Good doctor and a Patient

The good physician knows his patients through and through. Time, sympathy and understanding must be lavishly dispensed, but the reward is to be found in that personal bond which forms the greatest satisfaction of the practice of medicine. One of the most essential qualities of the clinician is interest in humanity, for the secret of the care of the patient is in caring for the patient.


Francis Weld Peabody
1881-1927

How do you show your patients that you care about them?

• I am now 90 years of age, so the interns and residents come to my retirement home on the second Tuesday of the month for a dinner and a teaching session.

• "The caring doctor studies all aspects of the patient’s case, but the patient does not know that, How do you show your patients that you care about them?"
  – One resident said that the doctor should smile, sit down, and use the patient’s name when speaking.

• "No patient likes a gloomy doctor, and no doctor should see a patient without using the patient’s name. The reverse is also true—the patient should know the doctor’s name. The history-taking period is when the doctor not only obtains vital clinical information, but also has the opportunity to know the patient as a person—as a human being. This is also when patients begin to know their doctor as a person. It is during the history-taking period that patients decide if the doctor is a robot with no feeling or a caring individual.

How do you know when the patient understands?

"At times, it is useful to hold the patient’s hand, elbow, or shoulder. Older people, especially, are often frightened and feel rejected. They feel comforted when they are touched. They recognize the human touch as a signal of caring because machines don’t comfort people."

• "Talk with others who are in the patient’s room. Speaking with the spouse and children is a must."

• "The doctor who asks questions like an interrogating lawyer and pays little attention to a patient’s answers is doomed to being a poor diagnostician, because the patient’s medical history is often the doctor’s best diagnostic tool. Patients should know that the doctor cares about them as persons, after the history-taking period is over."

• "How do you know when the patient understands? I suggest that the doctor should explain his or her diagnosis and treatment and then ask the patient to repeat it. You must teach. Use a white board and black marker—draw pictures—teach and teach."

Discuss the options in care with your patient

• "Never keep patients waiting for more than 10 minutes unless you explain to them why you are running late. Should you have an emergency, tell the patients who are scheduled to see you that you can’t see them on time. They will understand if you point out that you would go to them if they had an emergency."

• "Discuss whom the patient should call if you are not available. Introduce your back-up doctor to your patients. Assure your patients that during such times they will always be covered by the competent back-up physician."

• "Discuss the options in care with your patient. Learn to bring bad news to a patient with skill and empathy, including end-of-life discussions."

• "You, the doctor, should call a new patient the day after he or she has been seen in the office, in order to re-emphasize some of your previous discussion and to answer any questions."

• "You should call your patient when you receive any new information. Do not turn this action over to any other person."
Remember, patients can identify the doctor who is not sincere.

- “You should give your patients a copy of your work-up. They will especially appreciate a copy of your well-formulated ‘Problem List’ that you keep up-to-date on them.”

- “Finally, patients want to know their doctor as a person. Accordingly, all of a doctor’s actions should be colored by his or her personality. Every action and comment made by the doctor must be genuine. Doctors must not simply memorize their actions and comments like actors do when they act in a play. Remember, patients can identify the doctor who is not sincere.”

History taking
(The Art of Interviewing)

- Basic principles
  communication: enough time, friendly atmosphere, body language, more listening, ...

- The rule of 5 vowels (Audition, Evaluation, Inquiry, Observation, Understanding)

- Powerful diagnostic tool

Symptoms and Signs

- Symptoms - what a patient feels
  - constitutional
  - other

- Signs - what examiner finds

The major TASK of the interviewer is to sort out symptoms and signs .... This is to make a diagnosis

CV history (1)

CV history (2)

CV history (3)
Science of Physical Examination

Do not touch the patient

- state first what you see
- cultivate your process of observation

Sir W. Osler, 1849 - 1919

The four cardinal principles of physical examination

- Inspection
- Palpation
- Percussion
- Auscultation

The student must

„teach the eye to see, the finger to feel and the ear to hear“

VIDEO

Physical examination of CVS

HEART MURMURS
AND VALVULAR HEART DISEASES

Heart murmurs

- Murmur = Flow turbulence

- Murmur intensity is
  - directly proportional to:
    • $\text{[velocity]}^4$
    • $\text{[flow]}^2$
  - inversely proportional to:
    • viscosity (e.g. hematocrit)
    • orifice area

Heart murmurs

- “Pathologic” or “Innocent”
- “Maneuvers” – BASICALLY to differentiate
  - “Left-” vs. “Right-” sided
  - Stenotic vs. regurgitant murmurs
Heart murmurs: Loudness

I – very faint, heard only under optimal conditions
II – faint, but heard in the presence of the background noise
III – moderately loud
IV – very loud, but no thrill
V – very loud with thrill
VI – audible without stethoscope

Innocent Murmurs

• Common in asymptomatic adults
• Characterized by:
  – Grade I-II @ LSB
  – Systolic ejection pattern
  – No other abnormal sounds or murmurs
  – Normal intensity & splitting of S2
  – No evidence of LVH, and no ↑ with Valsalva

Common Valvular Heart Diseases
(by murmur timing/quality)

Systolic Murmurs:
• Aortic stenosis
• Mitral regurgitation
• Mitral valve prolapse
• Tricuspid regurgitation

Diastolic Murmurs:
• Aortic regurgitation
• Mitral stenosis

Aortic Valve Disease

Aortic Stenosis
Aortic regurgitation

Aortic Stenosis: Etiology

• Congenital bicuspid valve is the most common abnormality
• Rheumatic heart disease and degeneration with calcification are found as well

Normal Bicuspid Ao V “Normal” geriatric calcific valve
Aortic Stenosis: Symptoms

- Cardinal Symptoms
  - Chest pain (angina)
    - “Demand” >>> “supply” problem
  - Syncope (~ fainting)
  - Dyspnea on exertion & rest
- Other signs of LV failure (RV failure, edema)

Aortic Stenosis: Physical Findings

- Intensity DOES NOT predict severity
- “Diamond” shaped, systolic cresendo-decrescendo
- Decreased, delay & prolongation of pulse amplitude
- S4 (with left ventricular hypertrophy)
- S3 (with left ventricular failure)

Aortic Stenosis: Prognosis

<table>
<thead>
<tr>
<th>Symptom/Sign</th>
<th>Life expectancy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina</td>
<td>5 years</td>
</tr>
<tr>
<td>Syncope</td>
<td>2-3 years</td>
</tr>
<tr>
<td>Congestive Heart Failure</td>
<td>1-2 years</td>
</tr>
</tbody>
</table>

Tx:
- Valve replacement for severe aortic stenosis
- TAVI (Transcatheter aortic valve implantation)

Aortic regurgitation: Etiology

- Any conditions resulting in incompetent aortic leaflets:
  - Rheumatic heart disease
  - Dilated cardiomyopathy (e.g. hypertension, etc.)
  - Syphilis (chronic aortitis)
  - Collagen disorders (e.g. Marfan’s)
  - Connective tissue disorders
    - E.g. ankylosing spondylitis, rheumatoid arthritis, Reiter’s syndrome
  - Cystic medial necrosis
- Acute AR: aortic dissection, infective endocarditis
Aortic regurgitation: Symptoms

- Dyspnea, orthopnea, and chest pain.
- Nocturnal angina >> exertional angina
- With extreme reductions in diastolic pressures (e.g. < 40) may see angina pectoris

Aortic regurgitation: physical Exam

- Widened pulse pressure
  - Syst. – diast. = pulse pressure
- High pitched, blowing, decrescendo diastolic murmur at LSB (end expiration & leaning forward)
- Plethora of named signs assoc. w/ ↑ pulse pressure:
  - De Musset's S.
  - "Pistol shot": loud 'shot' over fem. arteries
  - Quincke's S: pulsations in the nail bed
  - Corrigan's Pulse
  - MANY others

Aortic regurgitation: natural History

Asymptomatic %/Y
- Normal LV function (~good prognosis) < 6
- Progression to symptoms or LV dysfunction < 6
- Progression to asymptomatic LV dysfunction < 3.5
- 75% 5-year survival
- Sudden death < 0.2

Symptomatic (Poor prognosis)
- Mortality >10

Tx: Medical → Surgery BEFORE LV dysfunction

Mitral Valve Disease:

Mitral Regurgitation
Mitral Valve Prolapse
Mitral Stenosis

Mitral regurgitation: Etiology

- Degenerative MR most common in developed countries ...
- Rheumatic etiology – mostly in less developed countries; in males >> females
- May also occur as a congenital anomaly ....
- Acute regurgitation may occur 2º
  - Infective endocarditis
  - Acute myocardial infarction
  - Trauma
Mitral regurgitation: Symptoms

- Fatigue
- Exertional dyspnea, and orthopnea (more prominent with chronic, severe MR)
- Hemoptysis and systemic embolization (occur less frequently in MR)

Mitral regurgitation: Physical Exam

- Apical holosystolic murmur
- Radiation to the axilla
- Palpable thrill at cardiac apex

Treatment
- Afterload
- Mitral valve repair...
- Mitral valve clips...
Mitral Valve Prolapse
Degenerative or myxomatous MV disease

Mitral Valve Prolapse: Epidemiology
- Affects 5-10% of population
- Most common cause of isolated severe MR
- Females >> males; Ages of 14 and 30 years
- Strong hereditary component (?) Autosom. Dom

Mitral Valve Prolapse: Symptoms
- Majority are asymptomatic for entire life
- Palpitations
- Chest pain (atypical):
  - Often substernal, prolonged, poorly related to exertion, and rarely resembles typical angina
- Syncope

Mitral Valve Prolapse: Complications
- Arrhythmias (PVC, PSVT>>VT)
- Transient cerebral ischemia (embolic – rare)
- Infective endocarditis (if assoc with MR)
- Sudden death (rare)
- Severe MR ...

<table>
<thead>
<tr>
<th>Differiating Characteristics</th>
<th>Barlow Disease</th>
<th>Fibroelastic Deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathology</td>
<td>Excess leaflet tissue due to accumulation of mesenchymal chondral cells</td>
<td>Loss of mechanical integrity due to impaired production of connective tissue</td>
</tr>
<tr>
<td>Typical age at diagnosis</td>
<td>Younger (&lt; 40 years old)</td>
<td>Older (&gt; 40 years old)</td>
</tr>
<tr>
<td>Duration of disease</td>
<td>Years to decades</td>
<td>Days to months</td>
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<tr>
<td>Physical exam</td>
<td>Mitral stenosis click and late systolic murmur</td>
<td>Holosystolic murmur</td>
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<tr>
<td>Leaflet Involvement</td>
<td>Multisegmental</td>
<td>Unisegmental</td>
</tr>
<tr>
<td>Leaflet lesions</td>
<td>Leaflet thickening and thickening</td>
<td>Thick leaflet with thickened involved segment</td>
</tr>
<tr>
<td>Chordal lesions</td>
<td>Chordal thickening and elongation</td>
<td>Chordal elongation and chordal rupture</td>
</tr>
<tr>
<td>Commissural classification</td>
<td>Type II</td>
<td>Type II</td>
</tr>
<tr>
<td>Type of dysfunction</td>
<td>Bilobed prolapse</td>
<td>Prolapse and/or flail</td>
</tr>
<tr>
<td>Complexity of valve repair</td>
<td>More complex</td>
<td>Less complex</td>
</tr>
</tbody>
</table>
Mitral Valve Prolapse: Physical Exam

- Most important finding: mid → late systolic click
  - Acute tensing of the mitral valve chordae
- Variable murmurs:
  - high pitched late systolic crescendo-decrescendo murmur,
  - Occasionally “whooping” or “honking” at the apex

Mitral Valve Prolapse: Treatment

- Reassurance
  - Asymptomatic pts w/o sev MR or arrhythmia.
  - Follow-up q 2-4 years, with ECHO
- Beta blocker treatment for atypical chest pain
- Infective endocarditis prophylaxis with
  - Systolic murmur &/or
  - Typical echocardiographic findings
- Severe MR - MVP, MVR, MVC

Mitral Valve Prolapse: Etiology

- Primarily a result of rheumatic fever
  (~99% of MV’s surgery show rheumatic damage)
- Rarely congenital
- Pure or predominant MS occurs in approximately 40% of all patients with rheumatic heart disease
- Two-thirds of all patients with MS are female.

Mitral Stenosis: Etiology

- Primarily a result of rheumatic fever
  (~99% of MV’s surgery show rheumatic damage)
- Rarely congenital
- Pure or predominant MS occurs in approximately 40% of all patients with rheumatic heart disease
- Two-thirds of all patients with MS are female.

Mitral Stenosis: Pathophysiology

- Scarring & fusion of valve apparatus
- MI valve area: 4-6 cm²
- Mild mitral stenosis:
  - MVA <2.5 cm²
  - Minimal symptoms
- Mod mitral stenosis
  - MVA >1.5 cm² usually does not produce symptoms at rest
- Severe mitral stenosis
  - MVA < 1.0 cm²
  - ↑ left atrial pressure → ↑ pulmonary venous pressure → dyspnea

“Mitra” view of mitral valve in patient with rheumatic mitral stenosis.
Mitral Stenosis: Symptoms

- Cough, palpitations, chest pain, orthopnea, pulmonary edema, hemoptysis
- Worsened by conditions that ↑ cardiac output (exertion, excitement, etc.)

Mitral Stenosis: Physical Exam

- First heart sound (S1) is accentuated and snapping
- Opening snap (OS) after aortic valve closure
- Low pitch diastolic rumble at the apex
- Pre-systolic accentuation (esp. if in sinus rhythm)

2D echo – Mitral Stenosis

- Progressive, lifelong disease
- Slow & stable in the early years
- Progressive acceleration in the later years
- 20-40 year latency from rheumatic fever to symptom onset
- Additional 10 years before disabling symptoms

Mitral Stenosis: Complications

- Atrial dysrhythmias - atrial fibrillation ...
- Systemic embolization (10-25%)
- Congestive heart failure
- Pulmonary infarcts (result of severe CHF)
- Hemoptysis
- Endocarditis
- Pulmonary infections

Mitral Stenosis: Treatment

- Endocarditis prophylaxis
- Anticoagulation if concurrent A-Fib or previous embolic event
- Valve repair/replacement, PTMV
• The acute coronary syndrome represents a wide spectrum of clinical syndromes from unstable angina pectoris through NSTEMI to STEMI.

Atherothrombosis is a generalized and progressive process. Atherothrombosis is characterised as sudden (unexpected) disruption of AS plaque (rupture or erosion) leading to activation of thrombocytes and the formation of thrombus.

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High Prevalence of Coronary Atherosclerosis in Asymptomatic Teenagers and Young Adults. Evidence From IVUS

- IVUS was performed in 262 heart transplant recipients 30±13.2 days after transplantation to investigate coronary arteries in young asymptomatic subjects.
- The donor population consisted of 146 men and 116 women (mean age of 33.4±13.2 years).
- Extensive imaging of all possible coronary segments was performed. Sites with intimal thickness ≥0.5 mm were defined as atherosclerotic.
- A total of 2014 sites within 1477 segments in 574 coronary arteries (2.2 arteries per person) were analyzed. An atherosclerotic lesion was present in 136 patients, or 51.9%.


Atherosclerotic changes in LAD in 17-year old men/boy (A). Severe AS changes in 32-year old women (B)

Is CAD in young different than in old age?

- Young pts have different risk factor profiles, clinical presentations, and prognoses than older patients.

- The relative importance of RF for the development of CAD according to age was evaluated in a study in which 11,016 men aged 18 to 39 years were followed for 20 years.
- The relative risks associated with the traditional risk factors. These included:
  - Age — RR 1.63 per six year increase
  - Serum cholesterol — RR 1.92 per 1.04 mmol/L increase
  - Systolic blood pressure — RR 1.32 per 20 mmHg increase
  - Cigarette smoking — 1.36 per 10 cigarette/day increase
- Many of young have multiple risk factors...

CONCLUSION

- Atherosclerosis begins at a very young age and that lesions are present in 1 of 6 teenagers.
- The prevalence of atherosclerosis varied from 17% in individuals <20 years old to 85% in subjects 50 years old.
- These findings suggest the need for VERY intensive efforts at coronary disease prevention in young adults nowadays...

Cardiovascular risk profile in young women and long-term risk of cardiovascular and all-cause mortality is favorable

- In a prospective study of over 7000 women of mean age 27 years at baseline followed for an average of 31 years, there were 47 CAD deaths.
- The CAD mortality rates for those with:
  - no risk factors
  - only one risk factor, or
  - two or more risk factors were:
    - 0.7,
    - 2.4, and
    - 5.4 per 1000 person-years, respectively

Dynamic coronary obstruction

ACS - ECG DIAGNOSTICS

Normal LAD coronary anatomy

Normal RCA anatomy
Left main CA

Tight stenosis of distal part of the main CA

Patient with exertional AP

Collateralized CA

Tight stenosis of LAD

Clinical utilisation of echokg

Multivessel CAD

Clinical complications, chronic CAD
Acute and chronic heart failure – systolic, diastolic, combined
Pericardial diseases ...
Valvular dis. ...
Congenital dis. ...
Cardiomyopathies
Complications of hypertension – LVH, regression, progression, diast. dysfunction
Cardiac source of emboli
Bacterial endocarditis ...
Acute pulmonary emboli
Aortic dissection, aneurysm
Many others ...

Answer to number of clinically relevant questions:

- Murmur
- LV/RV function
- Pericardial effusion
- WMA
- Prognosis of pt
- Therapy effects (cardiac treatment, chemotherapy inte)
- Assistance at interventional exams (ASD closure, resynchronisation therapy ...
- Assistance before and perioperative at valvular plastiques
- Management of atrial fibriation

Symptoms and signs

- Panystolic murmur s-/brill
- CHF/pulmonary edema
- Hypotension, cardiogenic shock

Diagnosis

Swan-Ganz catheterization

Stabilization

IABP
Inotropic agents
Afterload reduction

Definitive treatment

Mitrval valve replacement/surgery
Surgical repair +/- CABG
Aneurysm and LV pseudoaneurysm post ACS

- **True aneurysm**, protrudes in systole and diastole, wide neck, relatively rarely lead to rupture

- **LV False or pseudoaneurysm**, protrudes in systole and diastole, neck is narrow, it is rupture of myocardial wall. Its wall consists of parietal pericardium, always with thrombus and frequently lead to rupture...

Remodeling post ACS

WMA - echo
Less mobile thrombus in the LV apex post ACS

ACS – complications
ruptures, MR, defects

- Rupt. of ventricular septum
- Rupt. free LV wall
- Rupt. of papillary muscle

MR due to:
- dysfunction / rupture of papill. muscle, or valve supportive apparatus
- LV dilatation
Coronary artery territories

![Diagram showing coronary artery territories]

Typical distributions of the right coronary artery (RCA), the left anterior descending artery (LAD), and the circumflex artery (CX). The arterial distribution varies between patients. Some segments have variable coronary perfusion.

Reproduced from: Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. Eur J Echocardiogr. 2015;16(1):227-247. Illustration used with the permission of Elsevier. All rights reserved.