

# Critical Assessment of Diabetes and Complications: Honing your Detective Skills

Beverly Dyck Thomassian,  
RN, MPH, BC-ADM, CDE  
www.DiabetesEd.net



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## Objectives:



1. Identify common yet often under diagnosed complications associated w/ type 1 and type 2 diabetes.
2. State strategies to identify previously undiscovered diabetes complications during patient assessments.
3. Demonstrate steps involved in lower extremity assessment.

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## Patient is Gaining Weight

- 68 yr old female complains of 4 lb wt gain a week for past month. Wt 140lbs, BMI 27. BG levels 200-300s. B/P 142/96
- **Reported** daily meds include:
  - ↓ glyburide 10mg ac breakfast
  - ↓ Actos 30mg ac breakfast
  - ↓ Glargine 30units at night
  - ↓ Lispro sliding scale with meals
  - ↓ Synthroid (not sure of dose)
  - ↓ Lasix 20mg a day
  - ↓ Zyprexa 10mg a day



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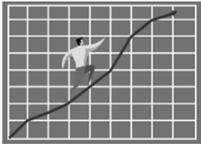
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## Fluid Weight Gain

- ▣ People with diabetes at greater risk for Congestive Heart Failure (CHF) due to increased CVD risk factors.
- ▣ Actos and Avandia, (TZD's), can cause fluid wt gain and worsen CHF.
- ▣ Metformin used cautiously in pt's w/ CHF due to increased risk of renal impairment

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## Thyroid Disease and Diabetes

- ▣ 27 mil Americans have over or under active thyroid glands, but more than half remain undiagnosed.
- ▣ More than 8 out of 10 pts w/ thyroid disease women.
- ▣ 15 to 30% of people w/ diabetes & their siblings or parents are likely to develop thyroid disease (compared to 4.5 percent of the general population).
- ▣ Check TSH on Type 1 & 2 annually or if indicated.

*AACE Website*

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## Thyroid & TSH\* Levels



AACE 2012 Guidelines

- ▣ \*Thyroid Stimulating Hormone - secreted by pituitary gland
  - ↳ controls thyroid hormone thyroxine production
  - ↳ first and best test
  - ↳ TSH Norm = up to 4.5 mIU/mL
  - ↳ Treatment based on TSH plus symptoms.
    - ▣ 4.5 – 10 based on risk, s/s
    - ▣ 10 or more = treat
  - ↳ Lower = hyperthyroidism
  - ↳ Higher = hypothyroidism

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## Hypothyroidism

- Hashimoto's thyroiditis – autoimmune thyroid
  - ↓ most common cause of hypothyroidism w/ dm
- Type 1 and type 2 at greater risk
- Screen annually for thyroid disease in diabetes
- Clinical features: fatigue, wt gain, dry skin, cold intolerance, depression, constipation, dyslipidemia
  - ↓ Higher risk of CVD – monitor risk
- Dx: high TSH, then test for free T4, autoantibodies, and thyroid scans as needed
- Tx: replacement with levothyroxine (75-125 ug)

*AACE Thyroid Guidelines*

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Diabetes: 30% Depressed  
12% of those, major depression  
70% don't receive treatment



- Treatment includes:
- ↓ referral to mental health professional
  - ↓ Medications

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## Novel / Atypical Antipsychotics Linked to Hyperglycemia

- Severe cases of hyperglycemia – even death reported
- Monitor BG regularly for DM patients started on this class of med
- If pt at risk for DM, determine fasting glucose before initiating therapy and monitor closely during treatment
- Weight gain may require increased dosing of diabetes therapies.

Summary of FDA warning statement for atypical antipsychotics, 2004

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### Novel/ Atypical Antipsychotics Linked to Hyperglycemia

- ↳ Zyprexa – olanzapine
- ↳ Geodon - ziprasidone
- ↳ Seroquel – quetiapine
- ↳ Risperdal - risperadone
- ↳ Clozaril - clozapine
- ↳ Abilify – aripiprazole
- ↳ Latuda - lurasidone



*Consensus Development Conference on Antipsychotic  
Drugs and Diabetes 2004*

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### New Insulin Start – No orders

- 71 year old woman, type 2 for 8 years
- Weight 90 kg
- DM Meds -
  - ↳ Metformin 2000mg day
  - ↳ Actos 15 mg (just started)
  - ↳ Admits to taking am meds ~ 4 xs a week, but always takes pm meds
- A1c 10.3% Checks BG ~ 5 xs wk in am (200-250) C/O of Many hyperglycemia SE

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### What Would You Start her on?



- Intensive insulin therapy based on her wt?
  - ↳  $90\text{kg} \times 0.5 = 45$  units a day
  - ↳ 7 units bolus each meal, ~ 20 units basal at hs?
- Start w/ 10 units Basal at HS?
- What factors would influence your decision?

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## What Would You Start her on?

- My insulin suggestion
  - ↓ Pre Breakfast - 20 units 70/30 insulin
    - 14 units basal / 6 units bolus
  - ↓ Pre dinner - 10 units 70/30 insulin
    - 7 units basal/ 3 units bolus
- BGM suggestion
  - ↓ 2 x's a day
  - ↓ Before breakfast, 2 hrs after dinner

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## Bev's Rationale



- Pt not very connected to diabetes
- Does not have a scheduled life
- Limited record keeping skills
- Overwhelmed with all her the medications she is already taking
- Start slow, gradually intensify
- Start where they are at...
- Safe and feasible short and long term?

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## Patient is Losing Weight

SR, 49 yr old woman w/ lean "type 2" 7 yrs.  
Monitors BG 1 x daily  
A1c 13.9%  
Insulin: 14 u Lantus at hs (uses pens)  
Humalog if BG > 200 (says too expensive)  
Also on Metformin 500mg BID  
At 5'7, her usual wt is 120, but now 106 lbs  
C/O of nausea, fullness, fatigue  
No health insurance



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## Physical Assessment and Referrals

- What do you include in physical exam?
- What referrals?



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## Comprehensive Diabetes Evaluation – Physical Exam

- Height, wt, BMI
- B/P – orthostatic hypo, hypertension
- Fundoscopic Evaluation (referral may be needed)
- Thyroid palpation
- Skin exam
- Comprehensive Foot exam (pulses, inspection, sensation, vibration)

ADA Clinical Practice Recommendations

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## Diabetes Detective

- What other comorbidities are you suspecting?
- Any labs you would like to check?
- What type of diabetes?
- Social situation?
- Consider her lack of insurance and income level during your discussion.
- Medication changes?



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## Suggested changes

- Regular insulin 3 times a day – 3 units if don't check BG (eat 45 gms of carb)
- If check BG, add 1 unit for each 50 pts above 150
- Try and eat 3 times a day – use liquid calories as needed, low fiber
- Check BG at least once a day
- Weekly phone call check in




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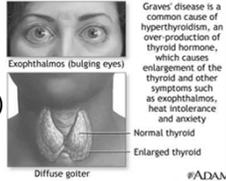
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## Hyperthyroidism

- Graves Disease (most common)
- 0.5 – 2.0% risk in type 1
- Autoimmune disorder:
  - ↓ Symptoms: wt loss, hypermetabolism, tremor, exophthalmos, palpitations, tachycardia, heat intolerance, nervousness, hyperglycemia
  - ↓ Diagnosis: Dx: low TSH, then check T3 & T4, autoantibodies, and thyroid scans
  - ↓ Treatment: antithyroid drugs, surgery, radioactive iodine. After treatment, may need thyroid replacement therapy.



AACE Thyroid Guidelines 2002

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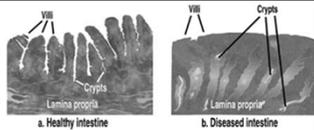
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## Celiac Disease

- Type 1 – Affects 1-16%
- Immune reaction to gluten - affects function of villi in intestine, decreasing nutrient absorption
- S/S: bloating, malabsorption, wt loss, fatty stools, diarrhea, muscle tenderness, failure to thrive
- Diagnosis: measure either anti-endomysial antibodies (EMA) titers or tissue transglutaminase.
- If positive, refer to GI specialist for endoscopy and biopsy of small intestine to confirm diagnosis.




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## Gastroparesis



- Gastroparesis: affects 20 – 30% of pt's w/ longstanding dm
- Delayed emptying of stomach contents due to nerve damage
- S/S include early satiety, fullness, postprandial hypo, vomiting
- Diagnosis: gastric emptying studies, post-prandial hypoglycemia
- Tx: improve BG, small, low fat & fiber meals meds: reglan, erythromycin

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## SR struggling w/ eating

- Gained 20 lbs
- Low blood sugar after meals
- Doesn't feel very hungry
- Doesn't want to check BG
- A1c 9.7%
- Strategies?
- Worries?



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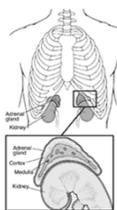
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## Addison's Disease

- 1 in 250 w/ type 1 (thyroid dx = > risk)
- Autoimmune destruction adrenal glands
- Cortisol deficiency
  - decreases hypoglycemia awareness
  - decreases glycogenolysis
- S/S weakness, wt loss, hypoglycemia, dehydration, hyperpigmentation, muscle weakness, salt craving, hyponatremia, hyperkalemia
- Diagnosis: test Anti-21- hydroxylase autoantibody, adrenocorticotrophic hormone cortisol stimulation test
- Treatment: oral hydrocortisone replacement



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## NonAlcoholic Fatty Liver Disease (NAFLD)

- Increasing worldwide prevalence

- ↓ 25% of adults
- ↓ 75% of people w/ DM or obese
- ↓ Up to 50% of obese children



NAFLD = greater than 5.5% fat in liver that can't be attributed to other cause .

*Due to Insulin Resistance and Obesity*

The Metabolically Benign & Malignant Fatty Liver - 2011

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## DM & Fatty Liver

- Fatty Liver and hepatic inflammation is associated with insulin resistance and measures of visceral adiposity
- It also predicts:
  - ↓ Incidence of type 2 diabetes
  - ↓ Heart disease
- Fatty liver disease is directly involved in the pathogenesis of these diseases. Maybe a cause?



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## Finding Liver Disease

- No markers are accurate for diagnosing NASH – only biopsy
- Obese pts or those with metabolic syndrome should be evaluated
- Signs of advanced disease include:
  - ↓ Portal hypertension, spider angiomas, reddening of palms, declining platelet counts an family hx

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## Treating NAFLD

- Since there is no approved treatment for NAFLD and almost every patient with NAFLD will have to change their lifestyle – lose weight, exercise, and eat a healthy diet – it is not necessary to biopsy routinely." *NIH Clinical Center, Dr. Yaron Rotman*

Wt loss of 7-10% linked with a 50% drop in liver fat

Clinical Endocrinology News 12/12



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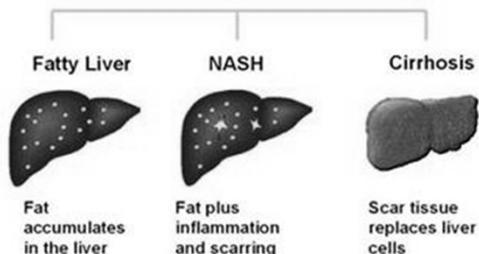
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## Natural History of NAFLD to NASH

### The Spectrum of NAFLD



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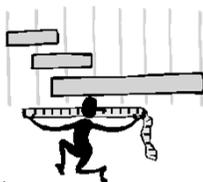
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## Natural History of NAFLD -

- Over 3.5 - 11 year period
- "Benign" Group
  - ↳ 60% remain stable
  - ↳ 13% have improvement
- "Malignant" Group
  - ↳ 28% progress to liver damage

The Metabolically Benign & Malignant Fatty Liver - 2011



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## Diabetes + Obesity = Progression to NASH

- 50% progress from "Benign" fatty liver to Steatohepatitis.
- 2-4 fold risk of developing advanced liver disease compared to those without diabetes.
- About 15% develop cirrhosis and are at increased risk for liver cancer

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## NASH

▣ *Represents the hepatic manifestation of metabolic syndrome:*

- ↓ Abdominal obesity
- ↓ Hypertension
- ↓ Diabetes
- ↓ Dyslipidemia



25 million Americans will develop NASH by 2025 with 20% progressing to cirrhosis, cancer or both

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Over Time Leads to  
**NASH or SteatoHepatitis ...**

- Fibrosis and Cirrhosis
- Liver Cancer
- Liver Failure

Future epidemic of liver transplants??

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## Liver Disease & Glucose

- Hepatitis-C > 40, 3x's rate of diabetes
  - ↓ Increased risk if familial history
- Cirrhosis: 80% of pts have glucose intolerance
- Hepatic failure: associated w/ hypoglycemia due to destruction of hepatocytes, increased insulin production, inadequate storage of glucose
- Hemochromatosis – up to 75% have diabetes
  - ↓ Condition characterized by excessive production and accumulation of iron in liver & other tissues. "bronze diabetes"

Levinthal, Gavin, Tavill, Anthony: Liver Disease and Diabetes Mellitus *Clinical Diabetes* 1999, v17, n2  
Annals of Internal Medicine 2000;133:592-599.

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## Important Stuff to Remember

- Always start with where the patient is at
- Consider the entire milieu
- Listen
- Keep it simple
- Check in often
- Open lines of communication with medical team



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## Lets take a look at his Lower Extremities and Assess

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## Lower Extremity Complications

- Combination of vascular, neurological, and musculoskeletal dysfunction
- After Lower Extremity Amputation (LEA), people have higher mortality rates and subsequent amputation



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## Lower Extremity Amputations Dropping over past 10yrs

- 60% of amputations in 7% of pop
- Higher in men, elderly, minorities, Chronic Kidney Disease (CKD)
- Lower extremity complications represent 20% of hospitalizations for elderly
- Amputations cost \$40,000
- Amputation associated w/ earlier death compared to revascularization
- 10 yr survival after LEA

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## Diabetes and Lower Extremity Ulcers

- Up to 15% of DM patients have ulcers in their lifetime
- Mortality with foot ulcers is twice usual



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## Risk factors for Foot Ulcers/ Amputation



- Previous amputation
- Past foot ulcer history
- Peripheral neuropathy
- Foot deformity
- Peripheral vascular disease
- Visual impairment
- Diabetic nephropathy (especially patients on dialysis)
- Poor glycemic control
- Cigarette smoking
- ADA Task Force - 2008

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## Pathway to Amputation –

Pecoraro, Frykberg

Minor Trauma (environmental)

+

Faulty Healing (intercurrent pathophysiology: circulation, WBC/platelet function)

+

Ulceration

Predicts 72% of amp



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## What Leads to Ulcers

- 86% single precipitating event leading to ulcer
  1. Tight shoe
- 3 classes
  1. Neuropathic
  2. Ischemic (hard to heal)
  3. neuro-ischemic (worst)

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## "I didn't notice"

- ▣ Needle in foot
- ▣ Pebble in shoe
- ▣ Stepped on a nail
- ▣ Cut too deep
- ▣ Shoes were rubbing
- ▣ Others?



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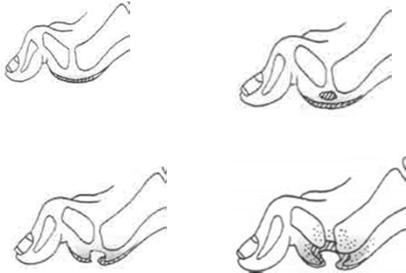
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## Pressure Area Breakdown



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## Walking Cast for Neuropathic Ulcers



Emotional aspects  
Impact on BG

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## Neuropathy Leads to Lower Extremity Complications

### Neuropathies

- Sensory
  - ↓ loss of sensation, painless trauma, repetitive low grade stress
- Motor
  - ↓ muscle atrophy, unbalanced tendon pulling, bone/gait changes, deformities, claw foot
- Motor + Sensory changes = ulcerations
- Autonomic
  - ↓ decreased perspiration, fissures, Charcot's foot

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## Stairway to Amputation

- Neuro + Peripheral Arterial Disease
- Injury or callus
- Wound
- Infected
- Cellulitis
- Gangrene
- Amputation



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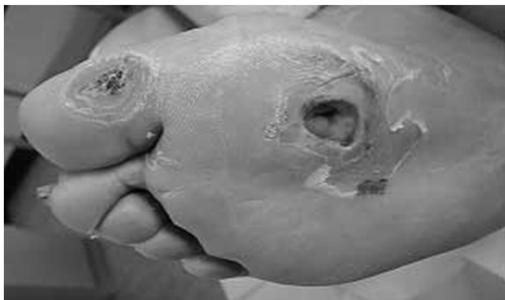
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## Neuropathic Diabetes Foot Ulcers



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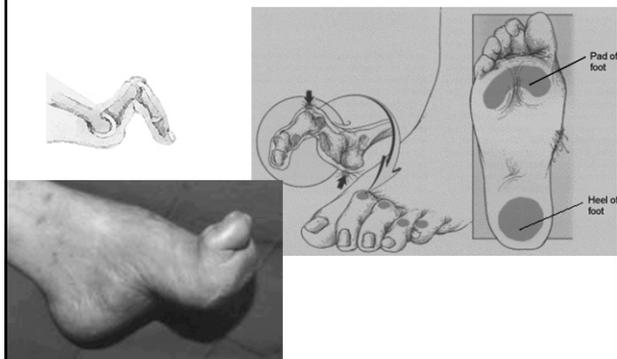
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## Foot Motor/Nerve Deformities



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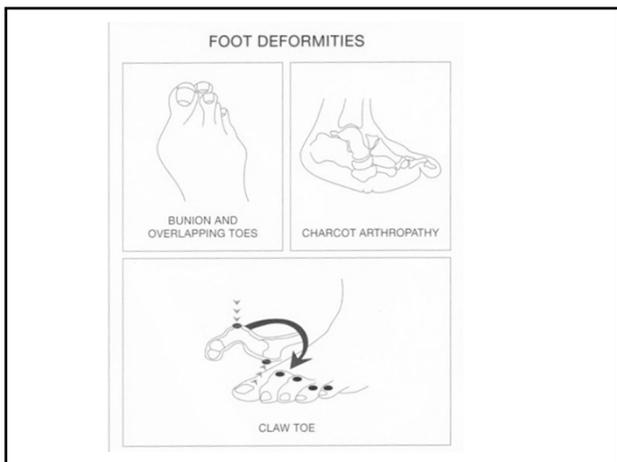
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## Circulation Issues lead to Lower Extremity Problems

- Peripheral Arterial Disease
- Vascular Disease
- Smoking

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**Peripheral Arterial Disease  
Assessment**



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**Pitting Edema**



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**Venous Ulceration**



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## 1<sup>st</sup> Step – Watch Pt Walk

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## Foot Exam – Patient History

- Previous foot ulceration
- Previous amputation
- Diabetes > 10 years
- A1c  $\geq$  7%
- Impaired Vision
- Neuropathic Symptoms
- Claudication



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## Foot Exam – Dermatologic Exam

- Dry Skin
- Absence of hair
- Ingrown nail edges, long or sharp nails
- Interspace maceration
- Ulceration
- Cleanliness



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## Flexibility Assessment

Stiff joint syndrome



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## Visual Inspection/Palpation

- ❑ Breaks in the skin
- ❑ Erythema
- ❑ Trauma
- ❑ Pallor on elevation
- ❑ Dependent rubor
- ❑ Changes in the size or shape of the foot
- ❑ Nail deformities
- ❑ Extensive callus
- ❑ Tinea pedis
- ❑ Pitting edema



VA Guidelines 2004

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## Foot Exam – Screening for Neuropathy

### Test

- ❑ Semmes-Weinstein monofilament 10g
- ❑ Vibration perception threshold testing
- ❑ Tuning Fork 128 Hz

### Significant Finding

- ❑ Lack of perception at one or > sites
- ❑ Vibration perception threshold >24 volts
- ❑ Abnormal vibration perception

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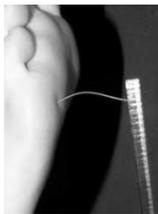
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## Loss of Protective Sensation

- Monofilament Testing
  - ↳ 5.07 touched to plantar surface and top of foot
  - ↳ C shape delivers 10 gms pressure
  - ↳ Test four sites
    - Plantar surfaces of
      - Each great toe
      - 1<sup>st</sup>, 3<sup>rd</sup> and 5<sup>th</sup> metatarsal head



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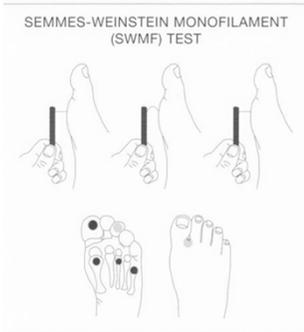
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## Monofilament Testing



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## Monofilament (MF) Procedure *(Int Consensus Grp)*

- Demonstrate procedure on pts forearm or hand
- Have pt close their eyes
- Test four sites in random sequence
  - ↳ (if callus or ulcer, test adjacent surface)
- Bow the MF and ask, "Do you feel it touch you, yes or no?"
- Randomly test at each site 3 times (one of which is a "sham" application – MF not applied)

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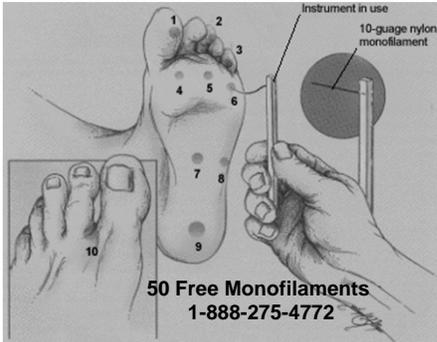
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5.07 monofilament =  
10gms linear pressure



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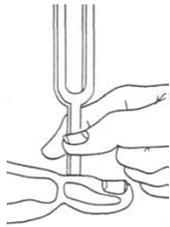
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### Tuning Fork to Detect Polyneuropathy



- ▣ 128 tuning fork
- ▣ Plantar halax
- ▣ Compare sensation to that of examiner

Back to Basics in Diagnosing Diabetic Polyneuropathy with the Tuning Fork! Meijer, et al Diabetes Care, Vol 28, #9 Sept 2005

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### Tuning Fork (TF) Procedure

- ▣ Demonstrate sensation to pt on wrist or elbow w/ and without vibration
- ▣ Ask pt to close eyes
- ▣ Apply TF perpendicularly with constant pressure to dorsum of hallux (1<sup>st</sup> great toe) just proximal to nail bed. Place your index finger of the hand beneath the pts toe to feel vibration and verify.

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## Tuning Fork Procedure

- Use initial sham test and apply non-vibrating TF to be sure pt does not mistake pressure for vibration and ask.. Is the TF vibrating? (No is right answer)
- Use "on-off" method to score.
- Conduct testing 2xs on each great toe

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## Tuning Fork Procedure

- On each test:
  - ↓ Ask pt to ID beginning of vibration
    - "Is it vibrating"?
  - ↓ Ask pt to ID cessation by dampening TF.
    - "Tell me when the vibrating stops"
  - ↓ The number of correct responses = 0-8
  - ↓ At least 5 incorrect responses = peripheral neuropathy

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## Foot Exam – Vascular Exam

<u>Test</u>	<u>Significant Finding</u>
■ Palpation of pulses <ul style="list-style-type: none"><li>↓ dorsalis pedis</li><li>↓ tibial</li></ul>	■ Absent pulses
■ Ankle – Brachial Index (ABI)	■ ABI <0.90, consistent w/ peripheral arterial disease

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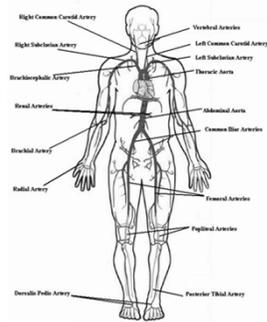
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## Vascular Status Assessment

- 1. Posterior tibial pulse
- 2. Dorsalis pedis pulse
- 3. Temperature
- 4. Appearance



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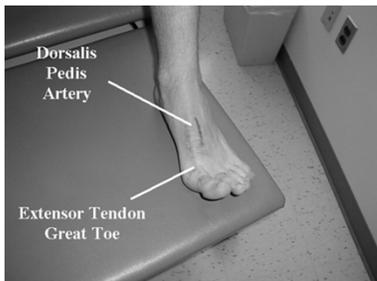
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## Dorsalis Pedis Pulse



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## Taking the DP Pulse



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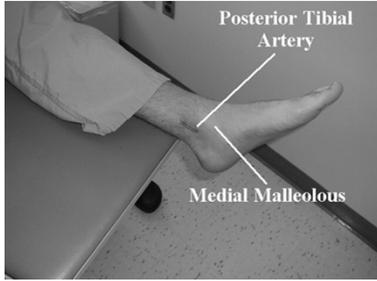
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## Posterior Tibial Pulse




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## Taking the Posterior Tibial Pulse




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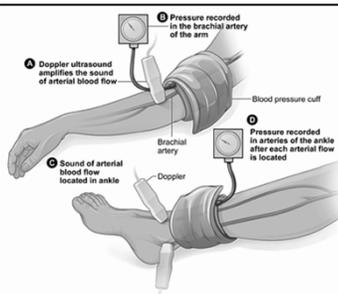
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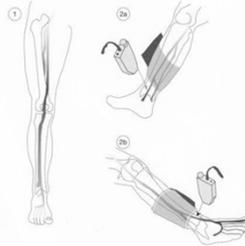
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## ABI Procedure



### CALCULATION OF ANKLE-BRACHIAL INDEX (ABI)



1. Anterior view, right lower limb, normal arterial anatomy
  2. ABI: Place blood pressure cuff above pulse. Place Doppler probe over arterial pulse.
    - a. Posterior tibial artery, ankle systolic pressure
    - b. Dorsalis pedis artery
- ABI calculation: Divide systolic leg blood pressure/systolic arm blood pressure. (ABI <math>< 0.9</math> is normal)

[http://www.nhlbi.nih.gov/health/dci/Diseases/pad/pad\\_diagnosis.html](http://www.nhlbi.nih.gov/health/dci/Diseases/pad/pad_diagnosis.html)

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# Ankle Brachial Index

- **Technique**
- Measure highest systolic reading in both arms
  - ↓ Record first doppler sound as cuff is deflated
  - ↓ Record at the radial pulse
  - ↓ Use highest of the two arm pressures
- Measure systolic readings in both legs
  - ↓ Cuff applied to calf
  - ↓ Record first doppler sound as cuff is deflated
  - ↓ Use doppler ultrasound device
    - Record dorsalis pedis pressure
    - Record posterior tibial pressure
  - ↓ Use highest ankle pressure (DP or PT) for each leg
- Calculate ratio of each ankle to brachial pressure
  - ↓ Divide each ankle by highest brachial pressure

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# The Ankle-Brachial Index

$$ABI = \frac{\text{Lower extremity systolic pressure}}{\text{Brachial artery systolic pressure}}$$

**Should be as Close to 1 as possible = normal circulation**

- The ankle-brachial index is 95% sensitive and 99% specific for PAD
- Establishes the PAD diagnosis
- Identifies a population at high risk of CV ischemic events
- The "population at risk" can be clinically and epidemiologically defined:

Lipfert JG. *Ultrasound Med Biol* 1996;22:391-8; Fegelson HS. *Am J Epidemiol* 1994;140:526-34; Baker JD. *Surgery* 1981;89:134-7; Ountel K. *Arch Surg* 1982;117:1297-13; Carter SA. *J Vasc Surg* 2001;33:708-14

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# Using the ABI: An Example

<p><b>Right ABI</b> 80/160=0.50</p>		<p><b>Left ABI</b> 120/160=0.75</p>	<p><b>ABI</b> (Normal &gt;0.99)</p>
<p><b>Brachial SBP</b> 150 mm Hg</p>		<p><b>Brachial SBP</b> 160 mm Hg</p>	<p><b>Highest brachial SBP</b></p>
<p><b>PT SBP 40 mm Hg</b> <b>DP SBP 80 mm Hg</b></p>		<p><b>PT SBP 120 mm Hg</b> <b>DP SBP 80 mm Hg</b></p>	<p><b>Highest of PT or DP SBP</b></p>

ABI=ankle-brachial index; DP=dorsalis pedis; PT=posterior tibial; SBP=systolic blood pressure.

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## Interpreting the Ankle-Brachial Index

<u>ABI</u>	<u>Interpretation</u>
1.00–1.29	Normal
0.91–0.99	Borderline
0.41–0.90	Mild-to-moderate disease
≤0.40	Severe disease
≥1.30	Noncompressible

Adapted from Hirsch AT, et al. J Am Coll Cardiol. 2006;47:e1-e192. Figure 6.

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## Ankle Brachial Index

- **False Negative Test: Diabetes Mellitus**
- Vessels in diabetics are poorly compressible
- Results in falsely elevated ankle pressure
- **Management**
  - ↓ Segmental Arterial Pressure indicated for ratio < 0.9
  - ↓ Consider angiography or Magnetic resonance angiography

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## Biomechanical Foot Assessment –

<u>Test</u>	<u>Significant Finding</u>
<ul style="list-style-type: none"> <li>■ Plantarflexion &amp; Dorsiflexion of ankles, great toes</li> <li>■ Watch pt ambulate</li> <li>■ Inspect Shoes</li> <li>■ Inspect for deformity</li> </ul>	<ul style="list-style-type: none"> <li>■ Diminished joint mobility</li> <li>■ Decreased vision, gait imbalance, need for assistive devices</li> <li>■ Ability to see/ reach feet</li> <li>■ Corn, calluses, bunions, prominent metatarsal heads, hammertoes, claw toes</li> </ul>

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## Risk Stratification: Population Approaches

- Reality that we cannot give maximum resources to all
- Screening: Appropriate for all, baseline
- Patient education: All need to know risks and self care
- Monitoring condition: Varies with degree of pathology: risk stratification

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## Risk Classification and Referral / Follow-UP

Cat	Definition	Action	Re Assess
0	No LOPS No PAD	Prevention Ed	Yearly
1	LOPS ± Deformity	Special foot wear Consider prophylactic surg if deformity can't be safely accommodated in shoe. Pt. Ed	3-6 mos
2	PAD ± LOPS	Consider prescriptive footwear. Vascular consult.	2-3 mos (by specialist)
3	Hx of amp ulcer	Same as Categ 1 Vascular consult prn	1-2 mos (by specialist)

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## Lower Extremity Assessment – High Risk

- If one or more high risk conditions
  - ↳ Evaluate more frequently, refer to specialist
  - ↳ + Neuropathy- examine each visit
  - ↳ Multidisciplinary care important
    - Vascular specialist
    - Podiatrist
    - Orthotist
    - Certified Wound Ostomy Continence Nurse
    - Podorthist
    - Neurologist
    - Pain specialist
    - Endocrinologist
    - Advanced Practice Diabetes Specialists

ADA – Strds of Care 2008

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## Onychomycosis

- Chronic Infection 50% of nail problems
- We treat on skin but reluctant in nails
- Mean duration of > 10 years
- Rarely resolves spontaneously
- Spreads to other nails, skin, other people
- May be source of more serious infections
- Affects quality of life
- Vicks Vapor Rub?



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## Patient Education

- Proper footwear – no going barefoot, even indoors
- Daily foot inspection – look between toes and on sole of foot
- Prompt reporting of any foot lesions, discolorations or swelling

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## Thank You



[www.DiabetesEd.net](http://www.DiabetesEd.net)

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