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Case Presentations
HYPERTENSION
Selection of Antihypertensive Therapy with Nutrients and Drugs

- Plasma Renin Activity (PRA), Aldosterone and ARR (Aldosterone Renin Ratio)
- Nutritional depletion evaluation
- Demographics
- Subsets of Hypertension: Individualized Rx
- Genetic phenotype (SNP)
  1. Those that predispose an individual to hypertension in some or all conditions and increase CV risk
  2. Those that predict response to a specific nutrition, drug or nutrient.
SELECTION OF ANTI-HYPERTENSIVE TREATMENT BASED ON BLOOD PRESSURE STRATIFICATION USING RENIN PROFILING PLASMA RENIN ACTIVITY (PRA) - LARAGH METHOD

- **Low Renin Hypertension (LRH):** Increased Intravascular Volume (Volume dependent)  PRA < 0.65 ng/ml/hour : 30% of patients

- **High Renin Hypertension (HRH):** Decreased Intravascular Volume : PRA > 0.65 ng/ml/hour  70% of patients
- **Very high Renin :** Volume Depleted:  PRA> 6.5 ng/ml/hour
**ARR: Aldosterone Renin ratio** (pg/ml / ng/ml per hour)

ARR over 80 is LRH or primary hyperaldosteronism

ARR over 40 is probably LRH with a sensitivity and specificity of 100% and 92% for primary aldosteronism.

ARR less than 10 is HRH

ARR between 10 and 40: not sure

Higher ARR is associated with development of CKD (ARR 66 vs 56) (JOH 2012)
Pathophysiology of Low Renin Hypertension (LRH)

- Reduced plasma renin activity-PRA
- Inappropriately increased serum Aldosterone level that cannot be suppressed with salt loading. Functional derangement in Aldosterone secretion.
- Low serum ionized calcium
- Increased serum PTH: Secondary hyperparathyroidism -SHPT
- Increased intracellular calcium especially mitochondria and cardiac myocytes and arteries. Increased oxidative stress and cardiac fibrosis
- Increased intravascular volume: “Wet Hypertension”
- Association between calcium and sodium metabolism
- More common in Blacks
- Commonly have hypomagnesemia, low zinc, low ionized calcium and low vitamin D.
- Treatment responsive to calcium, zinc, Vitamin D, CCB, diuretics and serum aldosterone receptor antagonists- SARA.
Pathophysiology of High Renin Hypertension (HRH)

- Elevated PRA
- Normal to low serum aldosterone levels
- Decreased intravascular volume
- Increased risk of CVD, MI, CVA compared to LRH
- Treatment responsive to ACEI, ARB, RI, BB and CAA. Also to nutraceuticals with similar mechanisms of actions as these drugs.
ARR is associated with insulin resistance and higher BP in Blacks

JASH 2012;6:56-65

- An increase in the ARR in blacks is associated with insulin resistance, metabolic syndrome, hypertriglyceridemia, microalbuminuria and higher blood pressure
- The highest tertile with ARR over 2.79 had the most abnormalities
- Insufficiently suppressed aldosterone in the presence of increased sodium intake
Measurement of PRA and Serum Aldosterone

- Random ambulatory serum levels of plasma renin activity (PRA) and serum aldosterone
- Most accurate in drug naïve patients
- Does not require alterations in patient position, time of day, sodium intake etc.
- Levels will be altered by concomitant anti-hypertensive medications which requires more sophisticated interpretation.
Treatment Selection in Drug Naïve Patients

- **Low Renin Hypertension (LRH):** Volume Drugs and Nutraceuticals: Calcium Channel Blockers (CCB), Diuretics, Serum Aldosterone receptor antagonists like Spironolactone and Epleronone (SARA), alpha blockers

- **High Renin Hypertension (HRH):** RAS or Renin Drugs and Nutraceuticals: Angiotensin Converting Enzyme Inhibitors (ACEI), Angiotensin Receptor Blockers (ARB), Direct Renin Inhibitors (DRI), Beta Blockers (BB), Central Alpha Agonists (CAA)
NUTRIENT TESTING

1. Determine nutrient deficiencies that contribute to the hypertension and vascular disease. Recommend: Micronutrient analysis (MNT) measures lymphocyte intracellular nutrient analysis for previous 6 months.

2. Replace nutrient deficiencies and re-evaluate at 3 to 4 months

3. Initiate therapeutic nutritional program

4. Initiate therapeutic nutritional supplement program with vitamins, antioxidants, minerals and nutraceuticals. May take up to 6 months to achieve maximal effect compared to drug therapy, but long-term BP reductions may be very similar.
1. **ACE I/D (DD Allele):** HBP, LVH, CRF, MAU, nephroangiogenesis, carotid IMT, MI, and CHD.
2. **COMT** (Catecholamines, CHD, MI, HBP, ASA, and vitamin E responses)
3. **MTHFR:** Methylation (1298C and 677T): hypertension, CHD, MI, CVA, thrombosis, homocysteine, ED.
4. **CYP 1A2:** Caffeine, HBP, MI, aortic stiffness, PWV, AI, tachycardia, arrhythmias, vascular inflammation, catecholamines.
5. **Corin:** Hypertension, CHF, volume overload, sodium sensitive, CVD, CRF, pre-clampsia, ANP, and BNP.
1. **GSHPx**: CHD, MI, hypertension, LVH, CHF, Glutathione, ALA 6 alleles, selenium.

2. **ADR B2**: HBP, PRA, inflammation and DASH diet with ACEI, ARB or DRI.

3. **CYP 4 A11**: Hypertension, ENaC and sodium, volume overload, CHD and Amiloride).

4. **CYP 4F 2**: Hypertension, ENaC and sodium, volume overload, CHD and Amiloride).

5. **AGTR1 ( ATR1AA)**: HBP, ARBs and potassium.

6. **NOS 3**: Nitric oxide, hypertension, MI, CHD, CVA, thrombosis, ED, oxidative stress, inflammation.
Natural Anti-hypertensive Compounds Categorized by Anti-hypertensive Class

Houston MC. Handbook of Hypertension Wiley Blackwell Oxford UK 2009
Houston MC. What Your Doctor May Not Tell You About Hypertension 2003

- **Diuretics**
  - Vitamin B-6 (Pyridoxine)
  - Taurine (diuretic, lower aldosterone)
  - Celery
  - GLA
  - Vitamin C (Ascorbic Acid)
  - K+
  - High Gamma/Delta tocopherols and tocotrienols

- Mg++
- Ca++
- Protein
- Fiber
- Coenzyme Q-10
- L-Carnitine
- Hawthorne Berry
Natural Anti-hypertensive Compounds Categorized by Anti-hypertensive Class

- Beta Blockers (BB)
  - Hawthorne Berry
- Central Alpha Agonists (CAA) (Reduced SNS Activity)
  - Taurine
  - K$^+$
  - Zinc
  - Na$^+$ restriction
  - Protein
  - Fiber
  - Vitamin C
  - Vitamin B-6
  - Coenzyme Q-10

Houston MC. Handbook of Hypertension Wiley Blackwell Oxford UK 2009
Houston MC. What Your Doctor May Not Tell You About Hypertension 2003
Direct Vasodilators

- Omega-3 FA
- MUFA (Omega-9 FA)
- K+
- Mg++
- Melatonin
- Soy
- Fiber
- Garlic
- Flavonoids
- Vitamin D and E
- Vitamin C
Nutrient and Nutraceutical with Calcium Channel Blocking (CCB) Activity

Houston MC. Handbook of Hypertension Wiley Blackwell Oxford UK 2009
Houston MC. What Your Doctor May Not Tell You About Hypertension 2003

- Alpha Lipoic Acid (ALA)
- Magnesium (Mg++)
- Vitamin B-6 (Pyridoxine)
- Vitamin C
- Vitamin E: high gamma/delta E with alpha tocopherol, (↑ cytosolic Mg+ + with ↓ Ca++), also diuretic
- N-Acetyl Cysteine (NAC)
- Hawthorne
- Celery
- Omega-3 fatty acids (EPA + DHA)
- Calcium
- Garlic
- Taurine
- EVOO
- Olive leaf extract
Angiotensin Converting Enzyme Inhibitors (ACEI)

- Garlic
- Wakame Seaweed
- Tuna protein/muscle
- Sardine protein/muscle
- Hawthorne Berry
- Bonito Fish (dried)
- Pycnogenol
- Casein
- Hydrolyzed Whey Protein
- Sour Milk and Milk peptides
- Gelatin
- EVOO
- Sake
- Omega-3 FA
- Chicken Egg Yolks
Natural Anti-hypertensive Compounds Categorized by Anti-hypertensive Class: ARB

Houston MC. Handbook of Hypertension Wiley Blackwell Oxford UK 2009
Houston MC. What Your Doctor May Not Tell You About Hypertension 2003

Angiotensin Receptor Blockers (ARB’s)

- Potassium (K+)
- Taurine
- Resveratrol
- Fiber
- Garlic
- Vitamin C
- Vitamin D
- Vitamin B-6 (Pyridoxine)
- Co Enzyme Q-10
- Celery
- Gamma Linolenic Acid
Take Home Points for Hypertension

- Always get a 24 hour ABM. A routine office BP is not adequate to determine treatment or the time of administration of drugs.
- Always measure endothelial function to predict risk for future CVD and assess function of blood vessel as well as BP.
- Measure Plasma Renin Activity (PRA), Aldosterone and ARR (Aldosterone Renin Ratio)
- Measure Nutritional depletions with micronutrient (MNT) evaluation evaluation
- Evaluate the Demographics
- Subsets of Hypertension: Precision and Individualized Rx
- Measure BP Genetic phenotype (SNPs)
  1. Those that predispose an individual to hypertension in some or all conditions and relate to CV target organ damage
  2. Those that predict response to a nutritional programs, drug or nutrient.

Treatment with DASH 2 diet, nutritional supplements and drugs (integrative therapy) based on level of BP, CVD target organ damage, concomitant diseases and the above test results.
CASE 1  HYPERTENSION CASE
Hypertension Case

Learning Objectives

1. Understand the pathogenesis of hypertension and the role of vascular biology, three finite vascular responses (inflammation, oxidative stress and vascular immune function), plasma renin activity (PRA) and aldosterone to select optimal integrative anti-hypertensive therapy to reduce blood pressure and decrease cardiovascular disease.

2. Review diagnostic testing for hypertension: 24 hour ambulatory BP (ABM), endothelial function and arterial compliance testing.

3. Be able to prioritize and personalize the most important nutritional, nutraceutical supplements and lifestyle treatments for hypertension.

4. Apply micronutrient testing in the treatment of hypertension.

5. Review briefly the optimal drug therapy for hypertension and combination use with nutraceutical supplements.
Case 1

- 55 year old black female with new onset hypertension
- Blood pressure 148/94 mm Hg in office with average blood pressure readings x 3
- 24 hour ambulatory blood pressure monitor: Mean blood pressure 146/92 mm Hg, BP load 52%, AM surges, non dipper.
- FH negative for hypertension and CHD. Non smoker, no ETOH, no caffeine.
- PE: Grade 1 KW retinal hypertensive changes, 2/6 systolic murmur. Normal weight, body fat 28% with visceral fat 32%.
- Lab normal except for microalbuminuria-MAU of 66, FBS 104 mg/dL, insulin level 22 and TG 298 mg/dL and HDL 42 mg/dL.
- EKG shows mild left ventricular hypertrophy
- PRA 0.20 ng/ml/hr
- Plasma Aldosterone: 10
- ARR (Aldo/renin ratio): 50
- Micronutrient deficiencies: GLA (gamma linoleic acid), Vitamin D (25 ng/ml), Zinc, Magnesium and Lipoic acid
- Low ionized calcium and borderline elevated parathyroid hormone level (PTH)
Questions

• Do you want any more information about history, physical or labs?
• Do you want any more lab tests at this time?
• What are the present suspected problems/ diagnosis?
• What type of hypertension is present?
• Why does she have hypertension?
• What do you know about this type of hypertension?
• What is your treatment plan?
**Endo-PAT2000**

**Patient Information**
- Name:
- Systolic BP: 110 mm Hg
- Gender:
- Diastolic BP: 78 mm Hg
- Height:
- BMI: 32.4
- Weight: 190 lb

**Study Information**
- Test Duration: 00:11:40
- PATographer: NK
- Recording Ver: 3.3.2
- Analysis Ver: 3.3.2
- Occ. Borders: Automated

**PAT Signals**

**Occluded Arm**

**Control Arm**

**Study Results**
- RHI: 1.44
- Endothelial Dysfunction
- Heart Rate: 85 bpm

**Recommendations**
- Physician’s Name: 
- Signature: [Signature]
Case 1

- Patient has low renin hypertension (LRH)
- Endothelial dysfunction at 1.44. Normal is > 1.67
- ECHO: mild LVH with mild mitral insufficiency
- Carotid duplex with mild bilateral increase in ICA IMT.
- Her genetic profile (SNPs) for hypertension is negative
- Use nutraceuticals and/or drugs that work in LRH and improve the 24 hour ABM.
- Replete nutrient deficiencies: GLA (gamma linoleic acid), Vitamin D, Zinc, Magnesium and Lipoic acid
- Start high dose treatment with nutritional supplements that improve LRH: GLA (gamma linoleic acid), Vitamin D, Zinc, Magnesium and Lipoic acid or others
- Treat the insulin resistance and hypertriglyceridemia
- Improve MAU
- Reduce the LVH
- Reduce the retinal hypertensive changes
- Decrease the mitral insufficiency
Case 1 Treatment

- Dash 2 Diet with 50 grams of refined carbohydrates.
- Combined aerobic (IT 20 minutes) and resistance exercise (40 minutes) 6 days per week at 60 minutes per session per the ABCT exercise program
- GLA 500 mg twice per day
- Vitamin D 4000 IU per day
- Zinc 50 mg per day
- R-Lipoic acid 100 mg per day with Biotin 2 mg per day
- Magnesium Chelates at 500 mg twice per day
- Omega 3 fatty acids 1.5 grams bid (DHA and EPA)
- Melatonin SR 3 mg at night
Treatment Results

6 weeks: BP 126/84 mm Hg

4 months

BP 118/78 mm Hg, 24 hour ABM is normal with mean BP 116/76 mm Hg, BP load < 5 %, no AM surges, normal dipping pattern.

Body fat 24 % with visceral fat 26 %

Mitral insufficiency murmur gone

Retinal changes improved

FBS 88 mg/dL, insulin level 9

TG 110 mg/dL, HDL 52 mg/dL

MAU 34

Vitamin D 62 ng/dl

Ionized calcium and PTH level are normal

MNT normal for micronutrient deficiencies.

Endopat 1.96 (normal is over 2.1)

LVH on EKG unchanged
DYSLIPIDEMIA
Lipoprotein Particles

Apolipoprotein A1 (HDL) or B100 (LDL)

Triglyceride

Cholesterol Ester

Unesterified Cholesterol

Phospholipid
Lipoprotein Sub-Classes

<table>
<thead>
<tr>
<th>Diameter (nm)</th>
<th>Density (g/ml)</th>
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<tbody>
<tr>
<td>5</td>
<td>1.20</td>
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<tr>
<td>10</td>
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<tr>
<td>20</td>
<td>1.06</td>
</tr>
<tr>
<td>40</td>
<td>1.02</td>
</tr>
<tr>
<td>60</td>
<td>1.006</td>
</tr>
<tr>
<td>80</td>
<td>0.95</td>
</tr>
</tbody>
</table>

Chylomicron
VLDL
Remnants
VLDL
Remnants
LDL
IDL
Chylomicron
Remnants

Lipoprotein Sub-Classes:
- Chylomicron
- VLDL
- IDL
- LDL
- HDL
- Lp(a)
### Lipid, Lipoproteins, and Apolipoproteins Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Optimal</th>
<th>Borderline</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apo A1</td>
<td>1.2</td>
<td>1.0 - 1.5</td>
<td>&gt;2.0</td>
</tr>
<tr>
<td>LDL-C</td>
<td>&lt;100</td>
<td>100 - 130</td>
<td>&gt;130</td>
</tr>
<tr>
<td>Apo A1</td>
<td>&gt;1.5</td>
<td>1.0 - 1.5</td>
<td>&gt;2.0</td>
</tr>
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</table>

### Inflammation Tests

<table>
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<tbody>
<tr>
<td>Lp(a)</td>
<td>&lt;200</td>
<td>200 - 300</td>
<td>&gt;300</td>
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</table>

### Diabetes Tests

<table>
<thead>
<tr>
<th>Test</th>
<th>Optimal</th>
<th>Borderline</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c</td>
<td>&lt;5.7</td>
<td>5.7 - 6.5</td>
<td>&gt;6.5</td>
</tr>
</tbody>
</table>

### Framingham Risk Score

- **Risk Category:** Low
- **10-Year Risk of Heart Disease:** 2%
- **10-Year Risk of Stroke:** 1%

### Non-fasting Lipid Panel

<table>
<thead>
<tr>
<th>Test</th>
<th>Optimal</th>
<th>Borderline</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>HDL-C</td>
<td>&gt;40</td>
<td>40 - 60</td>
<td>&gt;60</td>
</tr>
<tr>
<td>LDL-C</td>
<td>&lt;100</td>
<td>100 - 130</td>
<td>&gt;130</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>&lt;150</td>
<td>150 - 200</td>
<td>&gt;200</td>
</tr>
</tbody>
</table>

### Lipid/HDL Map

- **Optimal:** LDL-C < 100 mg/dL
- **Borderline:** LDL-C 100 - 129 mg/dL
- **High Risk:** LDL-C > 129 mg/dL

### C-reactive Protein (CRP)

- **Optimal:** <0.3 mg/L
- **Borderline:** 0.3 - 1.0 mg/L
- **High Risk:** >1.0 mg/L

### Interpreted:

This lipid profile is within normal limits and suggests a low risk of cardiovascular disease (CVD).

### Cholesterol Balance Score

- **Optimal:** 0.8
- **Borderline:** 0.9 - 1.0
- **Over Absorber:** >1.0
- **Over Producer:** <0.8

### Calculations:

- **Saturated Fat:** 15% of total calories
- **Unsaturated Fat:** 20% of total calories
- **Cholesterol:** <300 mg/day
- **Sodium:** <2300 mg/day
- **Fiber:** 25 g/day

### Diet Suggestions:

- Increase intake of fruits, vegetables, and whole grains.
- Reduce intake of red meat and processed meats.
- Limit intake of saturated and trans fats.
- Increase physical activity to at least 150 minutes per week.

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**Note:**

- Balancing dietary choices and regular physical activity can help maintain a healthy lifestyle.
- Consult a healthcare provider for personalized advice.
Impact of LDL Size Differences

At the same LDL cholesterol

100 mg/dL

100 mg/dL

Large LDL

Small LDL

Up to 70% More Particles

Cholesterol Balance
Nutritional Supplement Treatment for Dyslipidemia

Final Recommendations

- Red yeast rice 2400 to 4800 mg at night with food
- Plant sterols 2.5 grams per day.
- Berberine 500 mg per day to twice per day.
- Niacin (nicotinic acid B3) 500 to 3000 mg per day as tolerated pretreated with quercetin, apples, ASA. Take with food and avoid alcohol. Never interrupt therapy.
- Omega-3 fatty acids with EPA/DHA at 3/2 ratio 4 grams/day with GLA at 50% of total EPA and GLA and gamma/delta tocopherol.
- Gamma delta tocotrienols 200 mg hs.
- Aged garlic- Kyolic standardized 600 mg twice per day.
- Sesame 40 grams per day
- Phosphstidyl serine 300-600 mg bid
Nutritional Supplement Treatment for Dyslipidemia

Final Recommendations

- Pantethine 450 mg BID
- MUFA 20 to 40 grams per day (EVOO 4 tablespoons per day)
- Lycopene 20 mg per day and astaxanthin 15 mg/day
- Luteolin 10 per day
- Trans resveratrol 250 mg per day
- NAC 500 mg twice per day
- Carnosine 500 mg twice per day
- Citrus bergamot 1000 mg per day
- Quercetin 500 mg twice per day
- Probiotics standardized 15 to 50 billion organisms BID
- Curcumin 500-1000 mg twice per day
- EGCG 500-1000 mg BID or 60-100 ounces of green tea per day
- Pomegranate one cup of seeds/day or 6 ounces of juice per day.
Dyslipidemia Take Home Points

- Routine lipid testing will not identify dyslipidemia and CHD risk accurately
- Advanced lipid testing should now be routine in all patients
- LDL-P drives the risk for CHD > APO B > Non HDL C > LDL C
- Lp(a) is missed on routine lipid testing and is the HIDDEN RISK related to lipids for CHD, MI and thrombosis. “The CHD GAP”
- LDL-P is best treated with omega 3 fatty acids, niacin, berberine and red yeast rice. Statins are only 30-50% effective
- Small LDL is best treated with omega 3 fatty acids and niacin
- Lp(a) is best reduced with niacin and NAC
- Lp(a) attachment to the vascular wall is achieved with the Linus Pauling protocol with vitamin C, proline and lysine.
- High TG are best treated with omega 3 FA, niacin and low refined CHO diet
- Low HDL, small HDL and low HDL-P is best treated with niacin, pantethine, omega 3 FA and pomegranate seeds or juice.
- Remnant particles and large VLDL increase CHD risk.
- The most common lipid profile with insulin resistance is high TG with large VLDL, low HDL, high LDL-P with small LDL
Key References and Recommended Reading

CASE 2  DYSLIPIDEMIA CASE
Learning Objectives
Dyslipidemia Cases

- Review the underlying causes and mechanisms of dyslipidemia and dyslipidemia-induced cardiovascular disease.

- Understand and apply advanced lipid testing (lipid particle number and size and HDL functionality and quality) for the diagnosis and treatment of dyslipidemia in clinical practice.

- Prioritize effective methods to promote vascular repair, reduce vascular damage and define the interrelationships of the cardiovascular system, gastrointestinal tract and microbiome.

- Evaluate nutrition and nutritional supplements to treat dyslipidemia.

- Review 45 new mechanisms involved in dyslipidemia-induced cardiovascular disease and review the integrative therapies.
CASE 2

- 38 year old male in for physical exam
- Family history is positive for CHD early in life (age below 50 years in both parents)
- Normal weight, non smoker, excellent diet and exercise program
- History and PE are normal
- All labs are normal including a routine lipid profile: TC 124, LDL 70, HDL 41, TG 70 mg/dL
- Expanded lipid profile done (see results)
Low Fat, Vegetarian Diet (10 Percent Calories from Fat)

Pravastatin 40 mg qhs
Niacin 1000 mg q day

SUBCLASS LEVELS

Lipoprotein subclass levels (mg/dL) are given in parentheses above each bar. The height of the bar gives the percent of the population with equal or lower levels.

VLDL Subclasses (mg/dL Triglyceride)
- Large VLDL (V5+V6)
- Intermed. VLDL (V3+V4)
- Small VLDL (V1+V2)

LDL Subclasses (mg/dL Cholesterol)
- Large LDL (L3)
- Intermed. LDL (L2)
- Small LDL (L1)

HDL Subclasses (mg/dL Cholesterol)
- Large HDL (H4+H5)
- Intermed. HDL (H3)
- Small HDL (H1+H2)

NMR-DERIVED LIPID VALUES

Current NCEP Risk Categories

<table>
<thead>
<tr>
<th>Lipid</th>
<th>mg/dL</th>
<th>Desirable</th>
<th>Borderline-High</th>
<th>High</th>
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<tr>
<td>Total Cholesterol</td>
<td>124</td>
<td>less than 200</td>
<td>200 - 239</td>
<td>240 or greater</td>
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<tr>
<td>LDL Cholesterol</td>
<td>70</td>
<td>under 100</td>
<td>100 - 129</td>
<td>Borderline-high</td>
</tr>
<tr>
<td>HDL Cholesterol</td>
<td>41</td>
<td>60 or greater</td>
<td>59 - 40</td>
<td>Positive Risk Factor</td>
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<tr>
<td>Triglycerides</td>
<td>70</td>
<td>less than 150</td>
<td>150 - 199</td>
<td>Borderline-high</td>
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* Goal for patients with CHD or CHD risk equivalents
### LIPOPROTEIN PANEL

#### Coronary Heart Disease (CHD) Risk Categories

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<tr>
<th></th>
<th>Optimal*</th>
<th>Near optimal</th>
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<th>Very high</th>
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<tr>
<td>LDL Particle Number</td>
<td>under 1100</td>
<td>1100 - 1399</td>
<td>1400 - 1799</td>
<td>1800 - 2100</td>
<td>over 2100</td>
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*Goal for patients with CHD or CHD risk equivalents

<table>
<thead>
<tr>
<th></th>
<th>Lower-Risk</th>
<th>Intermediate</th>
<th>Positive Risk Factor</th>
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<tbody>
<tr>
<td>Pattern A (large LDL)</td>
<td>22.0 - 20.6</td>
<td>greater than 30</td>
<td>less than 11</td>
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<tr>
<td>Pattern B (small LDL)</td>
<td>20.5 - 19.0</td>
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<td>Large HDL</td>
<td>less than 7</td>
<td>7 - 27</td>
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<td>(cholesterol)</td>
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<th>Lower-Risk</th>
<th>Intermediate</th>
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<tr>
<td>Large VLDL</td>
<td>less than 7</td>
<td>7 - 27</td>
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<td>(triglyceride)</td>
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### RISK ASSESSMENT PANEL

Elevated LDL particle number and the lipoprotein traits of the metabolic syndrome affect CHD risk interactively. Risk is highest when both are present.

- Elevated LDL Particle Number > 1400 nmol/L
- Lipoprotein Traits of the Metabolic Syndrome*
  - Small LDL Pattern B (≤ 20.5 nm)
  - Reduced Large HDL (<11 mg/dL)
  - Elevated Large VLDL (>27 mg/dL)

*Other identifiers of the metabolic syndrome include abdominal obesity, elevated blood pressure, and fasting glucose ≥110 mg/dL.
CASE 2

- Lp(a) is 120 (normal is < 30)
- Hs CRP is 1.5 (normal <1.0)
- FBS 79 mg/dL
Problems

- What are the problems in this case?
- What is the role of HS CRP?
- What is the role of Lp(a)
- Describe the advanced lipid profile
- Is this patient at risk for CHD?
- Would you treat? If so, what treatment would you advise and why?
1. This patient needs no treatment and is at low risk for cardiovascular disease

2. This patient should be treated immediately with high dose statin

3. This patient should be treated with appropriate non drug treatment such as .....What ?

4. This patient should be treated with a combination of fibrates and statins
CASE 2: Treatment and Results at 2 months

- Niacin B3 1000 mg BID
- NAC 1000 mg bid
- Omega 3 FA 4 grams per day
- Vitamin C buffered 5000 mg per day
- Lysine 1000 mg per day
- Proline 500 mg per day
- Red Yeast Rice 800 mg hs
- Berberine 500 mg HS
- Pomegranate seeds 1/4 cup bid.

- LDL: 70 to 55 mg%
- LDL(p): 999 to 710
- LDL size: 20.3 to 22
- HDL: 41 to 50 mg%
- HDL 2 b: 16 to 38 mg%
- TG: 70 to 55 mg%
- Lp(a) from 120 to 70
Learning Objectives
CHD cases

Learn how to apply global cardiovascular risk scoring using the new expanded COSEHC and RASMUSSEN CHD scoring methods.

Review cardiovascular genomics and SNP’s, the top 5 CHD risk factors, the details of the correct analysis of each, the top 25 modifiable key CHD risk factors and the other 400 CHD risk factors, how to test and their interpretation.

Review, understand and integrate diagnostic, prevention and treatments for CHD, MI, Angina.

Review metabolic cardiology treatment for CHD and Angina.

Prioritize which laboratory and non invasive laboratory and cardiovascular tests should be evaluated in patients in the primary care setting and how to interpret and treat.

Review integrative medical treatments with nutrition, nutritional supplements, lifestyle and drugs for cardiovascular disease.
Mechanism Of Model

Oxidative Stress

Infinite Insults

Immune Dysfunction

Inflammation

Finite Responses
Cardiovascular Disease

Pattern Recognition & Toll-Like Receptor Activation

Inflammatory Cytokines

Oxidative Stress

Mitochondrial Immune dysfunction Failure

Myocyte & Vascular Cell Death

Decreased CV Function
Top 25 Modifiable CHD Risk Factors but over 400 CHD Risk Factors are now defined.
Houston MC. What Your Doctor May Not Tell You About Heart Disease 2012

- Hypertension (24 hour ABM)
- Dyslipidemia (Advanced Lipid Analysis)
- Hyperglycemia, metabolic syndrome, insulin resistance and Diabetes Mellitus
- Obesity
- Smoking
- Hyperuricemia
- Renal disease
- Elevated fibrinogen
- Elevated serum iron
- Trans fatty acids and refined carbohydrates
- Low dietary omega 3 fatty acids
- Low dietary potassium and magnesium with high sodium intake
- Inflammation: increased HSCRP
- Increased oxidative stress and decreased defense
- Increased Immune dysfunction
- Lack of sleep
- Lack of exercise
- Stress, anxiety and depression
- Homocysteinemia
- Subclinical hypothyroidism
- Hormonal imbalances in both genders
- Chronic clinical or subclinical infections
- Micronutrient deficiencies: numerous such as low vitamin D and K etc.
- Heavy metals
- Environmental pollutants
CARDIA X
Recommended Testing for Early Detection and Prevention of CHD
Genetic Testing

1. 9p21 (GG/CC): CHD, MI, ASCVD, DM, IR AAA, thrombosis, plaque rupture, inflammation and intracranial aneurysms.
2. 6p21.4: CHD, MI, DVT).
3. 4q25: Atrial Fibrillation, long QT and PR intervals.
4. ACE I/D (DD Allele): HBP, LVH, CRF, MAU, nephroangiogenesis, carotid IMT, MI and CHD.
5. COMT (Catecholamines, CHD, MI, HBP, ASA and vitamin E responses)
6. 1q25 (GLUL): CHD in DM, enterocytes and ED.
7. APO E: Dyslipidemia, CHD, MI, nitric oxide, statin response.
8. MTHFR: Methylation (1298C and 677T): hypertension, CHD, MI, CVA, thrombosis, homocysteine, ED.
9. CYP 1A2: Caffeine, HBP, MI, aortic stiffness, PWV, AI, tachycardia, arrhythmias, vascular inflammation, catecholamines.
10. Corin: Hypertension, CHF, volume overload, sodium sensitive, CVD, CRF, preclampsia), ANP and BNP.
CARDIA X
Recommended Testing for Early Detection and Prevention of CHD Genetic Testing

1. **GSHPx**: CHD, MI, hypertension, LVH, CHF, Glutathione, ALA 6 alleles, selenium.
2. **ADR B2**: HBP, PRA, inflammation and DASH diet with ACEI, ARB or DRI.
3. **APO A1**: Lipids, HDL, CHD, MI obesity.
4. **APO A2**: Lipids, HDL, CHD, MI, obesity.
5. **APC C 3**: Dyslipidemia, CHD, MI, dysfunctional HDL, inflammation, DM.
7. **CYP 4F 2**: Hypertension, ENaC and sodium, volume overload, CHD and Amiloride).
8. **AGTR1 (ATR1AA)**: HBP, ARBs and potassium.
9. **NOS 3**: Nitric oxide, hypertension, MI, CHD, CVA, thrombosis, ED, oxidative stress, inflammation.
10. **SCARB1**: Lipids, dysfunctional HDL with high HDL, CHD, MI.
Risk Factors: Men = 17, Women = 12
- Being male
- Age (years)
  Extra for cigarette smoking
- Systolic blood pressure (mm Hg)
- Total cholesterol conc. (mg / dL)
- LDL cholesterol (mg / dL)
- HDL cholesterol (mg / dL)
- Triglyceride (mg / dL)
- Height (inches)
- Creatinine conc. (mg / dL)
- Homocysteine (μmol / L)
- Prior MI
- Family history of MI pre- 60
- Prior Stroke
- LVH
- Diabetes
- Non-Diabetic, FBS (mg / dL)
## COSHEC ABSOLUTE RISK ANALYSIS FOR DEATH FROM CHD IN 5 YEARS

<table>
<thead>
<tr>
<th>Risk Score</th>
<th>% dying from cardiovascular disease in 5 years</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>0.04</td>
</tr>
<tr>
<td>5</td>
<td>0.07</td>
</tr>
<tr>
<td>10</td>
<td>0.11</td>
</tr>
<tr>
<td>15</td>
<td>0.19</td>
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<tr>
<td>20</td>
<td>0.31</td>
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<tr>
<td>25</td>
<td>0.51</td>
</tr>
<tr>
<td>30</td>
<td>0.84</td>
</tr>
<tr>
<td>35</td>
<td>1.4</td>
</tr>
<tr>
<td><strong>40</strong></td>
<td><strong>2.3</strong></td>
</tr>
<tr>
<td>45</td>
<td>3.7</td>
</tr>
<tr>
<td>50</td>
<td>6.1</td>
</tr>
<tr>
<td>55</td>
<td>9.8</td>
</tr>
<tr>
<td>60</td>
<td>15.6</td>
</tr>
<tr>
<td>65</td>
<td>24.5</td>
</tr>
</tbody>
</table>
COSHEC ABSOLUTE RISK CALCULATION

Houston MC et al Am J Medical Sciences 2005;329:276-291 and 292-305

- **VERY LOW RISK:** SCORE 0-10
- **LOW RISK:** SCORE 10-20
- **MODERATE RISK** SCORE 20-30
- **MODERATE/HIGH** SCORE 30-40
- **HIGH RISK** SCORE 40-50
- **VERY HIGH RISK** SCORE > 50

**NOTE TRIPLE RISK WITHIN EACH 10 POINT RISK SCORE**
Disease score 0-2: no CV events in 6 yrs
Disease score 3-5: 5% CV events in 6 yrs
Disease score over 6: 15% CV events in 6 yrs
Superior to Framingham risk score
Variables measured: CAPWA, BP at rest and exercise, LV mass by ECHO, microalbuminuria, BNP, retinal score, Carotid IMT and US, EKG.
<table>
<thead>
<tr>
<th>Test</th>
<th>Normal</th>
<th>Borderline</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Score for each test</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Large artery elasticity</td>
<td>(age- and gender-dependent)</td>
<td>(age- and gender-dependent)</td>
<td></td>
</tr>
<tr>
<td>Small artery elasticity</td>
<td>(age- and gender-dependent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Resting BP (mm Hg)</td>
<td>SBP &lt;130 and DBP &lt;85</td>
<td>SBP 130–139 or DBP 85–89</td>
<td>SBP ≥140 or DBP ≥90</td>
</tr>
<tr>
<td>Treadmill exercise BP (mm Hg)</td>
<td>SBP increase &lt;30 and SBP ≤169</td>
<td>SBP increase 30–39 or SBP 170–179</td>
<td>SBP increase ≥40 or SBP ≥180</td>
</tr>
<tr>
<td>Optic fundus photography</td>
<td>A/V ratio &gt;3:5</td>
<td>A/V ratio ≤3:5 or mild A/V crossing changes</td>
<td>A/V ratio ≤1.2 or A/V nicking</td>
</tr>
<tr>
<td>retinal vasculature</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carotid IMT</td>
<td>(age- and gender-dependent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Microalbuminuria (mg/mmol)</td>
<td>≤0.6</td>
<td>0.61–0.99</td>
<td>≥1.00</td>
</tr>
<tr>
<td>Electrocardiogram</td>
<td>No abnormalities</td>
<td>Nonspecific abnormality</td>
<td>Diagnostic abnormality</td>
</tr>
<tr>
<td>LV ultrasound LVMI (g/m²)</td>
<td>&lt;120</td>
<td>120–129</td>
<td>≥130</td>
</tr>
<tr>
<td>NT-proBNP (pg/dl)</td>
<td>&lt;150</td>
<td>150–250</td>
<td>&gt;250</td>
</tr>
</tbody>
</table>
Kaplan-Meier curves of time morbid events during 6 years of follow-up in the three Rasmussen Disease Score (DS) Groups. The difference among the curves (P = .0000) is highly significant. Two events after 72 months are not depicted.

6814 participants in MESA study evaluating CAC, Carotid IMT, ABI, BFMD, HS-CRP and FH of CHD compared to the FRS.

Results in multivariable analysis:
- CAC: HR 2.60 (p<0.001): BEST (also best in Rotterdam study, Kavousi study and others)
- FH: HR 2.18 (p<0.001)
- HS CRP: HR 1.28 (p<0.05)
- Carotid IMT: HR 1.17 (p=.13)
- BFMD: HR .93 (p=.52)
- ABI: HR .79 (p=.01)
CHD: Extraluminal Disease: Glagov Principal

- Minimal to mild CHD: Lumen Normal, Mild extraluminal atheroma
- Moderate CHD: Lumen normal size, Mild extraluminal atheroma
- Severe CHD: Lumen Stenosis, Severe extraluminal and intraluminal atheroma

- 95 - 99% extraluminal and intraluminal atheroma
- 1 - 5% stenosis

- 68% of MI: < 50% Stenosis
- 14% of MI: Significant Stenosis
- 62% men 1st symptom of CHD is MI
- 46% women 1st symptom of CHD is MI

Nissen, S.
ISH, August 200
Angiographically Inapparent Atheroma

Take Away Messages

- Start early and aggressive detection, prevention and treatment for CHD with advanced biomarkers and non invasive CV testing with integrative approach.
- Significant CHD can present with atypical clinical symptoms without classic anginal chest pain.
- All risk factors have a progressive continuum of risk starting at BP of 120/80, LDL-P of 1000 of 700 depending on lab, FBS of 75, homocysteine of 5, HS CRP of 1.0 and TSH of 2.0 etc.
- There are 25 TOP CHD risk factors, but there are over 400 CHD risk factors.
- Measure the 3 finite responses of inflammation, oxidative stress and vascular immune function and treat.
- Risk scoring with COSHEC and RASMUSSEN, CV genetics with CARDIA X, gene expression with CORUS, Micronutrient testing, all the advanced labs and CV tests with good history and PE will provide more individualized and precision treatments to prevent CVD and CHD.
- Endothelial dysfunction is accurate predictor of future CHD.
CASE 3

CORONARY HEART DISEASE CASES
Case 3

- 51 year white male stock broker with high stress job
- Chief complaint: fatigue. First visit 2011
- PMH: dyslipidemia, dysglycemia, fatigue, stress, mild chronic diarrhea and OA of back and neck.
- Non smoker, no ETOH or caffeine
- Fast food diet with heavy refined carbs
- Sedentary
- FH positive for early CHD.
- PE: 6 feet, 190 pounds, BP 110/72 mm hg. Soft systolic murmur. Otherwise normal
Case 3

- Lab 2011: FBS 106 mg/dL, HbA1c 6.2, homocysteine 13, TC 275, LDL 160, HDL 70, TSH 3.8. HS CRP 3. Vitamin D 31 ng/ml, BNP 320 elevated, microalbuminuria 78 (elevated), creatinine 1.3. All other labs are normal.
- EKG with NSSTT wave changes and RBBB
- CXR normal
- TMT normal with RBBB but hypertensive response to exercise: BP 230/92 mm Hg
- CAC (coronary artery calcium score 115: LAD 103 (see report)
- CAPWA (computerized arterial pulse wave analysis): C1 AC 21.8, C2 AC 11.2 normal: (see report
- ECHO: mild mitral insufficiency, mild calcification of aortic valve
- Carotid duplex: LCCA and RCCA increased IMT with smooth soft homogeneous plaque with 30% obstruction bilaterally.
Case 3

- **Endopat**: 1.44 (normal > 2.1) : see report (endothelial dysfunction)
- **MTHFR**: heterozygote 1298 C
- **MNT**(micronutrient testing): low CoQ10 and magnesium
- **CoQ10 level**: 0.9 (low) (normal is 3.0)
- **Omega index**: 4.2 (normal 8.0)
- **Advanced Lipid testing**: LDL-P 1800 (normal <1000), dense LDL , HDL  65 with normal HDL map
- **MPO** (myeloperoxidase) 730 (normal < 495)
- **Negative heavy metals and toxin screen**
- **Negative infectious disease testing.**
PROCEDURE: CARDIAC CT FOR CORONARY ARTERY CALCIUM SCORING

TECHNIQUE: Multi-detector computed tomography of the heart was performed during suspended respiration, and without the administration of contrast material. Post-processing was performed on a workstation to measure the amount of coronary-vascular calcium. CPT 70557

HISTORY: Screening

COMPARISON: None

RESULTS: Thomas. No significant abnormalities identified in the lungs or mediastinum. Note that the CT examination is limited to the heart and the adjacent lung and mediastinum.

CALCIUM SCORING:
Left main coronary artery (LMCA): 1.6
Left anterior descending artery (LAD): 10.6
Circumflex artery: 0
Right coronary artery (RCA): 0

TOTAL AGATSTON SCORE: 115

The probability of a nonzero score in a person of the same age, sex, and race/ethnicity is 68 percent.

The above stated Agatston score is at percentile 76 for subjects of the same age, gender, and race/ethnicity who are free of clinical cardiovascular disease and treated diabetes. This indicates that 30 percent of individuals of the same age, sex, and race/ethnicity will have the same or higher score.

Calcium Score (CACS) Interpretations (Agatston Score):
0 points: No identifiable atherosclerotic plaques. Very low cardiovascular disease risk. Less than 5 percent chance of presence of coronary artery disease (CAD). A negative examination.
1-100: Minimal plaque burden. Significant CAD very unlikely.
11-499: Mild plaque burden. Likely mild or minimal coronary stenosis (obscurance).
500-699: Moderate plaque burden. Moderate non-occlusive CAD highly likely.
>700: Severe plaque burden. High likelihood (>90 percent) of at least one major coronary vessel with a significant stenosis (>50 percent diameter).

Note: This examination is not to be considered a substitute for a clinical examination by a physician. Coronary artery calcium scoring is intended to be a risk assessment test for coronary artery disease only, and the results of this examination should be taken into careful consideration by the patient's own physician in the context of other factors such as relevant history, physical examination, and other indicated or related investigations.
# HDI/PulseWave™ CR-2000

Research CardioVascular Profile Report

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Research Subject Value</th>
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<tbody>
<tr>
<td>Systolic Blood Pressure (mmHg)</td>
<td>65</td>
</tr>
<tr>
<td>Diastolic Blood Pressure (mmHg)</td>
<td>55</td>
</tr>
<tr>
<td>Mean Arterial Blood Pressure (mmHg)</td>
<td>64</td>
</tr>
<tr>
<td>Pulse Pressure (mmHg)</td>
<td>40</td>
</tr>
<tr>
<td>Pulse Rate (beats/min)</td>
<td>54</td>
</tr>
<tr>
<td>Estimated Cardiac Ejection Time (msec)</td>
<td>378</td>
</tr>
<tr>
<td>Estimated Stroke Volume (ml/beat)</td>
<td>112</td>
</tr>
<tr>
<td>Estimated Stroke Volume Index (ml/beat/m²)</td>
<td>54</td>
</tr>
<tr>
<td>Estimated Cardiac Output (L/min)</td>
<td>6.0</td>
</tr>
<tr>
<td>Estimated Cardiac Index (L/min/m²)</td>
<td>2.9</td>
</tr>
<tr>
<td>Large Artery Elasticity Index (ml/mmHg x 10)</td>
<td>21.8</td>
</tr>
<tr>
<td>Small Artery Elasticity Index (ml/mmHg x 100)</td>
<td>11.2</td>
</tr>
<tr>
<td>Systemic Vascular Resistance (dynes-sec-cm⁻²)</td>
<td>9000</td>
</tr>
<tr>
<td>Total Vascular Impedance (dynes-sec-cm⁻²)</td>
<td>105</td>
</tr>
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</table>

**Endo-PAT2000**

<table>
<thead>
<tr>
<th><strong>Patient Information</strong></th>
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<tbody>
<tr>
<td>ID:</td>
<td></td>
</tr>
<tr>
<td>Age:</td>
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<tr>
<td>Height:</td>
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<td>User Field 1:</td>
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<tr>
<td>Gender:</td>
<td></td>
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<tr>
<td>Weight:</td>
<td>190 lb</td>
</tr>
<tr>
<td>Systolic BP:</td>
<td>110 mm Hg</td>
</tr>
<tr>
<td>Diastolic BP:</td>
<td>70 mm Hg</td>
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<tr>
<td>BMI:</td>
<td>32.4</td>
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<table>
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<tr>
<th><strong>Study Information</strong></th>
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<tr>
<td>Test Duration:</td>
<td>09:11:49</td>
</tr>
<tr>
<td>PATographer:</td>
<td>NK</td>
</tr>
<tr>
<td>Recording Ver:</td>
<td>3.3.2</td>
</tr>
<tr>
<td>Analysis Ver:</td>
<td>3.3.2</td>
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<tr>
<td>Occ. Borders:</td>
<td>Automated</td>
</tr>
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</table>

**PAT Signals**

- **Occluded Arm**

- **Control Arm**

| Baseline (03:14) | Occlusion (05:20) | Dilatation (03:12) |

**Study Results**

- RHI: 1.44
- Heart Rate: 85 bpm

**Recommendations**

- **Physician's Name**: [Signature]
- **Recommendation**: [Handwritten]

[Graphs of PAT signals are shown, showing baseline, occlusion, and dilatation phases.]
Case 3  Genetic Testing

- 9p 21 homozygote
- ACE I/D polymorphism
- Apo E 4/4
- MTHFR : heterozygote 1298 C
Is he at risk for CHD?
What are his risk factors for CHD?
What treatment does he need?
Case 3

Treatment 2011

- Red Yeast Rice 1600mg at night with dinner
- Omega 3 FA 3 grams per day
- Methyl folate 1 mg and B vitamins
- Mediterranean diet with refined CHO <75 grams
- Supervised progressive ABCT exercise program
- Berberine 500 mg twice a day
- CO Q 10 at 100 mg bid
- Magnesium Malate Chelates one bid
- Niacin 500 mg bid
- Pomegranate seeds ¼ cup bid
- Curcumin 1000 mg bid
- Vitamin K2 MK 7 500 micrograms per day
- Beet root extract product chewable bid
- Vitamin D emulsion 4000 IU per day to level of 60-80 ng
- Armour thyroid 60 mg per day
Case 3 : 6 months later

- BP 108/68 mm hg, 185 pounds
- FBS 92, Hb A1c 5.7, homocysteine 6.8, LDL 106, HDL 68, TSH 1.8, Vitamin D 70, hs CRP 0.6. MPO 350 (normal). All else normal routine lab
- Endopat: 1.94 (normal > 2.1) (mild endothelial dysfunction)
- MNT: normal CoQ10 and Magnesium
- CoQ 10 level: 3.1 normal
- Omega index: 7.2 (normal 8.0)
- Advanced Lipid testing: LDL-P 1290 (normal <1000), HDL map normal. Increased RYR to 3200 mg hs.
Case 3

- **2014:** Mild increase in dyspnea with exertion. No angina or chest pain. More fatigue. Admits to poor compliance with diet, medications, supplements and exercise.
- **2014 Corus gene expression:** see report: Score 27 with 29 % likelihood of obstructive CHD.
### Lipids, Lipoproteins, and Apolipoproteins Tests

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Optimal</th>
<th>Borderline</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apo A1</td>
<td>1.3</td>
<td>1.0-1.9</td>
<td>&gt;3.0</td>
</tr>
<tr>
<td>LDL C</td>
<td>126</td>
<td>100-190</td>
<td>&gt;200</td>
</tr>
<tr>
<td>Apo A1:AI</td>
<td>0.74</td>
<td>0.7-1.2</td>
<td>&gt;1.4</td>
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</table>

### Inflammation Tests

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<th>Parameter</th>
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<th>Borderline</th>
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<tbody>
<tr>
<td>hsCRP</td>
<td>&lt;3</td>
<td>3-10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Lp(a)</td>
<td>0.2</td>
<td>0.3-0.9</td>
<td>&gt;1.0</td>
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</tbody>
</table>

### Diabetes Tests

<table>
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<th>Parameter</th>
<th>Optimal</th>
<th>Borderline</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>HbA1c</td>
<td>&lt;5.7</td>
<td>5.7-6.5</td>
<td>&gt;6.5</td>
</tr>
</tbody>
</table>

### Discussion

- **HDL Map:** This HDL map is OPTIMAL and is associated with a low risk of CVD.
- **Lp(a) levels:** Elevated Lp(a) levels are associated with increased cardiovascular risk.

### Cholesterol Balance Score

<table>
<thead>
<tr>
<th>Component</th>
<th>Production/Absorption</th>
<th>Over Absorber</th>
<th>Over Producer</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.8</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Notes
- HDL-C values are low: 35 mg/dL or lower.
- LDL-C values are high: 160 mg/dL or higher.
- High triglycerides: >200 mg/dL.
- High fasting glucose: >100 mg/dL.

---

**Interpretation:**
- **Lp(a) levels:** Elevated Lp(a) levels are associated with increased cardiovascular risk.
- **HDL Map:** This HDL map is OPTIMAL and is associated with a low risk of CVD.

---

**Cardiac Risk:**
- **Lipoprotein(a) levels:** Elevated Lp(a) levels are associated with increased cardiovascular risk.
- **Triglycerides:** High triglycerides are associated with an increased risk of cardiovascular disease.
- **Glucose levels:** High fasting glucose levels are associated with an increased risk of cardiovascular disease.

**Conclusion:**
- Lifestyle modification and statin therapy if LDL-C lowering is indicated.
TEST DESCRIPTION

The Corus CAD test has been validated in two clinical studies, PREDICT (asymptomatic and asymptomatic patients referred for cardiac catheterization, NCT01680268) and COMPASS (asymptomatic patients referred for myocardial perfusion imaging, NCT01175946). The Corus CAD gene expression test measures the expression levels of 23 genes. An algorithm is applied to the gene expression results to calculate a score that indicates the likelihood of the presence of obstructive coronary artery disease (CAD) in a patient. The score ranges from 0-40.

The likelihood of obstructive CAD is based on a clinical validation study (COMPASS, NCT01175946). The study analyzed 431 non-diabetic patients who had no previously diagnosed myocardial infarction or revascularization, and who presented with typical or atypical symptoms suggestive of obstructive CAD. The prevalence of CAD in this study was 15%. The sensitivity, specificity, and NPV were 89%, 92%, and 96%, respectively at a pre-specified threshold of ≥15. The result of the test should be used by clinicians in conjunction with other tests and clinical information in their assessment of CAD in their patients, and in developing patient-specific clinical management plans.

Patient Name: [Redacted]
Medical Record #: [Redacted]
Blood Collection Date: 02-Apr-2014
Sample ID #: TRF281234
Date of Birth: 09-Feb-1953
Clinic Name: Hypertension Institute
Clinician: Mark Houston
Date Received: 03-Apr-2014
Date Reported: 05-Apr-2014

PATIENT REPORT

Likelihood of Obstructive CAD: 27%
95% Confidence Interval: 23-36%

TEST RESULT SCORE

12%
10%
8%
6%
4%
2%
0%

% of Patients with Obstructive CAD
predicted risk at each CAD score level

COMPASS Study Validation of Obstructive CAD
95% Confidence Interval

PREDICT Study Validation of Obstructive CAD

143S51

Comments:
No comments.
COSHEC ABSOLUTE RISK SCORE FOR CHD AND DEATH WITHIN 5 YEARS

- SCORE IS 41 : HIGH
- This calculates the absolute risk for CHD
- High risk for dying within next 5 years from CHD: 2.3%
RASMUSSEN CHD RISK SCORE

• TOTAL SCORE : 11
• HIGH RISK FOR CHD IS OVER 6
• PREDICTS ADVANCED CHD AT THIS SCORE
What do you want to do now?
Case 3

Stress test: 3 mm ST depression in inferior and lateral leads at peak exercise
No chest pain
Marked dyspnea
Hypertensive response to exercise at 250/100 mm Hg
HRRT abnormal at one minute (dropped only 8 beats)
Case 3

Exercise ECHO: positive for apical ischemia
Case 3

Coronary arteriogram: 95% LAD obstruction.
Single vessel disease
Successful stenting
Symptoms resolved
Case 3

What were his risk factors for obstructive CHD?
Case 3: CHD Risk Factors

- Stress, poor diet, lack of exercise. Increased BP with exercise.
- Inflammation and oxidative stress (high hsCRP and MPO)
- Dysglycemia: FBS 97 mg/dl and high GSP
- Dyslipidemia: Dense and high LDL P 1400, dysfunctional HDL
- Homocysteinemia
- Hypothyroidism
- Positive coronary calcium (115)
- Elevated LpPLA 2
- Carotid IMT with plaque
- Endothelial dysfunction: abnormal Endopat (1.44)
- Abnormal HRRT.
- CV Genetics: MTHFR heterozygote 1298, 9p21 homozygote, ACEI I/D, APO E 4/4
- Microalbuminuria
- Elevated BNP
- Low Co Q 10 and Magnesium
- Elevated MPO causing dysfunctional HDL, oxidative stress etc.
- Moderately elevated Corus gene expression test.
- Noncompliance for several years: meds and supplements
Take Away Messages

- Infinite insults to the CV system result in 3 finite responses to the artery: inflammation, oxidative stress and vascular immune function, that lead to endothelial dysfunction, vascular functional and structural disease, CHD and CHF.
- Endothelial dysfunction is the earliest marker and predictor of future CHD
- The cardiovascular system is an innocent bystander to the correct defensive response to these infinite insults that become chronically dysregulated.
- CV medicine is a process as are other “diseases” and all are interrelated within a metabolic systems biology approach.
- Start early detection with evaluation of biomarkers, expanded advanced CHD risk factor analysis, micronutrient testing, risk scoring systems, CV genetics, gene expression testing and non invasive and invasive cardiovascular testing.
- There is a continuum of risk with all CHD risk factors
- Begin aggressive scientifically proven nutrition, nutraceutical supplements and medications for prevention and treatment of CHD and CHF.
CASE  4

Coronary Heart Disease Case
Objectives

Learn how to apply global cardiovascular risk scoring using the new expanded COSEHC and RASMUSSEN methods.

Review Cardiovascular Genomics and SNP’s, the top 5 CHD risk factors, the details of the correct analysis of each, the top 25 modifiable key CHD risk factors and the other 400 CHD risk factors, how to test and the interpretation.

Review, understand and integrate diagnostic, prevention and treatments for CHD, MI, Angina.

Review metabolic cardiology treatment for CHD and Angina.

Prioritize which laboratory and non invasive laboratory and cardiovascular tests should be evaluated in patients in the primary care setting and how to interpret and treat.

Review integrative medical treatments with nutrition, nutritional supplements, lifestyle and drugs for cardiovascular disease.
History

- 58 year old white male attorney
- CC: fatigue for 8 years and mild DOE with steps and inclines only for 6 months. No chest pain. Mild memory issues for 5 years. Frequent URI’s for 6 years. Erectile dysfunction for 4 years
- PMH: negative except for GERD for 5 years, history of EBV and Hepatitis A
- Non smoker, 4 cups coffee per day, one glass red wine per day
- Stressful job. Married with 4 children
- Good diet. Eats lots of chicken and rice.
- Exercise 4 days per week for one hour without chest pain but has mild progressive dyspnea with exertion.
- Medications: Nexium 40 mg per day for 5 years and daily Cialis 5 mg per day
- No allergies
- FH positive for hypertension, dyslipidemia, DM, CHD and CVA
Physical

- BP 142/88 mm Hg   HR 78
- 6ft 1 inch   Weight 220 pounds
- WC 40 inches
- Body Fat  25%
- HEENT: mild glossitis
- Cardiac: no murmur, rubs or gallops
- No edema
- Skin : dry without rash
- Otherwise normal exam
What are the present problems?
What is your most likely diagnoses?
What other history might you wish to have?
What other tests do you want now?
Lab

- CHEM 12 normal except FBS of 102 mg/dL (high)
- CBC: PCV 38 (low) with MCV 104 (high)
- MAU 340 mg (high) Cystatin C elevated at 1.7 (1.04)
- Homocysteine 12 (high)
- Fibrinogen 490 (high)
- HS CRP 2.5 (high)
- Ferritin: 162 (high)
- 2h GGT 182 mg/dL (abnormal)
- HbAIC 6.2 (high)
- MNT – micronutrient testing: decreased B12, chromium, pantothenic acid, Co Q10, choline, Vitamin C, Vitamin D and copper. (see report)
- B12 level 143 (low)
- Vitamin D 15 ng/ml (low)
- TFTs normal except for TSH of 3.4 (high) / positive TPO antibodies
- CoQ10 1.5 ug/ml (low)
- LPP advanced lipid profile: LPP 891, dense LDL, low HDL 2 b. (see report)
Lab

- Genotype: heterozygote hemochromatosis, 2 SNPs for MTHFR (677 and 1298), Apo E 4/4, 9pq21 homozygote, heterozygote SOD and GSH, homozygote CYP 1A2.
- ADMA elevated
- Adiponectin low
- MPO high at 830
- Increased 8-OHdG and MDA with low PAO (antioxidant capacity)
- GGTP 82 elevated
- Ox LDL 99 elevated
- Hormones normal: Free testosterone and DHEAS
- Toxin Screen: Positive for arsenic
- Gluten sensitivity negative
- Positive IGG for EBV and Hepatitis A
TMT See report: Frequent PVVs with Ventricular quadrigeminy, anterior and lateral ST depression of 1-2 mm at peak exercise.

Endopat: see report. Endothelial dysfunction at 1.44, augmentation index is increased (stiff arteries with increased pulse wave velocity) and HRV is abnormal.

CWPWA (computerized arterial pulse wave analysis): C1 6 (low), C 2 5 (low)

Carotid duplex: Increased IMT with less than 50% plaque bilateral

ECHO: see report. Borderline normal EF at 50%, mild PI and trace TR.

MCG (magnetocardiogram): abnormal/positive

Retinal scan: Grade 1 KW changes

24 hour ABM: BP average 134/86mm Hg, non dipper, BP load 35% over 140/90 mm Hg, no surges in AM
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<tr>
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<th>PHASE</th>
<th>MSH</th>
<th>GRADE</th>
<th>HR</th>
<th>BP</th>
<th>RPP</th>
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<td>4.6</td>
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<td>14.0</td>
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<td>3.0</td>
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<td>160/72</td>
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<td>3.0</td>
<td>99</td>
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</table>
EXERCISE STRESS TEST REPORT

7/22/2013 09:26:57

Baseline Peak Values

66 bpm 155 bpm (96% of predicted 162 bpm max)
120/72 192/82 (at 01:08 mins of Recovery)
10.2 METS

Max Exercise

155 bpm (96% of predicted 162 bpm max)

10.1 METS

Test Criteria:
Protocol: Bruce
Elapsed time of test: 8:59
Maximum stage reached: Bruce: 3
Reasons for testing: New Pt. Physical

Test Evaluation:
Baseline ECG findings: NER
Description of any S-T segment changes: INF WIDE ST
UPSLIDING. BVALNT ST CHANGES. ANX/ITY ST DEPRESSION 2MM/WIDE
ST UPSLIDING
HR & BP response to exercise: Normal
Reasons for terminating the test: Adequate Test

Physical symptoms:

Paraarrhythmias: PVC'S, QUADRICOMITY

Functional aerobic capacity & physical response: 102%

Comments:

Physician: MCN
**LPP** Lipoprotein Particle Profile™

**Draw Date:** July 22, 2013  
**Report Date:** July 25, 2013

---

**Lipoprotein Particle Numbers (nmol/L)**

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<tr>
<th>Lipoprotein</th>
<th>Value</th>
<th>Reference Value</th>
<th>Alert (Notes Page 3)</th>
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<tbody>
<tr>
<td>VLDL Particles</td>
<td>61</td>
<td>&lt;63</td>
<td>Borderline High (12)</td>
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<tr>
<td>Total LDL Particles</td>
<td>889</td>
<td>&lt;260</td>
<td>Borderline High (15)</td>
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<tr>
<td>Non - HDL Particles</td>
<td>922</td>
<td>&lt;1000</td>
<td>Borderline (10)</td>
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<tr>
<td>HLp - Intermediate LDL Particles</td>
<td>144</td>
<td>&lt;150</td>
<td>Borderline (11)</td>
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<tr>
<td>Small - Dense LDL III</td>
<td>355</td>
<td>&lt;300</td>
<td>Borderline High (15)</td>
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<td>Small - Dense LDL IV</td>
<td>111</td>
<td>&lt;100</td>
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<td>Total HDL Particles</td>
<td>810</td>
<td>&gt;7000</td>
<td>Borderline (20)</td>
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<td>Large - Buoyant HDL 2b:</td>
<td>5823</td>
<td>&gt;15000</td>
<td>Borderline (20)</td>
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</table>

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**Biomarkers and Risk Factors**

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<th>Biomarker</th>
<th>Value</th>
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<th>Alert (Notes Page 3)</th>
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<tbody>
<tr>
<td>Apo B-100 (mg/dL)</td>
<td>94</td>
<td>40 - 100</td>
<td>Borderline (20)</td>
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<tr>
<td>LDL (mg/dL)</td>
<td>65</td>
<td>60 - 90</td>
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<tr>
<td>Metabolic Syndrome Traits</td>
<td>3.0</td>
<td>Zero</td>
<td>Probable (5)</td>
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<td>C-Reactive Protein (mg/L)</td>
<td>1.5</td>
<td>&lt;3.0</td>
<td>Borderline (9)</td>
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<tr>
<td>Insulin (uU/mL)</td>
<td>12.6</td>
<td>4.8 - 25.6</td>
<td>Borderline (20)</td>
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<tr>
<td>Homocysteine (mmol/L)</td>
<td>1.1</td>
<td>&gt;11.0</td>
<td>Borderline (20)</td>
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</table>

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**Lipid Panel (mg/dL)**

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<th>Lipid</th>
<th>Value</th>
<th>Reference Value</th>
<th>Alert (Notes Page 3)</th>
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<tbody>
<tr>
<td>Total Cholesterol</td>
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<td>&lt;200</td>
<td>Borderline High (1)</td>
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<tr>
<td>LDL - Cholesterol</td>
<td>150</td>
<td>40 - 130</td>
<td>High (2)</td>
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<tr>
<td>HDL - Cholesterol</td>
<td>81</td>
<td>&gt;40</td>
<td>Borderline (5)</td>
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<tr>
<td>Triglycerides</td>
<td>122</td>
<td>200 - 150</td>
<td>Borderline (5)</td>
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<tr>
<td>Non - HDL - Chol (calc)</td>
<td>162</td>
<td>&lt;160</td>
<td>High (5)</td>
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</table>
Summary of Deficient Test Results

Testing determined the following functional deficiencies:

- Vitamin B12
- Chromium
- Pantothenate
- Coenzyme Q-10
- Choline
- Vitamin C
- Vitamin D3
- Copper

Graphs and data indicate:

- Spectroxx: 
  - ... (data not clearly visible)

- Immunidex:
  - ... (data not clearly visible)

Additional notes: 085 865
**Telomere Test Results**

**Patient Telomere Score:** 6.58

**Percentile relative to patient age and population:** 39%

The Patient Telomere Score is a calculation of the patient telomere length derived from nucleated white blood cells obtained from whole blood. This result is graphed relative to the average telomere length of a sample population in the same age range. The higher the telomere score, the "younger" the cells.

A Patient Telomere Score that is above the black line (green box) is an above average Telomere score.

A Patient Telomere Score that is below the black line (red box) is a below average Telomere score.

If patient age was not provided, a horizontal red/green line will be shown which represents the patient Telomere score across all age groups.
Endo-PAT2000

Patient Information

ID: [redacted]  Name: [redacted]  Systolic BP: 110 mm Hg
Age: [redacted]  Gender: [redacted]  Diastolic BP: 78 mm Hg
Height: [redacted]  Weight: 190 lb  BMI: 32.4
Comments:

Study Information

Test Duration: 09:11:40  PATographer: NK
Recording Ver: 3.3.2  Analysis Ver: 3.3.2  Occ. Borders: Automated

PAT Signals

Occluded Arm

Control Arm

Study Results

RHI: 1.44  Endothelial Dysfunction
Heart Rate: 88 bpm

Recommendations

Physician's Name:  Signature: [signature]
**Augmentation Index (AI) - a measure of Arterial Stiffness**

- AI: 85%
- AI@75bpm: 72%

\[
AI = \frac{(P2-P1)}{P1} \times 100\%
\]

Averaged - 125 pulses

**Average PAT Waveform**
(from baseline segment)

**AI@75bpm in female population as function of age**

**Patient Relative to Age and Gender matched distribution**
**Time Domain**
- Mean NN: 917 ms
- SDNN: 45.69 ms
- RMSSD: 33.93 ms
- pNN50: 5.81%
- Triangular Index: 14.63

**Frequency Domain**
- LF (0.04-0.15 Hz): 159.37 ms²
- HF (0.15-0.4 Hz): 134.96 ms²
- LF/HF: 1.18
CTOR Echocardiography Report

Age: 58
DOB: 06/25/1955
Referring: Ordering: 
Priority: In/out: Location: St. Thomas Hospital

Study Date: 08/15/2013 1:21 PM

Vitals
HG: 
HR: 
Rhy: 
Wt lbs: 
T BMI: 
BSA: 
BP: / 

Measurements
LV Systolic Function
EF: 50 % > 50%
RV free wall thk: 

Conclusion

Borderline normal left ventricular systolic function. LVEF = 50 %.
Normal right ventricular size with normal function.

Interpretation Detail

Ind: CAB
LV: The left ventricle is normal in size with borderline normal systolic function. The ejection fraction is 50 %.
LA: The left atrial appendage is normal.
RV: The right ventricle is normal in size with normal function.
MV: There is trace mitral regurgitation.
AV: The aortic valve is trileaflet but mildly sclerotic.
TV: There is trace tricuspid regurgitation.
PV: There is mild pulmonic regurgitation.
IAS: The interatrial septum is normal. There is no interatrial shunt by color or saline contrast. The IAS is moderately mobile.
Ao: The descending aorta is normal.
Misc: The TEE probe was placed by Dr. Delboy M.D. 2D TEE examination was performed. Spectral and color doppler were performed.

Post-CAB

Gen: Procedures performed: CAB
RV: Compared to pre-CPB, the RV size is unchanged.
LV: Compared to pre-CPB, the LV function has improved. The LVEF post-op is 65 %.
Ao: The aorta is unchanged.
AV: The aortic valve is unchanged.
MV: The mitral valve is unchanged.
TV: The tricuspid valve is unchanged.
Cardiac Catheterization

• Left main 50-60 % stenosis
• Left anterior descending: Multiple stenosis at 35%, 60% and 95% high grade obstruction.
• Left circumflex 50% stenosis

• Successfully stented LAD 95% obstruction.
Cardiac Catheterization

- Final Report -

CATHETERS: 4-French JL4, 3DRG, and pigtail catheter.

CONTRAST: Omnipaque, total 150 mL.

HEMODYNAMIC DATA: Pre-angiographic aortic pressure 127/77 with a mean of 98. Pre-angiographic left ventricular pressure 132 with LVEDP of 24. Post-angiographic aortic pressure 107/59 with a mean of 84. Post-angiographic left ventricular pressure of 96 with LVEDP of 32 mmHg.

LEFT VENTRICULOGRAPHY: Left ventriculogram was performed in a single-plane RAO projection. The left ventricular cavity size, wall thickness, and global contractility were normal. The estimated left ventricular ejection fraction was 60% to 65%. Elevation of the LVEDP consistent with diastolic dysfunction. There was no demonstrable mitral regurgitation, and the aortic root appeared normal in diameter.

CORONARY ARTERIOGRAPHY:
1. Left main: Left main coronary artery is normal in caliber, bifurcating into the LAD and left circumflex system. The distal left main coronary artery had a focal 50% to 60% stenosis prior to its bifurcation.
2. The LAD: Left anterior descending coronary artery is a type 3 vessel. The LAD gives rise to 2 proximal diagonal branches with mild to moderate disease involving the proximal aspect. The first diagonal branch has a focal 30% to 35% stenosis with the second diagonal branch having a proximal 50% to 60% stenosis. The LAD has a focal eccentric lesion just beyond the second diagonal branch and first septal perforator branch in the mid segment of the vessel of 90%. The remainder of the LAD, as it extends in the interventricular groove to reach and wrap around the cardiac apex, demonstrates only mild luminal irregularities.
3. Left circumflex: Left circumflex coronary artery is a nondominant system. It gives rise to a trivial sized 1st, 2nd and 3rd marginal, followed by a small to moderate 4th marginal of proximal bifurcating manner and a terminal small to moderate caliber marginal branch. The ostium of the circumflex demonstrates some disease felt to be 30% to 50%. It is somewhat difficult to visualize due to overlapping at the angulation of the vessel.
4. The right coronary artery system is normal in caliber and dominant, giving rise to the RCA with bifurcating small 1st posterolateral branch and a 2nd and 3rd posterolateral branch. The right coronary artery demonstrates only mild luminal irregularities diffusely.

SUMMARY:
1. Preservation of left ventricular systolic function with elevation of the left ventricular end diastolic pressure.
2. Obstructive coronary artery disease involving a high-grade distal left main, including subtotal mid left anterior descending and ostial circumflex disease.

Page 2 of 3
(Continued)
Three Finite Responses of Vascular System to an Infinite Number of Insults: Functional Medicine Approach to Diagnosis

- **Inflammation**: Increased HSCRP, MPO, fibrinogen, ADMA
- **Oxidative Stress**: increased oxLDL, MPO, ADMA, 8OH dG, MDA, increased Fe and ferritin, low COQ 10 and micronutrients and low oxidative defense
- **Immune Dysfunction**: Increased TPO, low vitamin D, micronutrients
Problems and Diagnosis
Discuss reasons for each and treatment of each

- Fatigue
- DOE
- Decreased memory
- Frequent URI
- GERD on chronic PPI
- Stress
- Positive FH for hypertension, DM, dyslipidemia, CHD
- Increased coffee intake
- Endothelial dysfunction and erectile dysfunction.
- Hypertension: office and 24 hour ABM, non dipper with increased BP load and average BP and positive retinal scan grade 1
- Glossitis
- Dry Skin
- Dysglycemia: FBS, GTT and HgAIC
- Visceral obesity
- Hypothyroidism with positive TPO
Problems and Diagnosis
Discuss reasons for each and treatment of each

- Anemia with increased MCV
- Increased MAU and cystatin C
- Homocysteinemia
- Increased fibrinogen
- Increased ferritin
- Increased HSCRP
- Dyslipidemia: Increased LDL-P, remnant particles, and oxLDL
- Multiple nutrient deficiencies by MNT: B12, B5, chromium, CoQ10, choline, Vitamin C, Vitamin D, copper.
  - Low B12
  - Low Vitamin D
  - Low C0Q10
  - Increased ADMA
  - Low adiponectin
  - Increased oxidative stress with low oxidative defense
Problems and Diagnosis
Discuss reasons for each and treatment of each

- Increased ADMA
- Low adiponectin
- Increased oxidative stress with low oxidative defense
- Increased GGTP
- Increased arsenic
- Tachycardia (elevated heart rate above 66)
- Positive TMT with PVSs quadrigeminy
- Abnormal CAPWA with low C1 and C2 AC
- Positive MCG
- Carotid artery IMT increased with plaque
- Short telomeres
- History of EBV and Hepatitis A
- High arsenic level
Problems and Diagnosis: Answers

- Fatigue: CHD, micronutrient deficiencies, toxins, stress, anemia
- DOE: CHD, anemia,
- Decreased memory: low B12, choline, vitamin D, CoQ10 etc.
- Frequent URI: low vitamin D and B vitamins, stress, low oxidative defense
- GERD: Stress, diet
- Increased coffee intake: CYP A12 increases arterial stiffness, BP, HR and risk for CHD and MI
- Endothelial dysfunction: low NO and increased ADMA, vascular disease, 3 finite responses, micronutrient deficiencies
- Hypertension: office and 24 hour ABM: genetics, coffee, stress
- Glossitis: Low B12
- Dry Skin: Low B vitamins and vitamin D
- Dysglycemia: FBS, GTT and HgAIC: Low chromium, B12, visceral obesity, ox LDL, diet, stress
- Visceral obesity: arsenic, diet, IR.
Problems and Diagnosis: Answers

- Anemia with increased MCV: Low B12, PPI
- Increased MAU and cystatin C: hypertension, inflammation
- Homocysteinemia: MTFHR, PPI depletes B vitamins, caffeine
- Increased fibrinogen: inflammation from micronutrient deficiencies, arsenic, oxidative stress.
- Increase ferritin; Hemochromatosis
- Increased HSCRP: inflammation from chronic vascular disease, dyslipidemia, chronic infections, arsenic
- Dyslipidemia: Increased LDL-P, remnant particles, and oxLDL: toxins and infections, genetics, diet, visceral fat, IR.
- Multiple nutrient deficiencies by MNT: B12, B5, chromium, CoQ10, choline, Vitamin C, Vitamin D, copper.: PPI, stress, diet, oxidative stress, inflammation.
- Low B12: PPI
- Low Vitamin D: Diet, lack of sunshine
- Low CoQ10: stress
- Increased ADMA: PPI
- Hypothyroidism with positive TPO: diet, toxins, micronutrients
Problems and Diagnosis: Answers

- Low adiponectin: visceral obesity, adipokines, IR
- Increased oxidative stress with low oxidative defense: arsenic, low copper, low B vitamins, CoQ10, dysglycemia, dyslipidemia, hypertension
- Increased GGTP: toxin load, low glutathione, poor detoxification pathways.
- Increased arsenic: diet of rice, OJ and chicken. Causative in dyslipidemia and CHD.
- Severe CHD: family history, genetics, inflammation, oxidative stress, autoimmune dysfunction, BP, lipids, glucose, visceral obesity, caffeine, homocysteine, hypothyroidism, arsenic
- Tachycardia: abnormal PNS/SNS balance, anemia
- Quadrigeminy: CHD with ischemic induces PVCs
- Short Telomeres: increased risk CHD. Oxidative stress, inflammation, micronutrient def. from all causes noted
- History of EBV and Hepatitis A: causative in dyslipidemia and CHD
Problems and Diagnosis: Treatments

- Fatigue: Stent to LAD, replace micronutrients, remove toxins
- DOE: Stent to LAD, anemia
- Decreased memory: B12, choline, Vitamin D replacement
- Frequent URI: vitamin D and B vitamins
- GERD: probiotics, glutamine
- Stress reduction management
- Increased coffee intake: Stop all caffeine
- ED: arginine, citrullene, beet root juice, green vegetables
- Hypertension: ACEI or ARB and natural BP supplements
- Glossitis: B12
- Dry skin: B vitamins and vitamin D
- Dysglycemia: Visceral weight loss, chromium, B vitamins, diet
- Visceral obesity: exercise, diet, treat IR
Problems and Diagnosis: Treatments

- Anemia with increased MCV: B12
- Increased MAU and cystatin C: hypertension control (RAS), lipoic acid
- Homocysteinemia: 5 methyl folate, B6, B12, betaine, stop caffeine
- Increased fibrinogen: replace micronutrient def, remove arsenic
- Increase ferritin: phlebotomy, low iron intake
- Increased HSCRP: treat chronic vascular disease, dyslipidemia, chronic infections, arsenic, BP etc
- Dyslipidemia: remove: toxins and infections, statin
- Multiple nutrient deficiencies by MNT: B12, B5, chromium, CoQ10, choline, Vitamin C, Vitamin D, copper. Replace all
- Low B12: replace
- Low Vitamin D: replace to 60 ng/ml
- Low CoQ10: replace to 3 ug/ml
- Increased ADMA: stop PPI, start beet root juice, green vegetables, treat DM, BP and lipids.
- Hypothyroidism with positive TPO: improve diet, remove toxins, start T3-T4 replacement, replace nutrients
Problems and Diagnosis: Treatments

- Low adiponectin: reduce visceral obesity, treat CHD risk factors
- Increased oxidative stress with low oxidative defense: remove arsenic, start copper, B vitamins treat BS, lipids and BP
- Increased oxidative stress with low oxidative defense: replace all nutrients and treat CHD risk factors
- Increased GGTP: remove toxins
- Increased arsenic: chelation, reduce intake
- Severe CHD: Stent to LAD, control 3 finite responses and remove all insults, LDL-P to 700, FBS to 75 mg/dL, BP 120/80 mm Hg, HC to 5, HSCRP to 1.0, omega 3 FA, Kyolic garlic, vitamin K2MK 7, curcumin, quercetin, statin, ARB or ACEI, colchicine (?)
- Tachycardia: treat anemia, breathing techniques, HRV training
- Quadrigeminy: omega 3 FA, magnesium, reduce stress, exercise
- Abnormal CAPWA with low C1 and C2 AC: Vitamin C, increase NO and all micronutrients, BP, lipid and glucose control
- Positive MCG and carotid artery IMT and plaque: CHD risk factor Rx
- Short Telomeres: remove inflammation, oxidative stress and immune dysfunction.
- History of EBV and Hepatitis A: balance Th1/ Th2 with sterolins.
Specific Treatments

- Mediterranean diet, stopped all caffeine, increased dark green leafy vegetables to 8 servings per day with beets.
- Cardiac rehab then ABCT exercise interval aerobics and resistance exercise.
- Stress management and relaxation therapy and HRV breathing training.
- Crestor 10mg HS, berberine HCL 500 mg bid, niacin 500 mg hs with food and with quercetin 500 mg.
- Metoprolol 25 mg bid.
- ASA one per day.
- Quinapril 80 mg HS and melatonin 3 mg hs.
- Armour thyroid 60 mg per day.
- B12 lozenges sublingual 1000 mcg/day.
- Vitamin D 10,000 IU per day for one month then 4000 IU per day.
- Chromium 800 ug per day.
- CoQ 10 200 mg per day.
- B complex with methyl folate 5000 ug per day.
- Vitamin C 500 mg bid with copper 1 mg/zinc 50 mg per day.
- R lipoic acid 100 mg, NAC 1000 mg and whey protein 40 grams for glutathione.
- Oral chelation for arsenic.
- Omega 3 FA 5000 mg, vitamin K 2 MK 7 500 mcg, Kyolic garlic 600 mg bid, curcumin 1000 mg bid, magnesium chelates 500 mg bid and epsom salts bath.
- Phlebotomy for hemochromatosis.
- Probiotic 50 billion cfu per day with glutamine 1000 mg bid. Stopped PPI.
Treatment results 6 months

- All symptoms resolved
- Weight 195 lbs, WC 34 inches, 20 % BF
- BP 122/78 mm Hg, dipper, HR 62 b/m
- All labs normal
- LPP 608, LDL type A, HDL 2b and HDL P normal
- MNT normal
- Endopat normal 2.66 and CAPWA normal
- TMT normal
Take Away Messages

- Infinite insults to the CV system result in 3 finite responses to the artery: inflammation, oxidative stress and vascular immune function, that lead to endothelial dysfunction, vascular functional and structural disease, CHD and CHF.
- Endothelial dysfunction is the earliest marker and predictor of future CHD
- The cardiovascular system is an innocent bystander to the correct defensive response to these infinite insults that become chronically dysregulated.
- CV medicine is a process as are other “diseases” and all are interrelated within a metabolic systems biology approach.
- Start early detection with evaluation of biomarkers, expanded advanced CHD risk factor analysis, micronutrient testing, risk scoring systems, CV genetics, gene expression testing and non invasive and invasive cardiovascular testing.
- There is a continuum of risk with all CHD risk factors
- Begin aggressive scientifically proven nutrition, nutraceutical supplements and medications for prevention and treatment of CHD and CHF.
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