of etiology

- Lymph nodes: size, feel, fixity, matting, warmth
- Clubbing: signifying suppuration
- Skin/joint affection in collagenoses

B.3 General parameters

- Respiratory rate, depth, nature and pattern
- Pulse and blood pressure
- Level of consciousness

C. EXAMINATION OF OTHER SYSTEMS

C.1 Cause of respiratory disease is in other organ/s such as-

~ Primary cancer elsewhere causing secondaries in lung
~ Tuberculosis elsewhere
~ Multisystem involvement in collagen disorders, sarcoidosis

C.2 Affect of lung disease on other system (CONCEPT OF LUNG-HEART-BLOOD AXIS)

~ Cor pulmonale: Right Ventricular hypertrophy and/or failure
~ Polycythemia

D. EXAMINATION OF RESPIRATORY SYSTEM

D.1 This examination should be able to tell us which part/s of the lungs is/are involved and how. SUMMATION of the physical signs in the lung tells the ALTERED PHYSICAL STATE and the common ones (in alphabetical order) are :-

a. Atelectasis, Asthma
b. Bronchitis, Bronchiectasis
c. Cavity, Collapse, Consolidation
d. Emphysema, Empyema*
e. Fibrosis: Interstitial as well as Replacement
f. Hydrothorax*, Haemothorax*, Hydropneumothorax
g. Pulmonary edema
h. Pneumothorax, Pleural effusion(*similar signs)

AS THESE ALTERED PHYSICAL STATES DO NOT TELL US THE ETIOLOGICAL DIAGNOSIS

TWO IMPORTANT FACTS MUST BE REMEMBERED

- ONE :- Any respiratory disease can give rise to different signs (altered physical states) in the lung e.g. T.B. can cause cavity, collapse, effusion

- **TWO :-** Many different diseases can give rise to the same physical signs (altered physical states) e.g. Consolidation can be due to Pneumococci, Mycobacteria, Fungi, or Malignancy

D.2 DISTRIBUTION OF ALTERED PHYSICAL STATES

UNILATERAL

1. APICAL
(a) Tuberculosis
(b) Bacterial (Others)
(c) Fungal
(d) Malignancy

2. BASAL
(a) Bacterial Pneumonia
(b) Malignancy
(c) Fungal
(d) Tuberculosis

3. WHOLE LUNG
(Various causes)
(a) Pleural effusion
(b) Collapse
(c) Consolidation
(d)Fibrosis
(e)Pneumothorax

BILATERAL

1. APICAL
Tuberculosis

2. BASAL
(a) Pulmonary edema
(b) Bronchiectasis
(c) Interstitial fibrosis

3. UNIVERSAL
(a) Bronchitis
(b) Emphysema
(c) Asthma
(d)Tropical Pulmonary Eosinophilia
(e) Interstitial fibrosis
(f) Occupational diseases
(g) Allergic alveolitis

Hence, in lung diseases the two points to be determined are what is the altered physical state and where is it. This would give us the probable etiological causes.

E. INVESTIGATIONS

E.1 For SITE and confirmation of the Altered Physical State

- Various Skiagrams help (PA View, AP View, Laterals, Lordotic Views and Tomograms)
- Bronchography for the distal smaller bronchi
- Bronchoscopy* for trachea and larger proximal bronchi
- CT Scan (with high resolution- HRCT)

E.2 Etiological confirmation needs a specimen from the affected site. All this material is subjected to gram stain, AFB smear, culture, PCR, BACTEC, Pap stain, fungal elements, eosinophils, asbestos bodies.

- Sputum
- Swab (thru Bronchoscope)*
- Trans-tracheal aspirate
- Pleural aspirate
- Scalene/other node biopsy
- ENE Expert

} *All this material is subjected to gram stain, AFB smear, culture, PCR, BACTEC, Pap stain, fungal elements, eosinophils, asbestos bodies and sensitivity tasting*

E.3

- Is there corpulmonale present : Right Ventricular Hypertrophy or strains on ECG Chest skiagram showing right ventricular hypertrophy pattern
- Is there respiratory failure : Blood gases in the arterial sample
- Occasionally cardiac arrhythmias and polycythemia can occur consequent to respiratory illness and this need to be investigated.

E.4 Timed walking capacity is a measure of respiratory function. A 12 minute walking distance has often been used for this purpose and serially performing such a test can be an assessment tool for disease progression.

- **Sleep Studies**

NEUROLOGY

Site of Disease in the Nervous System

INTRACRANIAL

SUPRATENTORIAL	INFRATENTORIAL
1. Frontal Lobe	8. Mid Brain
2. Temporal Lobe	9. Pons
3. Parietal Lobe	10. Medulla
4. Occipital Lobe	11.Cerebellum
5. Basal Ganglia	
6. Thalamus	
7. Internal Capsule	

EXTRACRANIAL

1. Motor: Upper motor Neuron: (a) Pyramidal
   (b) Extrapyramidal
   Lower Motor Neuron
2. Sensory: (a) Posterior Columns
   (b) Lateral Columns
3. Cerebellar connections
4. Autonomic function: Lateral horns

COMMON ETIOLOGIES OF DISEASE IN NEUROLOGY

1. Trauma
2. Vascular diseases: Hemorrhage
   Thrombosis
   Embolism
3. Acute Infections
4. Chronic Infections
5. Malignancy
6. Benign Tumors
7. Degenerative/ Metabolic/Hereditary/ Genetic Diseases
8. Congenital diseases
9. Immunologically Mediated Diseases

COMMON PATHOPHYSIOLOGICAL ABNORMALITIES

NOTE: These are closely linked to the Sites mentioned in Box 1. and will not be described separately.

In the Nervous System each structure has a well defined function and indeed it is this loss of function which tells the site of disease. The Nervous system is not amenable to direct Examination by Inspection, Palpation, Percussion and Auscultation as the other Systems are.

COMMON PLAN AS APPLIED TO NERVOUS SYSTEM DISEASES

A. HISTORY

A.1 SYMPTOMS SUGGESTING C.N.S. INVOLVEMENT

- Motor -- Weakness
  -- Abnormal movements
  -- Wasting of muscle
- Sensory -- Tingling
  -- Numbness, hyperesthesia
- Special -- Fits
  -- Speech and gait disorders
  -- Coma

Miscellaneous -- Endocrinal manifestations like Cushings and Acromegaly

A.2 HISTORY: CAUSE OF DISEASE ONSET

1. Sudden onset -- Trauma/vascular*
2. Acute onset -- Acute infections
3. Sub-acute onset -- Chronic infections/ malignancy
4. Insidious onset -- Degenerative disorders

*Other causes may also lead to sudden onset through vascular involvement like in malignancy or infections, and the development of arteritis.

COURSE

- Progressive -- Chronic infections/ malignancy
- Slowly progressive -- Degenerative
- Regressive -- Acute infections

- Stationary -- After trauma/residual damage of infections
- Episodic -- Epilepsy/T.I.A.

DURATION

Malignant tumors do not last for more than 2 years despite all treatment. Duration is fairly established for various degenerative diseases. The common age of onset of various degenerative disorders is also known.

A.3 BACKGROUND HISTORY :

1. Congenital diseases
   - Presence of defect since birth
   - Congenital adverse factors during early pregnancy
   - Similar problems in siblings and other family members

2. Acute infections
   - Previous history of ear, nose and throat disease
   - Previous history of debilitating diseases like diabetes, cancer.
   - Epidemic/endemic diseases prevalent

3. Chronic infections
   - Past history of other organ involvement
   - H/o debilitating diseases and diminished immunity

4. Malignancy -- Nothing specific, but in secondary malignancy history of primary may be there

5. Degenerative disease

   - Family history is significant
   - Exposure to toxic material, occupational or environmental

6. Epilepsy
   - Trauma during childbirth
   - Family history positive
   - Personal history of stress precipitating it
   - Past history of parasitic diseases ENT diseases

7. Vascular
   - Previous history of heart diseases, Diabetes, Hypertension
   - Previous history of smoking
   - Oral contraceptive use in females
   - Family history of vascular events

A.4 HISTORY: DYSFUNCTION

- Disturbance of function: The patient himself (or family members) reports it
- Motor involvement : Paraplegia, Hemiplegia, Quadriplegia, Diplegia, Cranial nerve involvements
- Sensory involvement: Various modalities
- Specially coordinated functions: Gait, Speech, Abnormalities of consciousness
- Epilepsy

B. GENERAL EXAMINATION

B.1 APPEARANCE AND VITAL SIGNS

- Level of consciousness can be judged objectively and graded.
- Look for -- Hypertension, Fever, Respiratory rate and abnormality of its pattern

B.2 ETIOLOGICAL SIGNS

- Other congenital anomaly/anomalies
- Macro/micro cephaly
- Other skull deformities
- Lymphadenopathy / Subcutaneous masses
- Spinal bifida
- Neurofibromata
- Meningomyelocele
- Skin lesions -- Measles, Mumps, Chicken pox etc. Café-au-lait spots, skin lesions of secondary syphilis

B.3 Grading:

- Grading of muscle power
- Grading of coma - Will be covered in CNS examination

C. SYSTEMIC EXAMINATIONS OF OTHER SYSTEMS

C.1 EVIDENCE OF ETIOLOGY

-- Tuberculosis
-- Primary tumors
-- Secondaries in bones
-- Congenital anomaly of other organs
-- Metabolic disorder of spleen an liver
-- Premature atherosclerosis
-- Diabetes, Hypertension, Heart disease
-- Teeth and ENT for pyogenic focus

C.2 EFFECT OF NERVOUS SYSTEM DISEASE ON OTHER SYSTEMS

-- Cardiac arrhythmia
-- Gastric hemorrhage

D. EXAMINATION OF NERVOUS SYSTEM

This aims to find site, sub-site and sub-sub sites of lesions. Also primary and secondary sites, e.g. Mitral stenosis with Atrial fibrillation leading to embolism and syndrome of Middle Cerebral Arterial Occlusion.

Physical examination only indirectly reveals site as what we examine is dysfunction in a highly specialized organ and dysfunction is unlikely to pinpoint the site affected. (The actual change in respiratory system, for example, a consolidated lung can be diagnosed by physical examination but except for the peripheral nerves and retina, rest of the central nervous system is enclosed in membrano-fibro-boney chamber and cannot be directly examined.)

The study of dysfunction which is the only method for assessing site of disease is divided into the higher functions and spinal functions.

In examination of those functions which do not depend upon the integrity of a single modality alone like gait, speech, consciousness, localization is less precise; but if found normal does rule out significant disease of most of the nervous system.
Cranial nerve involvement is more specific but supranuclear VII, XII & III, IV & VI can be affected through indirect mechanisms and does not necessarily indicate the brain stem as the site of disease.

- Spinal functions including:
  - UMN
    - Pyramidal
    - Extrapyramidal
  - LMN
    - Anterior Horn Cell
    - Nerve
  - Sensory
    - Lateral column
    - Posterior column
- Cerebellar
- Autonomic Nervous System

These modalities can be affected "Vertically" from cerebral cortex down to the lowest spinal segment and then along the peripheral nerve up to the myoneural junction. In this regard, localization of the vertical site is most definite if there is involvement of LMN > UMN > Sensory.

Extrapyramidal and cerebellar involvement is less helpful in localization of site. Absence of a single deep tendon reflex and wasting are really important LMN signs. Similarly, the association of a segmental or nerve distribution of sensory involvement is very helpful. UMN deficit has an indeterminate pattern. From T-1—L-1, sensory involvement may not match the site. They may also be affected horizontally (as in transeverse myelitis many modalities may be affected).

D.1 SITE LOCALISATION

A. What **higher functions** are involved? Can they help to localize the site of lesion?

- ✓ Vertical
- ✓ Horizontal
- ✓ Cranial nerve
- ✓ Specified speech & gait disorders

B. Which of the 6 spinal function is involved.(The signs of each are well known)

Is LMN involved -- Where is the wasting
-- Which particular deep reflex is lost

Is pyramidal involved
-- Deep reflex of highest root value involved gives the lowest possible location of disease but a drop level is possible and partial lesions cortex are also known to cause confusion.

Sensory involved -- lateral column upper level of involvement
-- posterior column upper level of involvement
-- Care has to be taken regarding multiple areas of loss, drop levels, skip areas, saddle anesthesia, dissociate loss of modalities, spinal versus vertebral level, difference due to secondary phenomenon (vascular) above or below the site of compression.

Sometimes the pattern of these losses and their permutation/combinations can tell us about the possible causes.

D.2 CAUSES ACCORDING TO SITE OF INVOLVEMENT

Non-selective diseases

Cranium and Vertebra -- Multiple myeloma and secondaries
-- Osteomyelitis
-- Mastoiditis
-- Tuberculosis
-- Trauma

Meninges -- Bacterial
-- Viral
-- Aseptic Meningitis
-- Trauma - Old minimal trauma can cause subdural hematoma
-- Meningioma of Falx, sphenoidal ridge, CP Angle

Cortex and Subcortical

-- Congenital mal development
-- Birth trauma
-- Encephalitis - Can also affect the sub-tentorial region
-- Malignancy -- Primary
-- Secondary
-- Tuberculoma (especially in the posterior-fosa)
-- Other granulomata and cysts
-- Vascular

Multiple causes -- Leading too old scars and Epilepsy

Spinal cord -- Disc,Vascular, Tumours, Infections, melitis etc.

Selective diseases (Degenerative/ Metabolic/ Herditary/Genetic)

-- Parkinsonism -- Extrapyramidal
-- Cerebellar -- Degenerative diseases
-- Pyramidal -- MND, selective vascular involvements
-- L.M.N. -- A.N.C., M.N.D. and Polio
-- Nerve -- Multiple causes
-- Post col-tabes dorsalis

Note : Degenerative diseases refer to those disease where only the clinical phenotype is known and as yet the biochemical disturbance or the chromosome/gene or heredity is not known. Metabolic is used when the underlying metabolic abnormality is known. Genetic is used when the gene/mode of inheritance has been identified

E. INVESTIGATION OF C.N.S.:

The site investigations are primarily directed towards

-- Site of lesion
-- Nature of lesion
-- Disturbance of function

E.1 SITE:

Radiology -- Plain skiagram in various views
Contrast -- Myelography
-- Carotid angiography
-- Basilar angiography
-- Ventriculography
-- Pneumoencephalography

These also tells definite structural phenomenon like atrophy of cerebral/cerebellar hemispheres.

-- CT Scan with or without contrast
-- M.R.I.

E.2 NATURE OF LESION

1. C.S.F. -- Routine
   -- Serology
   -- Smears/cultures
   -- Toxic products
   -- Special tests- trace elements, catecholomines
   -- Metabolities of fat metabolism

2. Nerve and muscle biopsy

3. Biopsy of tissue obtained at craniotomy

4. L.N. Biopsy

5. If secondaries suspected then search for primary site.

6. MRI and PET Scans

E.3 IDENTIFICATION OF PATHOPHYSIOLOGY AND MEASUREMENT OF DYSFUNCTION

1. Brain -- E.E.G.
   -- Evoked potentials

2. Peripheral Nerves
   -- N.C.V.- Motor and/or sensory

3. Myoneural Junction and Muscles
   -- E.M.G.

4. Muscle enzymes

5. C.S.F. pressure

6. MRI and PET Scans

HEPATOBILIARY DISEASE

SITE OF DISEASE IN THE HEPATOBILIARY SYSTEM

1. *Capsule*
2. *Hepatocyetes*
3. *Portal tracts*
4. *Biliary tracts and Gall bladder*
5. *Hepatic veins*
6. *Reticuloendothelial cells*
7. *Collagen tissue*

PATHOPHYSIOLOGICAL SYNDROMES OF THE LIVER

1. **Hepatic cell Failure and Jaundice**
2. **Portal hypertension**
3. **Biliary inflammation and Obstruction**
4. **Others like Coagulation defects, Endocrinal problems**

LIST OF ETIOLOGY OF LIVER DISEASES

1. **Congenital**
   a. **Structural - Reidl's lobe, Cysts, Valves, Strictures**
   b. **Biochemical - Hyperbilirubinemias, Metabolic cirrhosis**

2. **Infections**
   Bacterial -- Acute or Chronic
   Viral
   Ameba and others (endemicity of these should be known)

3. **Neoplasia**
   Primary Hepatoma
   Secondaries from GIT, other sites
   Hematological and RES malignancies
   Gall bladder and Billiary tract malignancies

4. **Collagen diseases**
5. **Autoimmune diseases**
6. **Cirrhosis**
7. **Toxic Drugs, Toxins and Physical agents**
8. **Biomechanical Stones, Balves, Strictures**
9. **Hypersensitivity reactions**

ANALYSIS OF HEPATOBILIARY DISEASES ACCORDING TO THE COMMON PLAN

HISTORY

A-1. SITE OF INVOLVEMENT Involvement of liver and gall bladder is indicated by :

- Jaundice
- Right upper abdominal pain which may increase on deep inspiration and may radiate to the tip of the right shoulder
- Itching
- Fever with chills

A-2. SUB SITES of involvement in the liver are indicated by detailed analysis of the following symptoms :

- Biliary syndrome is indicated if there is fever with chill with colicky pain
- Obstructive jaundice is indicated by presence of scratch marks
- Liver cell failure is characterised by
  - -- Flapping tramors
  - -- Drowsiness (pre coma)
  - -- Gynecomastia
  - -- Loss of body hair
  - -- Diminished libido
  - -- Tremors OR Flaps
  - -- Hemetemesis
  - -- Melaena

NOTE: Some of these may not be complained of by the patient and only picked up on direct questioning or on examination.

A-3 ETIOLOGY OF DISEASE This is told by the :

- Onset
- Course of disease
- Duration of symptoms/signs
- Response to treatment

Acute infections are characterized by :

- Acute onset
- Progressive course
- Good response to treatment
- Short duration

Chronic infections are characterised by :

- Sub acute onset
- Slow progress
- Response to therapy is variable, amebic and pyogenic infections respond whereas chronic persistent viral diseases do not have a good response and the response does not persists if it occurs.

Malignancy

- Sub acute onset
- Progress is rapid,
- Does not respond to treatment
- Duration is never prolonged

Biliary stones

- Acute onset
- Intermittent course with recurrent acute attacks
- Long duration unless treated surgically and even then recurrences can occur

Congenital diseases

- May be present at birth or soon after.
- It may start later in life
- Each individual disease has its own characteristics with varying age of onset and progression
- Some congenital hyperbilirubinemias are intermittent

A-4 BACKGROUND ILLNESSES

Family history may be based on infectious disease and hereditary hyperbilirubinemias.
Past history of drug intake, jaundice, treatment with injections, vaccines, blood and blood products

- Personal history
- Alcohol use in detail
- Toxins exposure occupationally or accidentally

A-5 Measurement of dysfunction has to be analyzed in relation to a person's

- Occupational needs
- Daily activities' requirements

GENERAL EXAMINATION

B-1 VITAL SIGNS to be assessed :

- Blood pressure
- Pulse
- Respiration
- Consciousness

B-2 ETIOLOGICAL CORRELATES

- Skin pigmentation of Porphyrias
- Keyser-Fleischer ring is seen in the eyes (this is sometime visible to the naked eye)
- Primary melanoma of the skin may have secondaries in the liver
- Skin and eye changes can be seen in various collagen and other immunological disorders

B-3 IDENTIFICATION OF PATHOPHYSIOLOGICAL SYNDROMES AND THEIR QUANTIFICATION

- Edema and generalised anasarca
- Record of body weight
- Flapping tremors
- Behavioral abnormalities

EXAMINATION OF OTHER SYSTEMS

C-1 Disease in other system which may be leading to the Liver problems. Pericardial disease and chronic congestive heart failure can lead to Cardiac Cirrhosis. Mucoviscidosis can cause pancreatic failure. Occult colonic primary malignancy or Stomach cancer may spread to the liver.

C-2 Liver disease may affect other systems.
Burst liver abscess can effect pleura, lung or pericardium.
Cirrhosis may predispose to hyper acidity and ulcers.
Reticulo-endothelial disease can affect the spleen and lymph nodes.
Hematological malignancies can also cause enlarged lymph nodes and spleen in addition to an enlarged Liver

ABDOMINAL EXAMINATION: System Involved

D-1 Liver enlargement has many causes and these can be listed based on the liver consistency, surface, presence of tenderness, redness or edema.

Friction rub denotes capsulitis and can be found in Hemangioma, after a biopsy and after Gonococcal peri-hepatitis in women.

Splenomegaly and ascites
Splenomegaly is also seen in RES disease

D-2 A short list of possible causes can be made on the above mentioned
basis and can look for causes of enlarged liver

- A firm liver
- Tender liver
- Liver with splenomegaly
- Liver with Ascitis
- Permutations and combinations of the above

INVESTIGATIONS

E-1 SITE CONFIRMATION:

Ultrasonography has emerged as the first choice investigation for the liver and portal tracts because:-

- Can localize the disease
- Diffuse disease can be diagnosed by changes in echo pattern
- Portal vein size and growth at the porta hepatis can be seen
- Gall bladder is well visualised
- ERCP is often needed to supplement information about lower biliary tracts (this procedure has therapeutic indications also)

E-2 TESTS FOR ETIOLOGY

As no test material from the liver is directly available. Indirect methods of testing are used.

- Blood test for indirect effects of infection like Widal tests
- Immunological markers of various viruses
- Tests for Metabolic disease
- Enzyme estimations like Ceruloplasmin in Wilson's disease
- Serum Iron and Iron binding capacity for Hemosiderosis
- Material from the liver and gall bladder can be obtained from

  -- Duodenal aspirate
  -- Biliary tract cannulation
  -- Per operative sample

All material so collected can be examined, cultured and cell morphology identified.

- Biopsy is a very important part of etiological assessment.

E-3 FUNCTIONAL ASSESSMENT

Liver function tests give us an overview.

Portal pressure can be assessed by

-- Size of vein at porta hepatis
-- Extent of varices on endoscopy

Gall bladder functioning can be assessed by

-- Oral Cholecystography
-- Ultrasonography after a fatty meal also gives us a functional assessment of the gall bladder.

RENAL DISEASE

The common anatomical sites and sub-sites where diseases occur, the common Etiologies and the common Presentations (based on Pathophysiology) are given in the three boxes at the beginning before the discussion of the Common Plan as relevant to renal disease.

SITE OF DISEASE IN THE KIDNEYS

1. *Capsule*
2. *Cortex*
3. *Medulla*
4. *Pelvis and Ureters*
5. *Urinary Bladder and Urethra*
6. *Reno vascular - Artery and vein*

COMMON ETIOLOGIES OF KIDNEY DISEASES

1. **Congenital** — **Structural disease (often innocuous) may give rise to pressure effects and infections like in cysts. Abnormal size and shape of kidney predisposes it to infections.**
2. **Infections**
   - ξ **Acute Bacterial**
   - ξ **Chronic Bacterial**
   - ξ **Filarial**
   - ξ **Schistosomiasis**
   - ξ **Viral**

   "Some Infections affect specific sites like TB of the Renal Cortex"
3. **Neoplasia**
   - ξ **Benign**
   - ξ **Malignancy: Primary malignancies are common and secondaries are rare**
4. **Immunological -- Mainly Glomerular disease**
5. **Collagen disorders -- Glomerular or Vascular**
6. **Toxins -- Commonly cause Acute Tubular Necrosis**
7. **Metabolic -- Diabetes, Gout and Tubular diseases**
8. **Metabolic-Mechanical disease -- Renal Stones**

COMMON RENAL PATHOPHYSIOLOGICAL SYNDROMES

1. *Acute Nephritic Syndrome*
2. *Acute Tubular Necrosis*
3. *Nephrotic Syndrome*
4. *Chronic Interstitial disease*
5. *Pyelonephritis*
6. *Ureteric Syndrome*
7. *Cystitis Syndrome*
8. *End Stage Renal Failure*

COMMON PLAN AS APPLIED TO RENAL DISEASE

HISTORY ANALYSIS

A.1 Symptoms suggesting kidney involvement; classical symptoms are
- -- Ureteric colic
- -- Dull pain of renal capsule involvement
- -- Urinary abnormalities

Indirectly kidney involvement is often suggested as being one of the various pathophysiological stages like
- -- Anarsarca
- -- Hypertension
- -- Incessant vomiting
- -- Pulmonary edema
- -- Bone diseases with fractures

Detailed analysis of symptoms gives us the pathophysiological basis of symptoms and hence site of involvement. Pain in renal angle signifies involvement or stretching of capsule.
- -- Ureteric Colic is due to stone clots or infected material
- -- Bladder involvement is suggested by
- -- Hypogastric pain/discomfort
- -- Increased frequency of urine
- -- Stranguary (Trigone involved)

Chronic Renal tubular disease often does not give rise to symptoms per se but leads to metabolic abnormalities such as :-
- -- Acidosis
- -- Vitamin D resistant rickets
- -- Electrolyte disturbance

A.2 ETIOLOGY OF RENAL DISEASE

This depends on analysis of the onset, course, duration and response to treatment.

SUDDEN onset of edema (and Proteinuria) has a vascular cause and may be due to
- -- Inferior vena cava obstruction
- -- Renal vein Thrombosis

ACUTE onset of pain signifies
- -- Stone (No fever)
- -- Infection (fever present)
- -- Colicky pain suggests infections or stone in the ureter
- -- Dull pain in renal area suggests kidney infections

SUB ACUTE onset of symptoms with slower progress due to:

- -- Chronic infections: Usually respond to treatment with appropriate antibiotic
- -- Malignancy: progresses rapidly and does not respond
- -- Collagen diseases usually have long duration and remissions and exacerbations
- -- Long duration of renal disease especially if it starts early is often seen in congenital and metabolic disorders

It must be emphasized that the Onset, Course, Duration and response to treatment, all have to be considered together to assess the nature of the illness.

A.3 BACKGROUND HISTORY *provides important clues to the nature of the disease*

PAST HISTORY

- -- Diabetes
- -- Hypertension
- -- Eye and skin involvement can often precede kidney involvement as can collagen diseases
- -- Liver and lymph node involvement can precede the kidney in immunological disease

FAMILY HISTORY

- -- Metabolic diseases often involve many members in a family
- -- Renal failure in siblings may draw attention to the possibility of Alport's Syndrome where nerve deafness is also seen

PERSONAL HISTORY

- -- Living and/or visiting endemic areas of various diseases
- -- Schistosomiasis is prevalent in various countries of Africa and can involve the urinary system

-- Occupational history of aniline dye exposure
-- Smoking predisposes to bladder cancer
-- Analgesic intake can lead to interstitial Nephritis and renal failure should be asked for in patients of renal failure where the cause is not evident

It is important to direct the questions about background illness towards the syndrome present and its putative etiology rather than subjecting the patient to an all encompassing voluminous questionnaire which exhausts both. This logical step-by-step approach narrows down these questions, saves time and is adaptable to computer assisted diagnostic program development.

A.4 Extent of dysfunction caused by illness in the patient's routine life and occupational needs are also to be recorded.

GENERAL EXAMINATION ANALYSIS

B.1 Vital Parameters

These include pulse and blood pressure

-- Respiratory rate and rhythm
-- Level of consciousness

B.2 Etiological Correlates

-- Congenital abnormalities
-- Deafness
-- Ear abnormality
-- Skin involvement
-- S.L.E.
-- Scleroderma
-- Porphyria
-- Gouty tophi
-- Endocrinal syndromes

Recognized by facies and habitus

-- Myxedema
-- Thyrotoxicosis
-- Cushings syndrome
-- Diabetic complications

B.3 Identification of syndromes present and their measurement

-- Edema and anasarca, record weight
-- Flapping tremors (A record of patients writing daily can provide a measure of early encephalopathy/ tremor activity)
-- Dry skin
-- Coated tongue
-- Pallor

EXAMINATION OF OTHER SYSTEMS

C.1 Diseases primarily involving other systems may affect kidney

-- Hematological diseases
-- Purpura
-- Hemophilia
-- Other bleeding disorders
-- Metabolic diseases
-- Collagen diseases
-- Other system disorders

C.2 Renal diseases can often remain silent and effect other organs which may be involved with symptoms or examination alone may indicate the involvement of

-- Lung	-- Pulmonary edema
-- Brain	-- Coma
	-- Flapping tramors
-- Blood	-- Anaemia
	-- Polycythaemia
-- Bones	-- Rickets
	-- Osteomalacea
-- Metabolic	-- Acidosis

RENAL EXAMINATION

D.1 Kidney examination is usually not helpful in telling us the site of involvement except the site of tenderness.

Palpably enlarged kidney can however give us a list of causes.

Causes of unilateral and bilateral kidney lumps are known

INVESTIGATIONS: Need to be done to find and/ or confirm the Site of disease, its Nature and the Pathophysiological derangements occurring along with the consequential Metabolic abnormalities and also the Dysfunction present

E.1 SITE OF DISEASE

-- Urine examination

    -- Amount of proteinuria
    -- Casts
    -- Pattern of hematuria gives us the tentative site

-- Ultrasonography gives us better information about the size and structural abnormalities

    -- Stone
    -- Cortical thinning
    -- Small/large kidney

CT-Scan also supplements this information.

E.2 ETIOLOGY OF DISEASE

-- Urine examination can confirm
-- Infections
-- Metabolic Diseases
-- Renal Biopsy and Immunologic Staining

E.3 RENAL FUNCTIONS

-- Serum creatinine
-- Urea clearance or Creatinine tests
-- Differential Renal Functions can also be done
-- Hemoglobin

E.4 Measurements of swelling by serial weight record and Intake - output charting of fluids.

ENDOCRINAL HEMATOLOGICAL, METABOLIC AND PSYCHIATRIC DISEASES

Endocrinal System:

Hormones take part in different metabolic processes in the body distant to site of hormone production via the blood stream. Many hormones have interlinking effects. Few affect many target organs whereas many affect specific organs/ metabolic processes in particular organs only.

This heterogeneity does not make for a synthesized clinical approach.

Hematological Diseases

Although the hematological system-has a common site- blood; it is very heterogeneous as to the components particulate, fluids (liquids and gases- 0_2 and $C0_2$), biochemical products including glucose, minerals and hormones. And as blood circulates throughout the body and these above nutritional components interact with the various organ systems selectively and universally.

It is not a "single" organ system and not conducive to straight forward clinical approach as other systems with a dedicated work and place the human body.

Metabolic Diseases

Metabolic disorders are also too variegated to be put together in one group and approached by a dedicated clinical approaches?

Psychiatric Diseases

Psychiatric diseases likewise do not have a codified structure-function relationship so are not yet amenable to a unified clinical approach..

SUMMATION

This outline of The Common Plan applicable to medical diagnosis is important. As it is a sectional approach, small sections of the process are taken up, analyzed and the information from each is separately understood. It makes assessment easy and uniform especially for teaching the new students.

Another important part of this plan is that summation of all pointers to the Site, Etiology, Pathophysiology and Dysfunction can be done in totality (See the cover of this Book).

- The pointers to the anatomical Site (and sub-sites) can be summated from the Presenting Symptoms, A1, D1 and E1
- Those sections which give us etiological information are found in A3, A4, B2, C1, D2 and E2.
- Pathophysiological summation of information is available from A2, A5, B3, C2 and E3.
- Dysfunction assessment is also available from A2, B3, C2, E3 (as above) and E4

It is to be noted that any questions asked in the clinical scenario of any patient or in an examination can be answered from the relevant sections only, and searching for the right answers can then become a focused activity.

Note should also be made of the fact that no information regarding site can be made from the General or Systemic examination of the other systems. Pathophysiological information is not to be gained from examining the involved system.

DIFFERENT PARTS OF THE HISTORY ALONE GIVES US INFORMATION OF ALL THE FOUR PARTS OF THE DIAGNOSIS AND THAT IS ONE OF THE TWO REASONS WHY HISTORY IS SO IMPORTANT, THE OTHER BEING THAT HISTORY ALONE CAN HELP MAKE THE DIAGNOSIS IN 60-80% OF ALL CASES.

EPILOGUE

Clinical Therapeutics -- The Four Tiered Approach

The prerequisite for good treatment is the knowledge of the complete diagnosis of a patient's disease. This incorporates Nature, Site, Pathophysiology and Dysfunction. This knowledge is essential to attempt to treat the patient fully and correctly. Obviously knowledge has its shortcomings and all these parts of the complete diagnosis are not known for all human diseases. In an individual patient, all these parts may not be known.

In the clinical setting, the work up of a patient starts with the symptom analysis and the *First Tier* of treatment is based on attempting to *relieve the symptoms*. This of course is the time honored way and many systems of medicine (other than allopathic) are still in this phase of their treatment approach. The scientific base of therapy in most systems of Medicine is limited to observation.

The first tier therapy is very useful, universally applicable and by itself enough if the disease is self limiting and does not leave any structural damage (Anatomical abnormality).

The *Second Tier* of therapy is also aimed at *relieving symptoms but based on the pathophysiological concepts* so that edema is not just treated by diuretics but classified into Renal, Cardiac or Hepatic edema and the appropriate therapy is planned which may be spironolactone (and/or I.V. albumin) for Hepatic Edema; for Congestive Cardiac Failure diuretics, digoxin and enalapril may be used. These measures aimed at counteracting the pathophysiology do not, however, treat the nature of disease or the structural anomalies resulting from it. In cases where the disease is self limiting and no residual structural damage exists, this second tier of therapy is enough. This is the case in most viral infections, most trauma, some bacterial and other infections.

The allopathic system of medicine is well advanced in the understanding of pathophysiology. In fact, newer understandings have opened way for newer modalities of therapy. The discovery of Nitric Oxide and other gases which are found to have their effect, and are metabolized within seconds have opened new vistas for newer pathophysiological approaches to treatment.

However, this second tier of therapy is often of limited clinical use because many such processes are present in various sites and organs and treatment is usually not site/organ specific and side effects in other organs can be a problem, for example when beta blockers are used in heart disease they may cause bronchoconstriction in the lungs.

The *Third Tier* of therapy is based on correcting *macroscopic structural changes*. This is the surgeon's forte and is especially useful if the etiology is curable,

controllable or self limiting as in operation for post traumatic stricture of urethra; Mitral valve surgery and controlling the relapses of Rheumatic Fever or surgical correction of congenital cardiac structural defects (the cause having occurred during antenatal or natal period and therefore does not recur).

The future holds promise for correcting microscopic structural defects by using the operating microscope and even structural genetic defects by engineering.

However, this 3rd tier of therapy is not enough if the disease is not self limiting and non controllable. The classical example is Coronary Artery Bypass Surgery where narrowed arteries are bypassed by surgery but the process of Atherosclerosis which leads to the narrowing is not reversible and fresh narrowing keeps on occurring.

The *Fourth Tier* of therapy is the most difficult as it deals with curing/ controlling the *etiology* of disease. Many-a-times we diagnose this part of the disease process after the damage has already been done. Often the etiology is not known and sometimes its treatment modality is not known. The saving grace is that persistent structural damage and the mild pathophysiological changes are reversible with treatment or are self limiting. The control of disease at its outset (before irreversible structural or pathophysiological changes have occurred) requires determination, political will and the economic means, even when known.

Treatment at this tier is complicated for an individual who has a disease etiology which does not cause harm i.e. he is a carrier, treatment is not needed especially if there are no toxic effects, however, from the public health view point treatment of carriers helps in controlling spread. The logistics depends upon the ratio of clinical disease to inapparent infection and carrier rates to analyze the cost-benefit ratio for each individual disease, and also assessment of the logistics is required to make such ventures probable.

Few examples are in order, that overpopulation as a cause of many medical diseases is well known but the efforts to control it so far have not succeeded. Smoking is a hazard but the lack of political will and socio-economic conditions have hampered its control and that of the many known diseases arising from its use. The prevention of malaria is another example which succeeded initially and failed subsequently.

In this way rational therapy can only be done when a better therapeutic approach is done based on the total diagnosis of the patient as outlined in the preceding sections of this book.

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ATHEROSCLEROSIS AND REVERSE CHOLESTEROL TRANSPORT
SPLENOMEGALY AND THE ROLE OF VIRUSES
HYPERTENSION, MYXOEDEMA
MANAGING DIABETES DURING RAMZAN
CAROTID INTIMA MEDIA THICKNESS STUDIES IN ATHEROSCLEROSIS

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