BEDSIDE APPROACH TO CLINICAL CARDIOLOGY

Includes online access to complementary videos on bedside echocardiography

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CONTENTS

INTRODUCTION

The art of a physician is his/her clinical approach that revolves around his/her personal disposition, communication skills which includes the style of interaction, ability to listen and understand what the patient has to say. Obviously what is important here is doctor's sincerity and inquisitive questioning. These traits of a physician have a salutary effect on the quality of evaluation and diagnosis.

The science of medicine encompasses the development in medical engineering, advancement in tests and procedures and the therapeutic knowledge which today has become the barometer of competence and accordingly influencing factors for patient's confidence in doctors.

Consequently there has been an increasing dependency on the sophisticated investigation and procedures and less time spent by doctors at the bedside. As a doctor you should remember that in that case you are no way better than a clinical corroborator and you lose the credit of being called as a clinician. *You should still note that a patient never says no to a detail clinical interrogation and examination today.* The clinical examination is always a gold standard of medicine practice. It can never be replaced by any sort of modern machine or tool. Of course, such sophisticated investigations can strengthen or pin point your clinical diagnosis and also save time. Thus the benefit of modern science should be taken as an important substantiation for clinical practice and certainly not as a substitute.

"No greater opportunity or obligation can fall to the lot of a human being than to become a physician. In the care of the suffering he needs technical skill, scientific knowledge and human understanding. He who uses these with courage, with humility and with wisdom will provide a unique service to his fellow man and will build an enduring edifice of character within himself. The physician should ask of his destiny no more than this; he should be content with no less."

-Tinsely R. Harrison.

Many a time patients insist for an evaluation with a machine about which his or her knowledge is limited. But the clinician should not forget his basic tool of evaluation i.e., clinical examination. You may go for a bed side echo for a cardiac patient but must do a detail clinical examination before that. This brings two things to you. First, the patient's confidence and the second is your own confidence. This builds your competence slowly. Confidence you have to build first and then you earn competence. Both are complementary to each other. It is often seen that a busy clinician misses a significant murmur in his clinic which he realizes later when the patient comes back with an echo diagnosis of the same.

One more critical observation needs to be mentioned at this point that in a stable patient "no treatment should be initiated without a clinical or final diagnosis". Do not start writing the medicines just after listening to patient's ailments. This spoils the clinician's image and confidence. Whatever may be the situation your prescription should start with a diagnosis. It could be a provisional or working diagnosis.

This book is designed to provide you the proper guidelines as to (1) How the overall ideal patient doctor environment is to be developed, (2) A detail clinical history evaluation and physical examination to be done and (3) How to interpret the clinical database in terms of disordered function and potential etio-pathogenesis and establish a diagnosis of physical, mental, social or a combination of these disorders.

Approach of a Clinician

Right Ways

The translation of medical knowledge into good patient centric decision is a key goal of clinical reasoning and is the hallmark of an expert and successful clinician. The physician need to keep the under mentioned issues in mind to do proper justice to his role besides earning the image and reputation of a good physician.

Privacy of interaction

It should be ensured that interaction, interrogation and examination of a patient is kept private. Not only the patient has a right even the medical ethics demands so. It is therefore necessary that any examination or interrogation is with the consent or with permission of valid authority.

Clean record keeping

After performing the examination and consultation, proper recording should be done in the patient's case notes. These case notes are confidential and should be kept securely. They also constitute a legal document that could be used in a court of law. Keeping accurate and up-to-date notes is an essential part of good patient care.

Doctor's personal disposition

As rightly quoted by Tinselly R. Harrison, doctors still have a dignity in our society. If anybody spoiling it today it is only doctors themselves. First of all, your presentation has to be clean, sober, smart, decent and approachable. The way you dress is important in establishing a successful patient-doctor relationship. Your dress style and demeanor should never make your patient or your colleagues uncomfortable or distract them. Male doctors need to have a clean shave preferably with an acceptable hair cut. They should not stink due to sweats, shocks, smoking etc. Clean white coat with a tie obviously gives you a look of professionalism. Female doctors should tie back long hair and keep jewelry simple and limited so as not to impede hand washing or examination. Clothing of course matters a lot in female doctors. It should not be a factor of distractions while communicating with the patient or their attendants. Doctors or medical students should look after themselves properly and maintain their own health. They should protect themselves by proper diet, exercise, immunizations etc. Remember doctors are treated as next to God in the society, and hence they should not spoil the image by getting involved by developing or pursuing a sexual or improper emotional relationship with a patient or someone close to them. They should never express their personal beliefs, including political, religious or moral ones, to the patients in ways that cause them distress.

Communication skills

Communication skill is very important. Doctors who communicate well are able to identify a patient's problem more rapidly and accurately, while their patients benefit from a better understanding of their condition and its management. Good communication skills are the most important part of being a good doctor. These should always include: Maintaining good eye contact, active listening, ability to discuss difficult issues and going at a pace that is comfortable for the patient. If language is a barrier, try to

learn at least working language step wise, for examples good morning, how are you? What are your main complaints? Do you have any chest pain anytime? Do you have shortness of breath? etc.

The tone tenor and temperament of your voice is equally important in making a fruitful and purposeful communication.

Get to know how patient evaluated himself. It is equally important that a doctor not only creates positivity in patients he should also explore to know how patient evaluates his problems and ailments. For a doctor then it will be easy to share something which may not be a pleasant diagnosis.

Care and Courtesy

A patient while being interrogated or examined needs to be respected appropriately. Doctors need to avoid calling elderly patients by first name. They need to be addressed with due sensitivity. The doctors must as far as possible display a smiling and pleasant disposition so that patient can confidently look at them as friends and savior. Children need to be approached with care and sweet words.

Presence of a female attendant

It is always advisable and desirable that if a female patient is examined by a male doctor it should always be in presence of a female attendant. This is required as abundant precaution to avoid untoward incidences.

The issues stated however are not fully exhaustive and more can be added.

CHAPTER 1

SYMPTOM ANALYSIS FOR EVALUATION OF CARDIOVASCULAR SYSTEM

Diseases of Cardiovascular system can present with four cardinal symptoms of *angina, dyspnea, palpitation* and *syncope* may or may not be associated with pedal swelling, fatigability, jaundice, ascites, vomiting, cough and hemoptysis. The presentation of clinical history should be described in chronological manner (as described below). Clinical history narration should start with *Chief Complaints*, which should be enumerated with salient symptoms along with duration in a chronological manner. It should be followed by *History of Present Illness.* Here, one has to expand the chief complaints in details with relevant positive and negative history. For example, Mr. John complaints of Dyspnea on exertion for last 6 months (*chief complaints*). He has shortness of breath while walking a long distance for more than 1 km which he used to do without any difficulty six months before (*History of present illness*). At times he feels chest pain during his shortness of breath (*relevant positive history*). He is not a known case of Bronchial asthma (*relevant negative history*). So also Angina pectoris can usually be differentiated from the pain associated with pulmonary embolism, Pericarditis, aortic dissection, esophageal reflux or Costochondritis. Cough, hemoptysis and cyanosis if present may also provide additional information. One should also be familiar with clinical presentation mode of stroke (cerebro vascular accident) and TIA (Transient Ischemic Attacks) along with vascular diseases manifesting with Limb Claudication and skin discoloration.

Extra note of a life style of a patient has tremendous value to help out you to come close to a clinical diagnosis. The examples are,

Mr. Rajesh, 45 year old, wakes up early in the morning, goes for regular exercise, eats regular fruits and green vegetables, keeps monitoring of his body weight and does regular medical checkups. On the other side Mr. Deepak who is of same age and profession but has a heavy body, BMI around 30. He does not go for regular exercise and has no physical activity. He is a late riser, smokes regularly, does not like to take fruits and green vegetables and never goes for medical checkup. If both of them complaint of chest discomfort to you for evaluation, it becomes much easier to go with a clinical impression. Risk Factors do have always a basis for formulating a clinical diagnosis. Old medical records of parameters like blood pressure, pulse rate, blood sugar and ECGs of the patient always help in medical emergency situation to compare the present values with the baseline.

In this discussion, it is mandatory to understand the individual clinical parameters of cardio vascular diseases in details.

Assessment of functional class

Cardiac history taking is an art which incorporates particular functional class of the patient. This helps to assess the present symptom status, future follow up and the effect of the treatment on its original status. There are various methodologies to assess the functional status of the symptoms of the presenting patient. They are New York Heart Association (NYHA) functional class, Canadian Cardiovascular Society (CCS), Specific Activity Scale (SAS), Six-minute walks etc. Most popularly we use NYHA class in our case presentations (see Table no. 1.1).

TABLE 1.1: New York Heart Association (NYHA) functional class

Functional Class	Functional capacity	Objective assessment
Class I	Patients with cardiac disease but without resulting limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea or anginal pain.	No objective evidence of cardiovascular disease
Class II	Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnea or anginal pain.	Objective evidence of minimal cardiovascular disease

Class III	Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity results in fatigue, palpitation, dyspnea or anginal pain.	Objective evidence of moderately severe cardiovascular disease
Class IV	Patients with cardiac disease resulting in inability to carry on any physical activity without discomfort. Symptoms of heart failure or the angina syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.	Objective evidence of severe cardiovascular disease

Angina

It is usually described as a heavy chest pressure or squeezing, a burning feeling or a difficulty in breathing that typically builds over several minutes (2 to 10 minutes) and radiates towards left shoulder and arm, epigastrium, back and neck (see Figure 1.1).

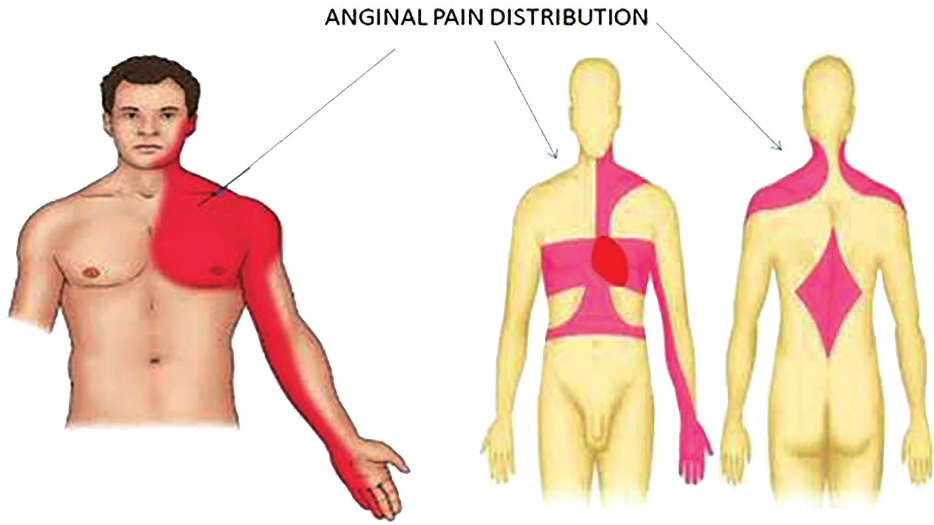

Figure 1.1: Various sites of typical angina of myocardial ischemia are cited here with red color.

Mechanism of angina

The mechanisms of cardiac pain and the neural pathways involved are poorly understood. It is presumed that angina pectoris results from ischemic episodes that excite chemo-sensitive and mechanoreceptive

receptors in the heart. Stimulation of these receptors results in the release of adenosine, bradykinin, and other substances that excite the sensory ends of the sympathetic and vagal afferent fibers. The afferent fibers traverse the nerves that connect to the upper five thoracic sympathetic ganglia and upper five distal thoracic roots of the spinal cord. Impulses are transmitted by the spinal cord to the thalamus and hence to the neocortex. Clinically a chest pain may be difficult to diagnose most of the time because of its diverse etiologies and individual variation in expressions. Hence, one brief chart of differential diagnosis in the finger tips are necessary to be kept ready. Also one should learn how to differentiate *atypical chest pain* from a typically described Angina (see Table 1.2).

TABLE 1.2: Differential Diagnosis of chest pain

System	***Syndrome***	***Clinical description***	***Key distinguishing features***
Cardiac	Angina	Retro-sternal heavy chest pressure or squeezing, a burning feeling or a difficulty in breathing and radiates towards left shoulder and arm, epigastrium, back, and neck	Precipitated by exercise, cold weather or emotional stress, duration of 2 to 10 min
	Rest or Unstable angina	Same as angina but may be more severe	Typically <20 min, lower tolerance for exertion, crescendo pattern
	Acute Myocardial Infarction	Same as angina but may be more severe	Sudden onset, usually lasting >30 min, often associated with sweating, shortness of breath, weakness, nausea and vomiting
	Pericarditis	Sharp, Pleuritic pain aggravated by change in position, highly variable duration	Pericardial friction rub

Vascular	Aortic Dissection	Excruciating, ripping pain of sudden onset in either front or back of chest	Usually happens in a setting of Hypertension or connective tissue disorder like Marfan Syndrome
	Pulmonary embolism	Sudden onset of dyspnea and pain, mostly Pleuritic type	Dyspnea, Tachypnoea, Tachycardia, right sided heart failure
	Pulmonary Hypertension	Substernal chest pressure, exacerbated by exertion	Pain associated with dyspnea & sign of Pulmonary Hypertension
Pulmonary	Pleuritis & Pneumonia	Pleuritic pain over the involved area	Pain Pleuritic and lateral to midline associated with dyspnea
	Tracheobronchitis	Burning discomfort in midline	Midline location with cough
	Spontaneous Pneumothorax	Sudden onset of unilateral Pleuritic chest pain with dyspnea	Abrupt onset of dyspnea and pain
Gastro-intestinal	Esophageal Reflux	Burning substernal & epigastric discomfort, 10–60 min duration	Aggravated by large meal and postprandial recumbency, relieved by antacid
	Peptic ulcer	Prolong epigastric or substernal burning	Relieved by antacid or food
	Gall bladder disorder	Prolong epigastric or right upper quadrant pain	Usually after 30 minutes of a fatty diet
	Pancreatitis	Prolonged, intense epigastric and radiates to back through left side	Risk factors like Alcohol or Hypertriglyceridemia
Musculo skeletal	Costochondritis	Sudden onset of intense fleeting pain	Chest wall tenderness present
	Cervical disc disease	Sudden onset of fleeting pain	Reproduced by neck movement
	Trauma or strain	Constant pain	Tenderness on palpation and movements

System	*Syndrome*	*Clinical description*	*Key distinguishing features*
Infectious	Herpes Zoster	Prolong burning pain in a dermatomal distribution	Vesicular rash in dermatomal distribution
Psychological	Panic disorder	Chest tightness or aching, often accompanied by dyspnea and lasting >30 min, unrelated to exertion or movement	May have other evidences of emotional disturbances

The American College of Cardiology (ACC) and American Heart Association (AHA) has laid guidelines that the following pain descriptions are uncharacteristic of myocardial ischemia:

- *Pleuritic pain (i.e., sharp or knifelike pain) brought by respiratory movements or coughing.*
- *Primary or sole location of discomfort in the middle or lower abdominal region.*
- *Pain that may be localized by tip of one finger, particularly over left chest.*
- *Pain reproduced with movements or palpation of chest wall or arms.*
- *Constant pain persists for many hours.*
- *Very brief episodes of pain that exists for few seconds or less.*
- *Pain that radiates to lower limbs.*

Causes of coronary artery disease

Most common cause of angina is Coronary Artery Disease. Apart from the commonest pathology atherosclerosis this artery may have many other rare systemic diseases. They are: Arteritis, Vasculitis in a transplanted heart, Kawasaki disease causing angina or myocardial infarction in children, Systemic Lupus Erythematosus, Polyarteritis Nodosa, Rheumatoid Arthritis, Ankylosing Spondylitis, Syphilis, Infective Endocarditis, Left Atrial/Ventricular thrombus, Left Atrial/Ventricular tumor, Prosthetic valve thrombus, Takayasu's disease, Embolism and fat or air embolism.

Bruising and damage to the artery where the catheter is inserted that may lead to pseudo aneurysm. There may be coronary mural thickening due to Amyloidosis, radiation therapy affecting the ostio-proximal parts, Hurler's disease, Pseudoxanthoma elasticum, Aortic dissection coronary spasm, Prinzmetal angina and congenital coronary artery disease like myocardial bridge and anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA).

While exploring bed side clinical cardiology one should simultaneously learn detail about behavior of *angina* in clinical presentation in an acute set up. By now we have discussed how to filter out the real or typical angina from the diverse presentations of *atypical angina*. We need to know how one patient admitted with acute chest pain, should be promptly and correctly evaluated since that will decide the emergency course of treatment for that moment to save life.

Acute coronary syndrome

Angina persisting for more than 15 minutes with a crescendo pattern with a tendency to intensify on mild exertion and may or may not be associated with sweating, dyspnea, nausea, vomiting, palpitation or giddiness is defined as Acute Coronary Syndrome (ACS). This usually occurs due to rupture or erosion of an atheromatous plaque. ACS may be divided into three categories according to accompanying bed side parameters to facilitate to intervene for life saving methods. First and most easily available tool is ECG. It may show ST elevation in contiguous leads with or without ST depressions in opposite wall leads (STEMI), only ST depressions, T wave inversion or even a normal ECG (Non STEMI or Unstable Angina). Next commonly used tool is a myocardial enzyme level in blood sample. It could be Troponin I, Troponin T or CK MB. If this enzyme is positive in such a case without presence of ST elevation, it could be called Non STEMI. When ACS case has ST depression or T inversion but cardiac enzyme is negative, it is UNSTABLE ANGINA. But unfortunately cardiac enzymes which are conventionally used today are detectable in most cases after 4 to 5 hours of the index angina. This is a sign of myocardial necrosis. The outcome (prognosis) after this point of time is not likely to be favorable.

Practically once a case of ACS is received and the first (presenting) ECG does not clearly show ST elevation, one has to administer all primary medications and repeat ECG after 30 minutes to observe any mild progression in ST elevation to rule in a diagnosis of STEMI. To substantiate this diagnosis one bed side Echocardiography should be done to look for a relevant Regional Wall Motion Abnormality (RWMA). Since "Time is Muscle" one cannot afford to wait till the cardiac enzymes are elevated. Once a diagnosis of STEMI is made he/she should be thrombolyzed after ruling out any absolute contra-indications. For NSTEMI or Unstable Angina (UA) Heparin should be started. If facility allows such patient has to be taken up for Coronary Angiogram (CAG) within 24 hours to delineate the coronary anatomy and if necessary an interventional procedure, angioplasty may be done to treat an obstructed coronary artery. To determine the risk of subsequent events in a case of ACS there are some clinical risk analysis scoring system in practice namely Thrombolysis In Myocardial Infarction (TIMI) risk score and Global Registry of Acute Coronary Events (GRACE) risk scoring system (Table 1.3 and 1.4).

TABLE 1.3: Grace Risk Score

Variable	Points
Age (years)	
<30	0
30–39	8
40–49	25
50–59	41
60–69	58
70–79	75
80–89	91
≥90	100
Heart rate (beats/minute)	
<50	0
50–69	3
70–89	9
90–109	15
110–149	24
150–199	38
≥200	46

Systolic blood pressure (mmHg)	
<80	58
80–99	53
100–119	43
120–139	34
140–159	24
160–199	10
≥200	0
Initial serum creatinine (mg/dL)	
0.0–0.39	1
0.4–0.79	4
0.8–1.19	7
1.2–1.59	10
1.6–1.99	13
0.2–3.99	21
≥4	28
Killip class	
I	0
II	20
III	39
IV	59
Cardiac arrest at admission	39
Elevated cardiac markers	14
ST segment deviation	28

TABLE 1.4: Interpretation of Grace Risk Score for In-Hospital Mortality

Score	Probability of in-hospital mortality (%)
≤60	≤0.2%
70	0.3%
80	0.4%
90	0.6%
100	0.8%
110	1.1%
120	1.6%

Score	Probability of in-hospital mortality (%)
130	2.1%
140	2.9%
150	3.9%
160	5.4%
170	7.3%
180	9.8%
190	13%
200	18%
210	23%
220	29%
230	36%
240	44%
≥250	≥52%

Thrombolysis in myocardial infarction (TIMI) risk score

The Thrombolysis in Myocardial Infarction (TIMI) Score is used to determine the likelihood of ischemic events or mortality in patients with Unstable Angina or non ST-segment elevation myocardial infarction (NSTEMI) (Table 1.5).

TABLE 1.5: TIMI Score

Score	*Risk of Death/MI/Urgent Revascularization by Day 14*
0–1	5%
2	8%
3	13%
4	20%
5	26%
6–7	41%

Each of the following criteria constitutes one point for TIMI scoring [Total 7 points].

1. Age ≥65 years
2. Three or more risk factors for coronary artery disease (CAD) (family history of CAD, hypertension, hypercholesterolemia, diabetes mellitus, tobacco use)
3. Known CAD (Stenosis >50%)
4. Aspirin use in the past 7 days
5. Severe angina (≥2 episodes in 24 hours)
6. ST deviation ≥0.5 mm
7. Elevated cardiac marker level.

This is how a case of ACS is dealt with. This part of book is not dealing with detail management of angina. This is attempted to make you aware of one extreme form of ANGINA which is very simply evaluated in the course of history taking. **Acute coronary syndrome is discussed later in ECG chapter.**

Dyspnea (Breathlessness) and fatigue

Dyspnea is also described as Breathlessness or Shortness of breath. It is defined as abnormally uncomfortable awareness about one's own breathing. This symptom is common for many pathological processes or diseases. Hence meticulous history evaluation needed to pin point or come close to a system(s) at fault. This is the cardinal symptom of cardiovascular and Respiratory system. Sometime an unaccustomed exercise can cause dyspnea in a normal healthy person. Basically the following diseases can cause dyspnea in a patient.

1. Pulmonary: Chronic Obstructive Pulmonary Disease (COPD), Bronchial asthma, Pneumonia, Pleural effusion, Pneumothorax, Pulmonary edema, Restrictive Lung diseases and Pulmonary embolism etc.
2. Cardio-Vascular: Valve diseases, Coronary Heart Diseases, Cardiomyopathy, Pulmonary Artery Hypertension and Systemic Arterial Hypertension.
3. Anemia: Severe anemia of any cause.

4. Metabolic derangements: Uremia and Acidosis etc.
5. Obesity: Gradually becoming obese persons usually do not complaint of dyspnea.

Persons becoming obese in short period commonly become symptomatic (Table 1.6).

TABLE 1.6: Differential Diagnosis of Dyspnea

Acute	Acute on Chronic	Chronic
Asthma	Infective exacerbation of COPD	COPD
Myocardial Infarction	Decompensated Chronic Heart Failure	Cardiac Failure
Pulmonary Embolism (PE)	PE complicating in CCF	Anemia
Cardiogenic Pulm Edema	Pneumothorax complicating COPD or Asthma	Pulmonary Hypertension
Pneumonia	Atrial Fibrillation/Flutter complicating COPD or Heart Failure	Parenchymal lung disease

Special types of Dyspnea

Orthopnea: Dyspnea on lying down supine is called Orthopnea, it is mostly seen in left heart failure situations.

Paroxysmal Nocturnal Dyspnea (PND): A classical symptom of heart failure, reported by the patient as sudden awakening with severe breathlessness after sleeping for 2 to 3 hours at night. This is mostly seen in Mitral Stenosis and other valve diseases, Ischemic Heart Disease and Cardiomyopathy.

Platypnea: Upright posture causes dyspnea usually seen in COPD, Hepatopulmonary Syndrome and Cyanotic Congenital Heart Diseases.

Trepopnea: By lateral decubitus posture the affected part of lung become nonfunctional and patient becomes dyspneic. Left Atrial Myxoma may obstruct flow by acquiring a lateral decubitus posture. Patients of Right sided pleural effusion in Decompensated heart failure may also experience Trepopnea.

Clinically Dyspnea should be assessed in the following factors:

1. Mode of Onset
2. Duration
3. Progress
4. Severity
5. Functional Class
6. Special character
7. Relieving factor
8. Associated symptoms

Clinical differentiation between cardiac and pulmonary dyspnea

In both case there is some quantity of previous ailments. Sometime it becomes easy to differentiate and sometime it is very difficult. From pulmonary etiology, dyspnea tends to develop gradually. But at times it may come suddenly due to exacerbation factors like infection of lower respiratory tract, pneumothorax or exacerbation of bronchial asthma due to sudden exposure to its allergens like cold, dust, paint etc. Patients of COPD may also get awaken due to cough and dyspnea. Here patient may bring out large amount of sputum and get relief. Cardiac patient presenting with dyspnea has a specific scale, like while exerting at a particular speed, gradient or quantity. Cessation of such activities gives relief. But during sudden development of dyspnea in cardiac disease patient there is usually a background such as history of myocardial infarction, valvular heart disease, sudden acceleration of hypertension, Cardiomyopathy with heart failure etc. In such acute cases patient also may bring out pink frothy sputum due to pulmonary edema. When both (cardiac and pulmonary) are present in one patient, it becomes nightmare for the clinician to differentiate.

Paroxysmal nocturnal dyspnea (PND)

Paroxysmal Nocturnal Dyspnea occurs due to development of pulmonary interstitial edema and typically occurs 2 to 4 hours after the onset of sleep. Patient usually stands up or sits at the edge of the bed till the symptoms resolve which usually takes 10 to 15 minutes. This episode so much frightening that patients usually adopt a propped up

bed to avoid such situations. A patient with PND is classified as NYHA class 3. A history of PND or orthopnea is only present in 20% of patients with heart failure and its absence does not rule out the diagnosis of heart failure. Commonest causes of PND are Mitral Stenosis, other Valvular heart diseases, Ischemic Heart Disease and Dilated Cardiomyopathy. Non-cardiac causes which mimic PND are Nocturnal Asthma, COPD, Pulmonary embolism, Obstructive Sleep Apnea, Anxiety and Hyperventilation.

Pathophysiology of PND

1. On prolong lying down for 2 to 3 hours (during sleeping) due to nullified gravity more venous return reaches right side of heart. Diaphragm is also mildly lax due to supine position.
2. The failing left ventricle and diseased mitral valve are suddenly unable to handle the output of a more normally functioning right ventricle resulting in pulmonary congestion and hence interstitial edema.
3. Decreased responsiveness of brain (respiratory center) during sleep leads to partial respiratory depression and decreased adrenergic drive of myocardium (no compensatory tachycardia).

It is important to note that though it is called nocturnal event it may occur in the day time also if the patients sleeps during daytime for the same duration.

Fatigue

It is very often complained by the patients that they have severe weakness. In fact most of the time it is wrongly expressed or misinterpreted. Due to severe exertional dyspnea patient may express it as weakness since there is incapacitation to perform at a regular norm. Fatigue is the manifestation of low cardiac output. This results from inadequate oxygen delivery to tissues due to low cardiac output. Sometime patients of heart failure being on large doses of diuretics may develop dehydration, hyponatremia or hypokalemia leading to severe weakness. Here history of medication to be obtained if the patient passes a huge quantity of urine or visits urinals frequently after taking a particular medicine.

Patients with Pulmonary hypertension, pulmonary thrombo-embolism and decreased pulmonary blood flow conditions like in Cyanotic congenital heart disease may also often complaints of fatigue.

Syncope

It is a transient episode of loss of consciousness (LOC) due to cerebral hypoperfusion. Its causes may be pathophysiologically different like (1) Neurally mediated (2) Orthostatic hypotension (3) Cardiac causes (4) Cerebrovascular and (5) Psychogenic.

How to interrogate a case of syncope

1. Was the LOC complete (In incomplete or presyncope patient may lose postural tone and tend to fall due to blackout, but manages to protect from a fall on the ground)?
2. Was LOC with rapid onset (occurs in cardiac arrhythmia) and short duration?
3. Was recovery spontaneous, complete and without any sequelae?
4. Was postural tone lost? (in epilepsy usually tone is increased).

If the answers to the above questions are *positive*, the episode was most likely to be a cardiac syncope (Table 1.7).

TABLE 1.7: Differentiation between seizures and syncope

	Seizure	***Syncope***
Warning	>50% have some aura	Lightheaded, blackout or blurring may be there
Onset	Sudden, any position	Usually occurs in sitting or standing, avoidable by changing the posture sometime
Features	Eyes open, rigidity, falls backwards, convulsions present	Eyes closed, falls forwards
Recovery	Confused, headache, sleepy, focal deficit	Pale, washed out, sweating, cold
Other features	Tongue bite, loss of sphincter control	Rarely loss of bladder control

History suggestive of cardiac syncope

History of syncopal episode

- ➢ Occurs during exertion
- ➢ Occurs when supine suddenly
- ➢ Associated with palpitation
- ➢ Associated with chest pain

Past medical history

- ➢ Known structural heart disease.
- ➢ Previous myocardial infarction.
- ➢ History suggestive of heart failure.
- ➢ History of valve disease.

Family history

- ➢ Family history of sudden death.

Common causes of cardiac syncope and presyncope

Reduced flow

Hypertrophic Cardiomyopathy (HCM), Aortic Stenosis (AS), Mitral Stenosis (MS), Pulmonary Stenosis (PS), LA Myxoma, Cardiac tamponade.

Vascular disease

Pulmonary emboli, Pulmonary hypertension, Acute Myocardial Infarction, Air embolism, Subclavian steal syndrome.

Tachy-arrhythmias

Supra ventricular tachycardia, Ventricular tachycardia, Ventricular fibrillation, Atrial fibrillation with rapid ventricular response, Wolff-Parkinson-White (WPW) syndrome, Brugada syndrome, Prolong QT syndrome (congenital, acquired or drugs).

Brady-arrhythmias

Atrio-Ventricular Block, Sick Sinus Syndrome, Pacemaker malfunction.

Palpitation

It is defined as abnormal, uncomfortable awareness of one's own heart beating. History of a palpitation should be carefully evaluated about the approximate rate, duration of symptoms, regularity of the rhythm and suddenness of the onset and offset. Also it is noteworthy to evaluate the triggering factors like exercise, alcohol, caffeine, drug (like beta agonists) etc. It may be useful to ask the patient to describe it by taping his/her fingers on the table to describe their heart beat during palpitations. Many a times patients wrongly present palpitation as shortness of breath to a house physician. One has to be very careful about it. Because if the attending clinician is not careful and he/she treats this particular patient of palpitation as a case of Shortness Of Breath and gives him/her an injection of bronchodilator the patient's condition will certainly aggravate and even it may lead to a critical situation. So leading question is always better in such cases to ask about the rapidity of the patient's heart rate, whether it is beating very fast to discomfort him/her.

Palpitation may be reported due to:

Sinus Tachycardia-gradual onset of rapid heart rate usually associated with Panic attacks, anxiety, beta-agonist medications in Asthmatic patients. *Premature or Ectopic beats*-Patients may describe it as "missed beats" or "forceful beats". This in fact relates to the pause that follows the ectopic beat which does prolong diastolic filling and then a forceful contraction occurs giving a thrust to the chest. Benign ectopic is considered when (a) no family history of sudden death (b) no other cardiovascular symptoms (c) their occurrence more during rest and disappearance during exercise and (d) a normal clinical cardiovascular examination and abnormal ECG. A malignant ectopic is to be considered if multifocal ventricular ectopic occurs more than 20,000 in 24 hours (as evaluated in 24 hours Holter monitoring). *Atrial Fibrillation (AF)*-This is the commonest arrhythmia. It can present silently, with intermittent palpitation or at time severe persistent palpitation depending on its response by the ventricle. This arrhythmia has to be picked up by tactful evaluation of history, like whether the patient can demonstrate it by tapping fingers irregularly. *Paroxysmal Supra Ventricular Tachycardia (PSVT)*-This arrhythmia has a sudden onset without any premonitory symptoms. Usually occurs in an apparently healthy person mostly in a structurally normal heart. It can persist for from minutes to hours. At times it resolves automatically or on taking rest or

even sometime warrants hospitalization for intervention. After this episode patient feels fatigue, dehydrated or even may pass more urine. This occurs due to release of Atrial natriuretic peptide consequent to Atrial wall stretch. Electro Physiology Study (EPS) in some cases may reveal a concealed tract as in case of Pre-excitation syndrome. At times congenital heart diseases like Atrial Septal Defect (ASD) may make its first presentation in the early or late adulthood. *Ventricular Tachycardia (VT)*-This is the most fatal arrhythmia. It can cause palpitation with severe giddiness or syncope due to reduced cardiac output. This is usually preceded by chest discomfort since it is one of the commonest complications of Acute Myocardial Infarction (AMI). This can also be a presentation of Hypertrophic Obstructive Cardiomyopathy (HOCM). This can also be observed in certain familial rhythm disorders like Brugada syndrome, Arrhythmogenic Right Ventricular Dysplasia (ARVD) or Right Ventricular Outlet Tract Ventricular Tachycardia (RVOT VT) etc. Such patients need immediate evaluation to find out the cause and plan for treatment.

Patients with a regurgitant valve lesion often presents with palpitation which is usually due to high volume causing forceful beats. Sometime patients of mitral valve prolapse may present with palpitation.

Other associated symptoms

Edema (Pedal edema or generalized swelling)

Edema is a sign of congestion due to heart failure. It is initially in the lungs causing pulmonary edema presenting with dyspnea, PND etc. But, in later stage when pulmonary congestion shifts to venous side due to subsequent right heart failure it appears on the dependant part of the body. In ambulatory patients it is found in the legs and in bedridden patients it is present in the sacral area. In the children it is also found in peri-orbital area due to low tissue pressure around eyes. Once the pedal edema appears the patient has approximately five kilo grams of gain of body weight. But pulmonary edema or congestion may occur without gain of weight in certain situations like Mitral Stenosis and Acute Left Ventricular Failure due to various causes.

Cough

A nonproductive or dry cough may herald a left heart failure in chronic form. Patient complaints of cough more on lying down than on an erect posture. This is usually associated with dyspnea. Such patients may bring

out pink-frothy sputum some time if they suffer a pulmonary edema or PND. History of hypertension, rheumatic heart disease, ischemic heart disease or Cardiomyopathy is to be evaluated in such cases. Certain drugs particularly ACE Inhibitor causes cough by inhibiting the metabolism of Bradykinin. Other drugs like Beta blockers and Aspirin at times can cause bronchospasm and then cough. Sometime hyperacidity with incompetent gastro-esophageal valve may cause postural cough when lying flat by inducing pharyngo-laryngeal reflex. *(Sometime asthmatics, chronic smokers and patients with pulmonary eosinophilia may also have cough of a similar character).*

Hemoptysis

History of hemoptysis should always be well evaluated many a times since the patients often confuse hematemesis (vomiting of blood) with hemoptysis (coughing out blood). A traditional concept in India exists that whenever there is history of hemoptysis, first rule out pulmonary tuberculosis. This is still acceptable in today's medical practice. However when there is history of small quantity of blood coming out with cough at times mixed with sputum without a history of fever, it could be a cardiac cause. This happens in the initial stage of pulmonary venous hypertension most commonly in mitral Stenosis.

Vomiting

This often happens in acute coronary syndrome particularly in Acute Inferior Wall M.I. (STEMI) due to over activity of Parasympathetic nervous system. Causes of vomiting in acute coronary syndrome may also be due to drug induced like Aspirin and Morphine or it could be due to a fatal complication like cardiac rupture.

Sweating

This usually happens as an excessive Sympathetic activity in response to pain. Sometimes it can accompany vomiting, bradycardia and hypotension; this is over activity of Parasympathetic system. Many a times sweating may be the only presentation of acute coronary syndrome (painless Myocardial Infarction or Silent M.I.). In case of accelerated hypertension patient may present with severe sweating with or without dyspnea. Since cardiovascular disorder is usually accompanied by co-morbidities like Diabetes mellitus, sweating may occur in such patients due to severe hypoglycemia.

Fever

Fever in acute set up of cardiovascular disease could be due to acute myocardial infarction raising body temperature from 101 to 102 degree F within 24 to 48 hours (due to necrosis of myocardium with mild surge of Leukocytosis), pulmonary embolism (due to immune reaction to pulmonary embolus absorption). Fever can also occur in chronic set up of cardiovascular disease like Infective Endocarditis, Rheumatic Fever, Pericarditis, Left Atrial Myxoma and other immunogenic Arteritis etc.

Asymptomatic patient

Sometime patient brought you denies any complaint. Most of such patients have adapted a limited physical activity to avoid any symptoms. They are comfortable with their minimized exertion. If they are asked to do a standard exercise suitable for his/her age they deny to do so which finally reveals the physical condition. It is observed that about 40% people in common society of India carry some heart ailments without any CVS symptom. They are detected by cardiovascular evaluation to carry at least one of the high risk factors like hypertension, diabetes, dyslipidemia or an abnormal ECG.

History Taking

The ailments of the heart can be divided in the following categories for better understanding:

(1) Congenital heart disease
(2) Valvular heart disease
(3) Coronary heart disease
(4) Myocardial heart disease
(5) Pericardial heart disease and
(6) Miscellaneous diseases affecting heart.

History of illness of a patient is a sealed treasury for the physicians. One has to open it systematically, carefully and methodically to derive its full benefit to arrive at a close diagnosis. This book is targeted for undergraduates, postgraduates as well as practicing clinicians. Hence, one has to ascertain that in a cardiovascular evaluation you *should be able to draw maximum three close diagnoses after completion of history taking.*

After you complete the *physical examination* you should be able to *come down to two* close diagnoses. And the *relevant cardiac investigation(s) should pick up the final diagnosis from one of these two diagnoses*. So the questions you are going to ask the patient with the available time should be relevant and specific.

Before you start the history taking it is mandatory to read the symptomatology of cardiovascular system. Because, eyes cannot see what the mind does not know. The major symptoms associated with cardiac disease include *chest discomfort (Angina), dyspnea, fatigue, edema, palpitations, and syncope*. Cough, hemoptysis, and cyanosis are additional examples. Claudication, limb pain, edema, and skin discoloration usually indicate a vascular disorder. Many cardiovascular diseases may present with non-cardiovascular symptoms like weakness of one side or a part of body (stroke due to cerebral embolism), sudden limb pain due to thrombo-embolism to a limb artery, sudden onset unilateral blindness due to central artery of retina thrombo-embolism, Jaundice (chronic heart failure leading to hepatic congestion) and oliguria due to hypovolemic status (heart failure). Once the patient says he or she has shortness of breath, the clinician should be able to understand what is happening. This is called *"cooking of hypothesis".* For example, here the patient with a history of shortness of breath might have had a heart disease, a lungs disease, upper respiratory tract obstruction disease or anemia or a patient with morbid obesity. Many factors will be considered after physical examination. But, as a discipline of history one has to start asking some relevant questions or leading questions. This is because the patient does not know how to subdivide or break up his or her symptoms. The patient may be asked whether this shortness of breath comes at rest or it comes during physical exertion only. Has he/she been told by a doctor as an asthmatic in the past? If shortness of breath comes with exertion, then at what extent of exertion it does come? Is it on heavy exertions like climbing up stairs beyond third floor or simply walking few steps even inside the room? After the exact type of shortness of breath has been found out, the duration of this illness to be ascertained. Whether it is progressive in nature i.e., it was coming after climbing up 3 stairs 6 month back and now for last 15 days it comes even after one stair. This predicts that the pathology of the hypothesized disease is progressive in nature. It could be a valve lesion (say mitral Stenosis), or a coronary artery disease with progressive myocardial ischemia manifesting as angina equivalent, or even a failing

heart. Similarly when a patient complaints of chest discomfort or angina, its duration, mode of appearance and relief to be asked. The approach to the patient with known or suspected cardiovascular disease begins with a directed history and targeted physical examination, the scope of which depends on the clinical context at the time of presentation. There is a decline in bedside skills today which has spurred an increase in the utilization of noninvasive imaging studies to establish the presence and severity of cardiovascular disease. In most of the time these unnecessary tests can be avoided or at least reduced. No doubt an integrated approach may save time but in a country like India affordability is also an important concern.

History of a patient as you know needs to be evaluated in a stepped manner like history of present illness, history of past illness, family history, occupational history and some time treatment history. History as we know should be informative, relevant and precise. Although we all know the steps of clinical history evaluation, following few lines will help in recapitulating the method.

Before going to history in detail it is important to review the symptomatology of cardiovascular disease (described above). Once the symptomatology is clearly understood it becomes easier to analyze the history to draw the outlines of provisional diagnosis.

Chief complaints

One has to start with the most important and persisting ailment which has a longest duration. Subsequently the accompanying symptoms those appear later but persist either continuously or intermittently. This should be always in a chronological manner. For example, chest discomfort on exertion since one month and intermittent exertional breathlessness for last 15 days.

History of present illness

In this paragraph one has to elaborate the chief complaints with relevancy. For example, the patient was apparently alright one month back. He first felt *chest discomfort (angina)* while going to his office on a bicycle on an up gradient road. He used to get relief after getting down from bicycle and taking a break. But gradually he started getting the discomfort even while riding it on a plain. Now he gets it even on walking up the gradient. For last 15 days the patient has been experiencing

shortness of breath (breathlessness) while walking on an up gradient. This elaboration appears to be short, but it is relevant and informative. This tells that this patient has a heart disease which is causing myocardial ischemia on a higher demand i.e., physical exertion increasing heart rate and myocardial workload causing a stress to myocardium and asking for more oxygen and nutrition. But the existing coronary supply is unable to meet the extra demand of the myocardium (which is usually met by a normal coronary system). Hence there could be two possibilities, either the myocardium mass is more like ventricular hypertrophy or there is coronary artery disease causing obstruction to flow in high demanding situation.

The second symptom of *breathlessness (dyspnea)* appearing after few days probably tells about the transient pump failure of myocardium which might have been caused by the myocardial dysfunction due to less coronary blood supply or due to decreased left ventricular compliance particularly in case of wall hypertrophy. This way one has to postulate the clinical hypothesis in mind before the physical examination is taken up.

Another important symptom related to cardiovascular system is *palpitation.* Very often this is misinterpreted by the patient and then wrongly presented to doctor too. Most of the time "palpitation" is narrated as breathing difficulty by the common man. If the doctor is insincere in extracting proper history in such case he may cause more harm to this patient. For example if patient is suffering from palpitation and presents to doctor as a complaint of breathing difficulty the doctor may give him Deriphyllin (bronchodilator, a beta-2 agonist) group of medicine which will increase the palpitation to a higher state, thus the patient may become worse. Palpitation literally means "fast beating". In fact due to very fast heart beating (heart rate more than 150 per minute) one may feel difficult to breath normally, which may be expressed to the doctor as breathing difficulty. But, it is doctor's duty to correctly evaluate history and interpret. A patient of heart disease may have palpitation due to volume overload like a *regurgitant valvular lesion* or *anemia heart failure condition*. *Ischemic heart disease* can also cause palpitation by inducing supra ventricular or ventricular tachycardia by various mechanism. A congenital acyanotic left to right shunt lesion like *Atrial Septal Defect* or *Rheumatic valve disease* may also cause paroxysmal supraventricular tachycardia *(PSVT)* or Atrial Fibrillation *(AF)* with rapid ventricular response leading to frequent palpitation. Most of

the time no cause or identifiable structural heart disease is detected for such palpitations, here Electro Physiology Study *(EPS)* helps to indentify the lesion and rectify it by Radio Frequency Ablation *(RFA).* Some time metabolic diseases like Thyrotoxicosis and diseases like cancer, sepsis, chronic obstructive pulmonary disease, obstructive sleep apnea and chronic kidney disease may be responsible for AF. One critical question if asked to a patient of a palpitation like whether this fast beating goes regularly or irregularly one can draw an idea about the presence of AF (irregularly) or PSVT (regularly). The fourth cardinal symptom of cardiovascular diseases is *syncope*. It is the temporary/transient loss of consciousness with spontaneous recovery usually caused by a fall in blood pressure or a long pause in heart rhythm. Amongst all causes about 10% are from cardiovascular diseases, other causes are Neurally mediated reflexes, orthostatic hypotension etc. From cardiovascular causes commonest is rhythm abnormality, followed by valvular and coronary artery disease. Here in the history of syncope one has to be careful in differentiating cardiac syncope from Epileptic seizures, Cervical Spondylitis related vertebral artery insufficiency, micturition syncope, vaso-vagal syncope etc. Most important complaints by the patient of a cardiovascular disorder could be a sudden black out while sitting or performing a work or walking. The patient loses balance after the black out and falls down most of the time injuring him/her. If it happens any time whether in resting condition or during activities it could be rhythm disturbances. It could be due to heart conduction block due to long pause between two consequent beats (at least 3000 ms) like Complete Heart Block (CHB), Sinus pause due to Sick Sinus Syndrome (SSS) or a very fast rhythm causing severe decrease in cardiac output like Ventricular Tachycardia (VT). If the syncope appears on moderate exercise it could be due to low and fixed cardiac output consequent to a narrow ventricular outlet like severe Aortic Stenosis (AS) or Pulmonary Stenosis (PS) or pulmonary embolism. Some time multiple symptoms may co-exist with same pathology or rarely with some other pathology.

Relevance of intermittent symptoms

Some relevant symptoms appear in the duration of present illness repeatedly but disappear and remain absent at the time of patient's presentation to the doctor. They are highly essential to be accounted and to be mentioned in the history of present illness. For example,

in a rheumatic mitral Stenosis case the patient had 3 episodes of hemoptysis within first 3 months. But, no such symptoms existed for the following six months. There is every chance of omitting this point while presenting the history. But it is very important point in establishing a provisional diagnosis of Mitral Stenosis. So also, few episodes of Paroxysmal Nocturnal Dyspnea (PND) in the initial stage of the similar illnesses should not be overlooked. On the contrary, some irrelevant symptoms at times appear to be severe, need not be mentioned. For example, patient had a severe gastro-enteritis due to food poisoning after attending a party for which he/she was admitted in the hospital for 3 days which might not affect the original disease progression. All together one has to cook up some relevant diagnosis before finalizing the summary of the clinical history.

Relevance of Age

Age of a patient gives a gross platform on which many diseases are ruled out or ruled in. For example, a 6 year child comes with history of frequent squatting and discoloration of nails on moderate exercise, one cannot bring a provisional diagnosis of a Coronary Artery Disease. So also a 72 year patient with history of angina and dyspnea should not be thought of congenital heart disease. But, at the same time a young male of 22 year complaining of anginal episode should not be overlooked though he may be looking normal by appearance. Since premature atherosclerosis is speedily increasing in countries like India, one young patient getting an acute myocardial infarction is quite common nowadays.

Relevance of sex

A female of 34 year with history of angina or dyspnea may not compel you to bring Ischemic Heart Disease as a first diagnosis. Because, her reproductive age has natural protection from severe atherosclerosis due to raised estrogen hormone levels. Male sex has preponderance for atherosclerotic arterial disease. But, females of reproductive age in India complaining of dyspnea or angina should be first suspected of rheumatic heart disease and an ischemic heart disease may be brought as second line thinking if she carries the co-morbidities like diabetes, Dyslipidemia, hypertension and a strong family history of coronary heart disease.

Relevance of Religion

In India, people's life styles are mostly guided by the religions. Some are very much fond of *red meat* while some are purely vegetarians. Some vegetarians use most *oily deep fries* along with *pickles* and excessive *butter* or *ghee* which have direct impact on the process of *atherogenesis*. The divergence of lifestyle has been auto designed as per the climate and geographical situation of the states also. Hence, extraction of such history helps a lot in advising a modified life style.

Relevance of Occupation

Sedentarity has been highly focused in area of atherosclerotic heart disease. Hence office workers are more prone than the field workers to get a coronary heart disease. But, one interesting issue needs to be discussed here. Contrary to our common belief, poor worker classes are also prone to atherosclerotic heart disease due to multiple factors. These are tobacco use, gradual adaptation of stressful lifestyle, consumption of unhealthy and cheaper oils for deep fries etc. Hence, a detail food history may sometime show ways to hypothesize a provisional diagnosis.

History of past illness

This part of clinical history some time plays very important role in ruling in or out a diagnosis. For example, a typical migratory polyarthritis with fever in late childhood or adolescent period may certainly rule in a diagnosis of a rheumatic heart disease. Of course absence of such history does not rule out a possibility of being a rheumatic heart disease. Secondly, a past history of Pulmonary TB might say that the patient has gone for profound fibrosis causing severe Dyspnea. It may bring a thought to keep it in a second line diagnosis while evaluating a case of severe dyspnea. Thirdly, a past history of Acute Myocardial Infarction, Angioplasty or a Coronary Artery Bypass Graft (CABG) surgery give sufficient clues to rule in a possibility of a similar event at present since recurrence is not uncommon. Co-morbidities like Diabetes mellitus, Hypertension, Dyslipidemia, Hypo or Hyperthyroidism are very essential risk factors for coronary heart diseases. One has to mention these relevantly. Sometime a past history of a foot gangrene followed by amputation, mostly in smokers-due to Peripheral Artery Disease (PAD) gives an excellent clue to look for coronary artery diseases (CAD) since CAD is two times more common in patients with PAD.

Family History

The concept of inclusion of family history in patient evaluation has to be understood well before you start asking questions. Since basically cardiovascular disease is a non-contagious and non-infective it certainly points to pick up a hint on heredity. For interrogating a young patient suspecting an atherosclerotic coronary heart disease one has to ask if patient's father (or elder brother/any male member) had heart attack at an age below 55 years. Female relatives like mother, sister or aunt getting a heart attack at an age below 65 years is also taken as a significant family history. If somebody's father had died of heart attack at 62 years of age, this cannot be taken as suggestive family history for coronary heart disease. Of course, presences of co-morbidities in the family like Diabetes and Hypertension to be considered as important risk factors from family history point of view. However, in case of a rare and fatal disease like Brugada syndrome and other channelopathies a family history of sudden cardiac death at younger age is highly contributory.

Personal History

While evaluating patient's personal data like his/her life style pattern one has to mark him/her as healthy *lifestyle* adaptor or a faulty one. If the patient *smoke*s or *chews tobacco* or uses *gudakhu (a form of tobacco paste used to rub over gums)* or equivalent, this data imposes a very important impact on the cardio vascular disease evaluation. *For smoking cigarettes* one formula is used i.e., pack years. Example, *One pack-year is smoking 20 cigarettes a day for one year.* If someone has smoked 10 cigarettes a day for 6 years they would have a 3 pack-year history. Someone who has smoked 40 cigarettes (2 packs) daily for 20 years has a 40 pack-year history. Studies have found that relative risk for cardiovascular disease is 2.1 in chronic smokers than recent or short duration smokers. Ex-smokers or who has already quit smoking at least 2 years before has less risk than current smokers. Heavy drinking of alcohol, i.e. more than 60 ml per day is detrimental for atherosclerotic heart disease and Cardiomyopathy. For a female case, one has to ask about her menstrual cycles, menarche, present flow and menopausal status.

Sleep history

In the present practice of medicine the clinical history will not be complete if a sleep history is not obtained. Patient may say-he cannot sleep as much as he wants (insomnia), or has excessive daytime sleepiness (hypersomnia).

For an abnormal pattern of sleep, it is better to ask to the bed partner or roommate of the patient. History of snoring, breathing interruptions during sleep, wakening frequencies, timing of sleep, its duration, day time nap, etc., to be evaluated. History of snoring and struggling respiration may be present in case of obstructive sleep apnea (OSA). In such cases cardiovascular complications are expected like hypertension, arrhythmias, heart failure and coronary artery disease. Excessive desire to sleep and unwillingness to get up in morning from bed along with easy fatigability are symptoms related to hypothyroidism.

Treatment History

Sometime few medications make the patient to develop cardio vascular symptoms. An unmonitored patient with beta blocker therapy may land up in severe bradycardia and syncope or presyncope. Oral contraceptives (conventional old pills) in females may cause Venous Thrombo Embolism and Myocardial infarctions. Of late, with the newer generations of such pills the incidences have come down due to less androgenicity of these agents. Other non-cardiac drugs like salbutamol when over dosed may cause severe palpitation confusing many clinicians as tachy-arrhythmia. Decongestants like pseudo-ephedrine and phenylephrine also raise blood pressure and cause severe headache and chest discomfort. A common mistake by many patients is taking double doses of anti hypertensive drugs and complaining of dizziness. Many patients on calcium channel blockers (nifedipine and amlodipine) come to OPDs with complaints of bilateral pedal edema. One should not hurry up to diagnose it as pathological pedal edema and consider heart failure.

Social history

Although this part of history is neglected in most of the clinical presentation it plays key role in some difficult cases. A person in the society is something like a bolt in the nut. How is its catch or attachment with the nut i.e., society? Some people simply suffer due to maladjustment in the society. One has to take a proper history from the patient as well as from his/her colleagues or friends at times. The patient's personality can be judged from this assessment. One live example of the importance of this history is given below.

One 24 year young man, recently married was admitted in ICU of a well reputed hospital in Chennai with a diagnosis of acute anterior wall myocardial infarction. He was non-diabetic, non-hypertensive,

non-smoker or tobacco user, non obese and had no suggestive family history of premature coronary heart disease. His lipid status was normal. There was no history of marital disharmony. It was very difficult to stratify his coronary risk. On extensive history exploration it was revealed by his friend colleagues that this person keeps himself aloof from others and remains silent. He does not react to any emotional insult or trauma. Probably the stress which he carries in mind has not been released by any conventional manner. This also builds atheromatous plaques and makes vulnerable for coronary event. That is the cause for so many social advices and illustrations to take part in recreations, sports, social and cultural activities. All these build a sound social health. This part of history should not be left behind while assessing a patient as a whole. This is important when a stress generated etiology is strongly suspected but no visible clue is available.

Occupational history

It is a known fact that the mental stress in the occupation or profession has a deep impact on health of a person. This has been the main factor for diabetes, hypertension and coronary event. Jobs where target achievement is a prerequisite stands to be very important risk factor. People working in control rooms, medical professionals and other high risk job doers are on the edge of such disease.

Summary of the Clinical History

This is one of the most important parts of a case presentation which is rarely described in other books. Please remember at the end of your summary of clinical history you should bring maximum 3 possible bed side clinical diagnosis. Here are some illustrations of summaries:

Case no. 1

A 22 year girl of poor socio-economic background (doing all domestic works).

Chief Complaints

Progressive breathlessness since last 2 years, intermittent palpitations since last 6 months.

History of Present Illness

2 years back patient was free from presenting symptoms. She, being a poor woman and uneducated was doing all house hold works like, cleaning the house and its premises, washing cloths and utensils. She used to carry water from a 200 meter distance. To start with she felt shortness of breath initially with carrying full buckets of water 2 years back. Gradually she had to reduce it to only half a bucket of water and some part of house hold works. Now she is incapable of doing her own routine activities without break due to breathlessness. Initially about one and half year back she had 3 to 4 episodes of cough with sputum with streak of blood, which disappeared spontaneously. One year back she had severe suffocative feeling in the night after sleeping for about 3 hours, for which she had to get up from bed and walk a few step inside room opening the windows. She used to feel better within half an hour and then again used to go to sleep. This has been there for four to five times during the last six months. Now for last six months she has been experiencing occasional palpitations at rest as well as during mild exertion. It goes off spontaneously. There is no history of chest pain/fever/blue discoloration of nails or tongue/pedal swelling/giddiness or syncope during this period.

History of past Illness

At the age of 10 year she had prolong fever with multiple large joints swelling and pain one after the other for about a month. For this she was treated in local hospital as an OPD patient.

Family History

None of her siblings had similar illness.

Personal history

She is from a poor socioeconomic background. Her father is a daily laborer. She stays in a slum with 10 X 10 square feet room with her 4 brothers and sisters and parents. She left school study at 4th standard to assist her mother at home. Her menstrual cycles are normal. No history of any addiction.

Treatment history

She was taken to a government hospital one year back for her present complaint. She has been taking some injection every 21 days since then.

Summarizing my case

22 year female of poor socioeconomic background symptomatic in form of progressive exertional dyspnea for 2 years and palpitation for six month and with intervening history of hemoptysis and paroxysmal nocturnal dyspnea with past history suggestive of rheumatic fever is presently on three weekly prophylactic injections.

My provisional diagnosis will be

1. Rheumatic Heart Disease [Favor-p/h/o RF, Prophylactic injection probably long acting Penicillin].
2. Severe Anemia in heart failure [Favor-poor socio/dyspnea and palpitation. Against-Rh Fever/Proph Inj].
3. Cardiomyopathy in Heart Failure [Favor-almost similar presentation. *Against*-Rh Fever/Proph Inj].

    ❖ Now, after having said Rheumatic Heart Disease as first clinical diagnosis on history basis, it is necessary to comment on the valve of your preference which has or have been affected in this case. As per statistics in rheumatic valvular heart disease Mitral Valve is maximally affected and two-thirds of all mitral valve diseases are seen in females in form of Mitral Stenosis. So also in this case my valve of preferences will be *mitral valve* and lesion probably Mitral Stenosis with Mitral Regurgitation.

Case no. 2

47 year, clerk by profession**.**

Chief Complaints

Heaviness in chest on exertion since last 6 months, shortness of breath on exertion since last 2 months.

History of Present Illness

Patient was apparently alright six months back. He used to climb 3 flights of stair cases in his office at 3rd floor regularly without a break for last 20 years. Six months back he started experiencing mild heaviness in the center of his chest after reaching 3rd floor. This discomfort was explained by him as squeezing type which used to radiate to his jaws and used to stay for about 5 to 6 minutes and relieved by taking rest.

After one month he started feeling same discomfort at 2nd floor on the stair case. He had to take a break for 10 minutes before he could take another attempt to reach his office at 3rd floor. Presently patient cannot climb more than 5 steps without chest pain and sweating. He also experienced shortness of breath while climbing these stairs since last 2 months. This shortness of breath has progressively increased now and he cannot climb more than 6 steps with a stop to take breath for 2 minutes. Patient has no complaints at rest. During this last six months he never had palpitation/giddiness/syncope/fever or cough.

History of past Illness

He is known case of Type 2 Diabetes mellitus and Hypertension since last 10 years and on regular treatment. He had no similar illness in the past. There is no past history of pulmonary TB, Bronchial asthma.

Family History

None of his family members had a history of premature heart attack. His both parents are Diabetic and mother is Hypertensive.

Personal history

Patient is a sedentary worker. He has been smoking 10 to 12 cigarettes a day for last 12 years (12 pack years). He never goes for regular walk or exercise. He hardly takes fruits and green vegetables in his regular diets. He has never gone for a preventive check up for his cardiac risk analysis. He is married and has 2 children.

Treatment history

Patient being a known case of DM and HTN he is taking Tab. Metformin 500 mg twice daily and Tab. Amlodipine 5 mg daily once at night. After the present ailments he had consulted a local doctor who had prescribed him Tab. Sublingual nitrate 5 mg only to take during chest pain.

Summarizing my case

A 47 year male, diabetic, hypertensive, smoker and faulty life style maintainer has typical history suggestive of chronic stable angina NYHA CLASS II. He has all 4 major risk factors like, DM/HTN/smoking and physical inactivity to develop accelerated atherosclerosis.

My provisional diagnosis is:

1. CORONARY ARTERY DISEASE, CHRONIC STABLE ANGINA NYHA CLASS II [Risk Factors & typical angina].
2. SEVERE PROGRESSIVE ANEMIA OF UNCERTAIN CAUSE [It can cause ANGINA & DYSPNOEA resulting in Heart Failure, But NO H/O face puffiness or pedal swelling. All 4 major Risk Factors are *against* it].

❖ The above two different type of cases have illustrated that while extracting history one has to exercise care so that *specificity, relevancy and descriptive* nature of fact presentation should only be picked up. There may be many distracting type of symptoms explained by the patient during your interrogation. For example patients may tell you that –"I have this type of gas problem on and off" etc. "I had taken more non-veg diet during the last one month, for that I have been suffering" etc. But the clinician has to listen all and pick up only relevant facts.

❖ One more important point has to be remembered that at times patient may deny any progression of his/her dyspnea or angina on exertion. Here the attending doctor may be highly misguided. Patient may even say that he/she is rather asymptomatic. This does not mean that the patient is lying. In fact the patient has adapted a low profile lifestyle according his/her capacity and now restricted his/her physical activity to such an extent that there is no symptom. Here you have to ask some tricky questions like, can you walk in same speed what you used to do six months before? Can you walk as fast as me? Somehow you have to interrogate such a way that if he/she admits that he/she cannot perform this much because of fear of developing angina or dyspnea. This is how hidden symptoms may be unveiled.

Summarizing the clinical history concisely and then coming out with few close provisional diagnoses is the real art of medicine.

Fever is never out of cardiovascular system (CVS)

It is often observed that in a history of fever cardiovascular system is sidetracked. After an exhaustive search for a cause of fever the door of the cardiovascular system is knocked. By this time fever takes a long

course of around 2 weeks. There are three ways with which a febrile patient gets CVS examined.

First, when a patient complaints of chest pain or heaviness of chest along with fever and severe weakness. An early auscultation may reveal a S3 or murmur or a pericardial rub. This opens up the window of CVS and the patient undergoes further investigations like a bedside Echocardiography. This may reveal (i) a very weakly contracting myocardium (myocarditis), (ii) a valvular vegetation causing turbulence in linear flow (Infective Endocarditis) or (iii) a minimal fluid in pericardial sac (Pericarditis).

Secondly, situation may also arise when a patient does not have any symptom relevant to CVS. Even on auscultation the precordium is silent. It may be an unwanted investigation to ask for an Echocardiogram on 3rd, 4th or 5th day of fever. Thus it gets delayed for completion of all rare and uncommon relevant tests like immunological profile, blood culture etc. Finally an Echocardiogram gets a signal and a huge pericardial effusion is revealed. The fluid after pericardiocentesis gets some positive features of suspecting Tuberculosis. This can happen even when all other indirect test for Tuberculosis is negative. Then this fever gets a way to its end. Echocardiogram may also reveal some large vegetation which is clinically silent and may help to establish or rule out a possibility of Infective Endocarditis.

Thirdly, an arterial bruit may reveal a possibility of Arteritis specially auscultation near subclavian, carotid or brachiocephalic artery.

Hence, it is always advisable that in whatever situation a fever case arrives, do a careful and detail cardiovascular examination.

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