Evidence-Based Physical Diagnosis

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No Financial Relationships to Disclose
Outline

• Vital Signs

• Listening to the Heart

• The Funduscopic Exam
Likelihood Ratio

Positive: Probability of a person with a disease testing positive divided by probability of a person without a disease testing positive

Negative: Probability of a person with a disease testing negative divided by probability of a person without a disease testing negative
Systemic Inflammatory Response Syndrome (SIRS) (≥2)

- Fever or hypothermia (>38ºC or <36ºC)
- Tachycardia (>90)
- Tachypnea (>20)
- High/low WBC (>12, <4, ≥10% bands)

WBC = white blood cell count.

Question: If a nurse records a respiratory rate of 20, you should...?

A – Assume that R is between 18-22

B – Assume that most of the time this is correct

C – Assume that it is wrong
Bias in Reporting Respiratory Rate

Key Point:
Count Respiratory Rate Yourself!

Can We Identify and Treat the Patient with Severe Sepsis Early?

SIRS + infection → Sepsis + end organ failure → Severe sepsis + SBP<90 after 500ml/bolus

Sepsis → Severe Sepsis → Septic Shock
## Frequency of Sepsis Syndromes

<table>
<thead>
<tr>
<th></th>
<th>Sepsis</th>
<th>Severe Sepsis</th>
<th>Septic Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cult (+) 26%</td>
<td>18%</td>
<td>4%</td>
<td></td>
</tr>
<tr>
<td>Cult (-) 35%</td>
<td>21%</td>
<td>3%</td>
<td></td>
</tr>
</tbody>
</table>

~ 50% Sensitivity for the Definitions

On High Risk Wards – 10% Prevalence
PPV ~ 50% and NPV ~ 88% for SIRS Predicting Sepsis

Rangel-Frausto et al JAMA 1995;273:117-123
# Sepsis: The Patient with Pneumonia

- Likelihood Ratio -

<table>
<thead>
<tr>
<th>Finding</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR &gt; 28</td>
<td>2.7</td>
<td>0.9</td>
</tr>
<tr>
<td>T &gt; 37.8</td>
<td>2.2</td>
<td>0.7</td>
</tr>
<tr>
<td>HR &gt; 100</td>
<td>1.7</td>
<td>0.8</td>
</tr>
<tr>
<td>All are Normal</td>
<td>0.3</td>
<td>2.2</td>
</tr>
<tr>
<td>Dull Percussion</td>
<td>3.0</td>
<td>NS</td>
</tr>
<tr>
<td>Rales</td>
<td>1.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Bronchial BS</td>
<td>3.3</td>
<td>NS</td>
</tr>
<tr>
<td>Egophony</td>
<td>4.1</td>
<td>NS</td>
</tr>
</tbody>
</table>

SIRS CRITERIA PREDICT BUT EGOPHONY IS MOST USEFUL

McGee
In Shock – Differential Diagnosis

- Sepsis – Decreased SVR
- Cardiac – Decreased CO
- Decreased Intravascular Volume
- Endocrine – Adrenal Insufficiency
  Pheochromocytoma
  Hyperthyroidism
- Obstructive – Massive PE
  Cardiac Tamponade
If a patient with severe sepsis has a pulse Ox of 93%

A – it is likely to be at least 93%
B – it is likely to be 90-93%
C – there is a 50% likelihood that it is really <90%
# Pulse Oximetry in ED Patients with Severe Sepsis and Septic Shock

<table>
<thead>
<tr>
<th>No</th>
<th>Pulse Ox Value</th>
<th>Blood Gas Value $\text{SaO}_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>28</td>
<td>$\geq 98%$</td>
<td>all had $&gt;90%$</td>
</tr>
<tr>
<td>31</td>
<td>94-97%</td>
<td>10% $&lt; 90%$</td>
</tr>
<tr>
<td>22</td>
<td>90-93%</td>
<td>50% $&lt; 90%$</td>
</tr>
</tbody>
</table>

Pulse oximeters overestimate measures $\text{SaO}_2$ in this patient population

## Hypovolemic Shock

<table>
<thead>
<tr>
<th>Condition</th>
<th>Sens%</th>
<th>Spec %</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dry Axilla</td>
<td>50</td>
<td>82</td>
<td>2.8</td>
<td>NS</td>
</tr>
<tr>
<td>Dry Mucous Membranes</td>
<td>49 – 85</td>
<td>58 – 88</td>
<td>3.1</td>
<td>0.4</td>
</tr>
<tr>
<td>Sunken Eyes</td>
<td>62</td>
<td>82</td>
<td>NS</td>
<td>0.5</td>
</tr>
<tr>
<td>Abnormal Skin Turgor</td>
<td>73</td>
<td>79</td>
<td>3.5</td>
<td>0.3</td>
</tr>
</tbody>
</table>

Note: confusion and weakness were not predictive

* > 3 seconds persistent tenting after 3 seconds pinch

McGee
Looking at the Blood Pressure

170/50  Wide PP  Possible Aortic Insufficiency
110/90  Narrow PP  Any Hypermetabolic state

Possible Aortic Insufficiency
Any Hypermetabolic state

Proportional BP:
$S-D$
$S$
$< 25\%: CI < 2.2$
Looking at Systolic BP: 3 Scenarios
130 / vs 90/
Possible Subclavian Steel

INTRODUCTION

SYNONYMS—
(a) Subclavian Steal Phenomenon

(b) Subclavian Steal Steno– Occlusive Disease
Inter-Arm Differences in BP

462 Subjects – 4 Recordings
Supine and after 10’ Rest
Δ SBP 1.1 mm (range - 9 to 11)

Δ DBP 0 mm (range – 10 to 10)

Orme et al age aging 1999;28:537-42

Meta-Analysis 20 Studies:
in proven subclavian stenosis
-50% occlusion by Angiogram
Δ SBP 36.9 mm (Cl_{95} 35.4 – 38.4)
Δ SBP ≥10: RR 8.8 (Cl_{95} 3.6 – 21.2)

Clark et al Lancet 2012; 379:905-14
Subclavian Steal

Cause – Atherosclerosis
Takayasu Arteritis
Kinking over 1st Rib
(golfers; baseball pitchers)

Symptoms - Ischemia of Arm; Vertebrobasilar insufficiency: dizzy, ataxia vertigo, diplopia

Possible Vertebral Artery Bruit over a Suboccipital Region
Systolic BP: Right Arm vs Right Leg

110/ vs 90/

Possible Coarctation of the Aorta

Wikipedia
Systolic BP: Right Arm vs Right Leg

Possible Aortic Insufficiency
**Hill’s Sign and Severity of AI**

Variability of Arm vs Leg SBP

SBP Leg > SBP Arm Normally

- Usually < 10 mm Hg; if > 20: positive Hill’s sign

\[ \text{If } \Delta \text{ of } 20 - 40 \text{ mild} \]
\[ 40 - 60 \text{ moderate} \]
\[ > 60 \text{ severe} \]

False positive: Selective Upper Extremity Atherosclerosis

False Negative: . Atherosclerosis of Lower Extremity or Interference in Pulse Wave ie AS, MS

See Sapira, Art and Science of Bedside Diagnosis
LWW 2000 Philadelphia
Count the Number of SIRs Criteria

Count the RR Yourself!
  Don’t believe a “20”!

In shock – consider the differential Dx

Look at PP and Proportional BP: $S - D_S$

Right Arm vs Left Arm: Possible Subclavian Steel
  Mean Difference is 37; $\geq 10$ mm
  Predictive

Right Arm vs Right Leg SBP:
  R/O Coarctation if Arm $> Leg$
  R/O AI if Leg $> Arm$ by $\geq 20$ mm
Listening to the Heart: Systematic Approach

PMI – Location, Size

Rate – Just a Number

Rhythm – Regular or Irregular

$S_1$ – Avoid the Term “Normal” Describe findings

$S_2$

$S_3/S_4$

Murmurs - Location, Systolic or Diastolic Grade, Features
The PMI

Size: A Dime Normally

If Quarter: 92 – 100% Sensitivity for left vent dilatation


If MR: Nickel, Quarter, 50¢ Correlates with Mild, Mod, Severe MR, Respectively

Location: Lateral to MCL

Predicts \( \text{LVent DILATATION} \)

\( \text{PPV} \geq 50\% \)

\( \text{LR 5.1}^* \)

Prolonged Thrust: Over 2/3 systole

Predicts \( \text{LVent HYPTERTROPHY} \)

\( \text{LR 5.6}^* \)

Br Heart J 1062; 24:1/09-21

*McGee
Regarding Rate: Just a Number

Quietly Resolve Never to Say

“Regular Rate and Rhythm”

or Record “RRR”.

Closure of Mitral and Tricuspid Values

The Longer Time it Takes for Ventricular Contraction (The Longer the PR Internal), The Valves are Lifted Nearer to Closure by Blood Filling the Ventricle, and thus the Softer the first Heart Sound.
If you had Bionic Ears and you Listen to $S_1$
How would you Identify a 1\textsuperscript{st} Degree AV Block?

A Soft $S_1$

How Would You Identify a Patient with Wenckebach Phenomenon?

Increasing Softness of $S_1$ and then Return to Soft, Softer, Softest with Each $S_1$
First Heart Sound

Variation in $S_1$ Amplitude First Noted by Griffith in 1912

Heart 3 1912; 143

Soft $S_1$ if P-R 200 - 500 msec

Am Heart D 1947; 34:809

Below PR Values of 200 msec; inverse relation between P-R and $S_1$ Amplitude
How Would $S_1$ Sound in the Presence of Complete A-V Dissociation?

Where Would the AV Valves be at the Time of Successive Systolic Contraction of the Ventricle

In complete Heart Block There is Variation in $S_1$

Completely Variable Intensity of $S_1$

L.R. 24.4*

*Circulation 1974; 50:17-24

*McGee
Normal if “Constant Intensity”
(Do you want to wager on the P-R interval?)

Very Soft if P-R >200 msec

Loud if P-R Approaches 120 msec

Totally Variable Intensity if Complete Heart Block:
Also Regular Bradycardia and Jugular Cannon A Waves

Patterned Intensity with Mobitz 1, 2nd Degree Block
Physiological Splitting of $S_2$ with Inspiration, More Blood to Right Heart and Delayed Closure of Pulmonic Valve

Listen to $S_2$ over pulmonic area
Possible Sounds:
- Physiological Split
- Fixed Split
- Paradoxical Split
- Single Sound
- Cannot Tell
$S_2$ – If Right to Left Atrial Shunt
Atrial Septal Defect

Already Delayed Pulmonic Closure
Before Inspiration

Fixed Split is Possible

Wikipedia
Fixed Splitting of $S_2$ that is Wide

I - Delayed Closure of Pulmonic Valve
  RBBB – Electrical
  ASD
  Pulmonic Stenosis
  Acute Massive Pulmonary Embolus

II - Early Aortic Valve Closure
  Mitral Insufficiency
  VSD
  | A | P |
  During Inspiration:

  | A | or | P |
Fixed Split S2

The Heart Sound Series

"S2 - Fixed Split"

By: Dr. Parth S. Solanki
dr_parth_solanki@yahoo.co.in
$S_2$: Paradoxical Split

With Inspiration

In Paradoxical Split – There is a Delay in Aortic Value Closure Usually

Consider: Severe Aortic Stenosis or LBBB
So Far...If Heart Examine is Normal

PMI – Dime Size in MCL

$S_1$ – Constant Intensity (Do you want to wager on the PR interval?)

$S_2$ – Physiological Split
S₃ and S₄

S₃ – Marker of CHF – “Kentucky”
S₁ / S₂ / S₃

S₄ – Maybe Normal in Young Adults
“Tennessee”
S₄ / S₁ / S₂
Analogy to tensing of a handkerchief between two hands: Abrupt tensing produces sound
Slow tensing is silent

\[ S_3 \]

\( S_3 \) during exaggerated early diastolic filling: energy transmitted to ventricular walls causing vibration and sound

Quote from McGee
In Mitral Insufficiency

PMI: Often Displaced Laterally
Increasing Size with Increasing Severity
At the bedside, Grade the MR!

Murmur Often Holosystolic
Grade 3/6 and High Pitched Sound
Radiation to Axilla
Aortic Insufficiency

Look at PP
  SBP Leg > Arm: Hill’s Sign

Grade the AI at the Bedside
  Listen: Patient Leans forward Holds Breath

Early: Aortic Area or Erb’s Point
  Soft 1 – 2 Diastolic Decrescendo Murmur

Later: As Severity Increases, the Murmur can be Longer and More Harsh
Predicting Severity of MR (N=170) and AI (N=40)

Intensity 0-5 for MR
    0-3 for AI

Correlation of Severity with Intensity:
    \( R^2 = .54 \) for MR
    \( R^2 = .45 \) for AI

Murmur \( \geq 3 \) for AI: 71% had Severe
    \( \geq 4 \) MR: 91% had Severe

Desjardins et al Amer J Med 1996; 100: 149-56
Aortic Stenosis

Usually at Aortic Area
Harsh 3/6 Systolic Crescendo – Decrescendo Murmur
Radiating up to Clavicle and Carotids
With Increasing Severity – Murmur will have a Delayed Peak in Intensity (Longer Murmur)

LR for delayed peak in Severe is 4.4*

*McGee

Delayed Closure of Aortic Valve: If Severe AS – Single S₂ or Paradoxical S₂

A NORMAL S₂ RULES OUT AS
Aortic Stenosis Severity

Single $S_2$ or Paradoxical $S_2$
Correlates with Severe AS
- See Sapira

$\geq 3$ of Following Predicted Mod or Severe AS:

- Clavicular Murmur
- Reduced Carotid Volume
- Soft $S_2$
- Max intensity Sternal Edge
- $L \ R \ 40 \ (Cl_{95} \ 6.6 - 240)$

Differential Diagnosis of Left Sternal Border Systolic Murmurs

Tricuspid Insufficiency: Most Likely
Large CV Wave – LR 10.9 for Mod or Severe TR

VSD: If Long and Harsh, Suspect; Maybe a Wide Fixed Split $S_2$

ASD: Some Times

HOCM – Also Hear Aortic Murmur (Usually Not Radiate; if Stand Patient Up: Increased Intensity of Murmur with HOCM:
Sensitivity, specificity, LR for HOCM: 95%, 84%, 6.0

See McGee
Mitral Stenosis

Focal Area Over Apex
(Unlike MR) – Slowly “inch”
Stethoscope Across Apex

Left Lateral Decubitus Position

Diastolic “Rumble” with
Sense of Distance from Chest

Consider if Any Unclear Cause of Dyspnea
So Far... If Normal Heart

PMI – Dime Sized, 5th Left ICS Mid Clavicular Line

Rate – A Number

Rhythm – Regular

$S_1$ – Constant Intensity

$S_2$ – Physiological split

$S_3/S_4$

Murmurs
The Funduscopic Exam: What Internists Could Learn

Glaucoma

Increased Intracranial Pressure

Hypertension

Atherosclerosis and Left Ventricular Hypertrophy

Diabetes Mellitus

Retinal Infiltrates:

Red, White, Brown
Funduscopic Examination for the Internist

Dilating the Pupil

Contraindications –

1. Known narrow angles glaucoma – ask!
2. Possible neurological disease – you are watching pupils
3. Lens implants – ask the ophthalmologist first!
4. Anterior uveitis

Preferred mydriatic – 0.5% or 1% tropicamide (mydriacil ophthalmic) 2 drops

Repeat in 20 Minutes
Approach

1. Wash hands first!

2. With the patient facing the ceiling, cup the lower palpebral sac and drop two drops from a distance of 1 inch above the globe.

3. Have the patient close the eye and allow him/her to blot lightly below the eye any fluid on the face.

4. Place a note at the head of the bed and place a note in the medical record.
Normal fundus

Vessels emerge from nasal side of disc. Arteries are narrower than veins.
I- Intraocular Pressure and risk for Glaucoma

• Normal cup to disc ratio is .35; a large ratio of >.70 suggests glaucoma.

• In glaucoma you may also see nasalization of blood vessels, pallor of the disc, vertical ovality of the cup, and splinter hemorrhages on the disc.
Increased cupping
Cup:disc ratio > 1/3
= Glaucoma
II- Increased Intracranial Pressure and risk of Brain Herniation

- Loss of sharpness of the disc edges (more predictive if nasal)

- Presence of normal venous pulses means normal pressure; if absent unilaterally, suggests increased pressure. 20% of healthy people have absent venous pulses.

- Loss of normally present venous pulses in more sensitive than blurred disc edges for brain edema
Papilloedema

- Blurred disc margin
- Engorged, tortuous veins
- Congested, pink disc
- Disc swollen / raised
Spontaneous Venous Pulses
Optic Disc – Spontaneous Venous Pulsations
III- Atherosclerosis

- A-V nicking look at vessels > 2 dd from the disc and there must be not just tapering but absolute length of the vein not seen near the artery.

**Note:** Hypertension accelerates these changes, and many of these patients will also have hypertension. It is said that 96% of patients with the above strict criteria for nicking and have hypertension have cardiomegaly. (LVH).

- Copper and silver wiring of arterioles
- Hollenhorst plaque – golden yellow glistering plaque, a cholesterol embolus. A true sign of atherosclerosis. Listen to the carotids!

Sapira
Emboli and Infarcts

Small fleck a ‘Hollenhorst’ plaque caused from platelet/fibrin/cholesterol embolus. Resulting in an infarct (gray area above and right of the plaque).
“Dot” & “Blot” Hemorrhages
Roth Spot

Pale-centered hemorrhage. Caused by several conditions, not only bacterial endocarditis.
The Eye Exam

Internists Should be Able to Identify

- Increase Intracranial Pressure
  1) Loss of Venous Pulses
  2) Blurred Discs
- Glaucoma
  C:D Ratio > 0.7
- Early Diabetes Mellitus
  Dot/Blot Hemorrhages
- Hypertension
  A:V Ratio 1:2, 1:3...
  Associated Atherosclerotic changes: Silver/Cooper Wiring
  Hollenhorst Plaques. If AV Nicking: LVH!
- Retinal Hemorrhages, Exudates, Brown/Black exudates
Accurate Physical Diagnosis

Informative

Moves Patients to a Higher Prevalence for Specific Illnesses → Increasing Predictive Value of Subsequent Testing

Critical Part of the Doctor – Patient Relationship

Fun!