RAMON ORTIZ CARRASQUILLO

THE THYROID AND THE CARDIOVASCULAR SYSTEM

Disclosure

- Ramon Ortiz Carrasquillo MD,FACP,FACE
  - Has received honorarium as Speaker &/or Consultant for the following Pharmaceutical Companies: Abbott, Amylin, Astra Zeneca, BMS, GSK, Janssen, Lilly, MSD, Novartis, Novo Nordisk, Roche,Pfizer, Sanofi-Aventis, Shering Plough, Takeda
  - Has received Grants &/or has contractual relationship as Principal Investigator for the following Pharmaceutical Companies: Abbott, Astra-Zeneca, BMS, Elcelyx, Lilly, MSD,Novartis, Novo Nordisk, Roche, Takeda

THE THYROID GLAND AND THE HEART SHARE A CLOSE RELATIONSHIP THAT ARISES IN EMBRYOLOGY

IN ONTOGENY, THE THYROID AND THE HEART ANLAGE MIGRATE TOGETHER

- THYROID HORMONES HAVE INTIMATE RELATIONSHIP WITH CARDIAC FUNCTION
- UBIQUITOUS EFFECTS OF THYROID HORMONE ON THE MAJOR COMPONENTS OF THE ENTIRE CIRCULATORY SYSTEM: THE HEART,THE BLOOD VESSELS AND THE BLOOD
- SOME OF THE MOST SIGNIFICANT CLINICAL SIGNS AND SYMPTOMS OF THYROID DISEASE ARE CARDIAC IN NATURE

Objectives

- Cellular mechanisms of thyroid hormone action
- Thyroid hormone effects on the heart
- Effects of TH on cardiovascular hemodynamics and vasculature
- Effects of TH on BP regulation
- Clinical manifestation of thyroid disease from a cardiovascular perspective
- Changes in thyroid hormone that arise from heart disease

Physiological actions of thyroid hormones

- Regulates nuclear transcription of genes for protein synthesis
- Increase celular metabolism and growth rates
- Facilitates neural differentiation and mental processes
- Stimulates carbohydranated fat metabolism
- Decreases cholesterol, phosphplipids, and TG's
- Decrease body weight
- Increase heart rate, respiration, and muscle tone

Cellular mechanism of thyroid hormone action

- Thyroid gland secretes mainly (85%) T_4 which is converted to T_3 by 5'monodeiododinase in various tissues
- The heart relies mainly on serum T_3 as there is no myocyte deiododinase
- T_3 binds to thyroid hormone nuclear receptors
- TRs bind to thyroid hormone response elements in the promoter region of positively regulated genes and mediate the induction of transcription
- TRs bind to TREs in the presence or absence of ligand
- While bound to T_3 TRs induce transcription and in the absence of T_3 they repress transcription

Physiological actions of thyroid hormones

- TR ALPHA IS THE PREDOMINANT TR IN THE HEART AND IS THE PREDOMINANT SUBTYPE THROUGH WHICH T_3 BINDS TO NUCLEAR TR'S
- T_3 ACTIVATION OF SIGNALLING PATHWAYS INITIATES CHANGES IN GENE EXPRESSION WHICH ARE COMPATIBLE WITH THE PHYSIOLOGIC EFFECTS OF THYROID HORMONES

Thyroid hormone effects on the myocyte

- Genomic
- Regulation of the expression of key structural and regulatory genes:

  myosin heavy chain genes which encode the 2 contractile proteins

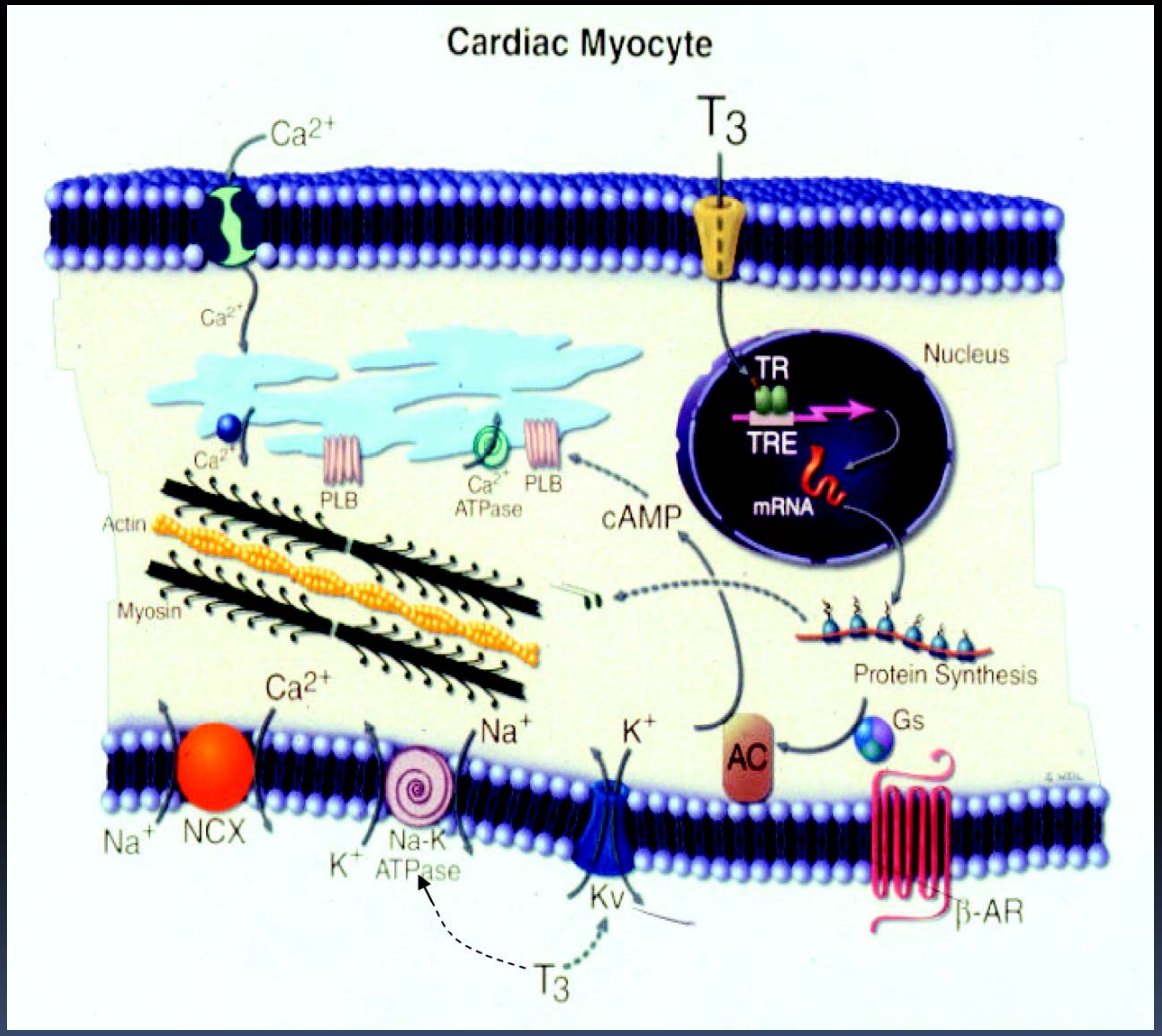
  sarcoplasmic reticulum Ca+ ATPase and its inhibitor phospholamban which regulate intracellular calcium cycling

  Beta adrenergic receptors

  sodium ATPase

Thyroid hormone effects on the myocyte

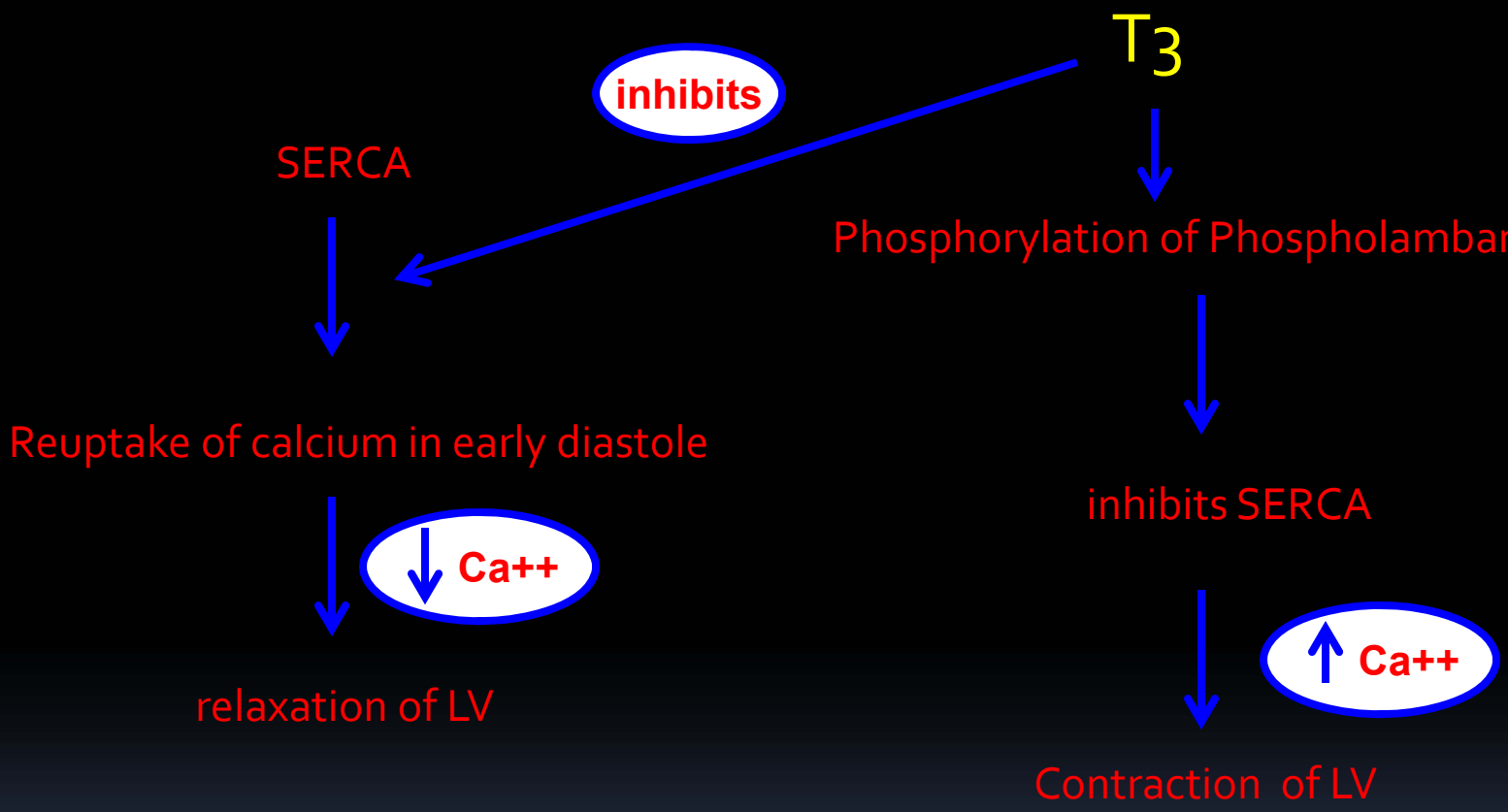
- Nongenomic
- Changes in various membrane ion channels for sodium, potassium and calcium
- Effects on actin polymerization
- Effects on adenine nucleotide translocator 1 in the mitochondrial membrane
- Intracellular signalling pathways in the heart and vascular smooth muscle cells



Irwin Klein, and Sara Danzi Circulation. 2007;116:1725-1735

CARDIAC CONTRACTION

SERCA PHOSPHOLAMBAN SYSTEM



Cytosolic Calcium

increase -- contraction

decrease-- relaxation

THYROID HORMONES EFFECTS ON THE HEART

- PROMOTE BOTH PHYSIOLOGICAL AND PATHOLOGICAL MYOCARDIAL HYPERTROPHY
- REGULATION OF INTRACELULAR CALCIUM IMPORTANT FOR BOTH NORMAL SYSTOLIC AND DIASTOLIC FUNCTION
- THE SPEED OF DIASTOLIC RELAXATION IS MARKEDLY INFLUENCED BY LOWERING OF THE CALCIUM LEVELS

- The pacemaker-related genes, are transcriptionally regulated by thyroid hormone.
- Stimulation of -adrenergic receptors accelerates diastolic depolarization and increases heart rate.

Thyroid hormone-responsive genes

- Sarcoplasmic reticulum Ca2+ ATPase and its inhibitor phopholamban which regulate the uptake of calcium into the sarcoplasmic reticulum during diastole
- Alpha-myosin heavy chain(fast myosin)and beta-myosin heavy chain(slow myosin)
- Ion channels which coordinate the electrochemical responses of the myocardium

-Ion channels sodium potassium ATPase

-Voltage gated potassium channels

-Sodium calcium exchanger

Non genomic effects of T3 on CVS

- Occur rapidly do not involve TRE-mediated trancriptional events

  changes in various membrane ion channels for Na,K,&Ca
- Effects on:

  -Actin polimerization

  -Adenine nucleotide translocator 1 in mitochondrial membrane

  -Variety of intracelular signaling pathways in heart & vascular smooth muscle

Thyroid hormone effects on CV hemodynamics

- Decreased resistance of peripheral arterioles through a direct effect on VSM and decreased mean arterial pressure
- Activation of RAS with renal sodium absortion
- Increased erythropoeitin synthesis and thus increased in red cell mass
- Increased in blood volume and preload:
- in hyperthyroidism CO 50%-300% higher
- in hypothyroidism CO 30%-50% lower

Thyroid hormone effects on the vasculature

- T_3 EXERTS DIRECT EFFECTS ON VASCULAR SMOOTH MUSCLE CELLS TO PROMOTE RELAXATION
- T_3 DOSE-DEPENDENTLY REDUCES EXPRESION OF ANGIOTENSIN II TYPE 1 RECEPTOR AND REDUCES THE INCREASED CA++ AND CONTRACTILE RESPONSE TO ANG II
- T_3 STIMULATES NO PRODUCTION VIA ACTIVATION OF THE Enos SIGNALING PATHWAY
- T_3 ALSO PROMOTES ANGIOGENESIS AND INCREASES THE DENSITY OF SMALL ARTERIOLES INCLUDING CORONARY ARTERIOLES; THIS EFFECT MAY BE SPECIALLY IMPORTANT FOLLOWING MYOCARDIAL ISCHEMIA AND IN THE PROCESS OF MYOCARDIAL ISCHEMIC RECONDITIONING

Thyroid hormone effects on natriuretic peptides and erythropoietin

- Expression for each gene of the natriuretic peptides (ANP and BNP)is regulated by thyroid hormone
- Natriuretic peptides are secreted by cardiomyocytes
- They regulate salt and water balance and play a role in the regulation of the BP
- They are altered in disease states that affect cardiac function (AF;CHF)
- Thyroid hormones also regulate erythropoeitin concentration (anemia in hypothyroidism ;no increased hb in hyperthyroidism due to increase in blood volume)

Thyroid hormone effect on RAS

- Thyroid hormones acts first to lower SVR which causes mean arterial pressure to decrease
- In response to a decreased in mean arterial pressure the RAS is activated and renin secretion is increased thus:
- Increased levels of ANG1 and 2, ACE and aldosterone
- Therefore whereas thyroid hormone decreases SVR and afterload it increases renin and aldosterone while increasing blood volume and preload and increase in CO

Thyroid hormone effects on BP regulation

- Bp is altered across the entire spectrum of thyroid function
- Changes are similar to physiological response to exercise

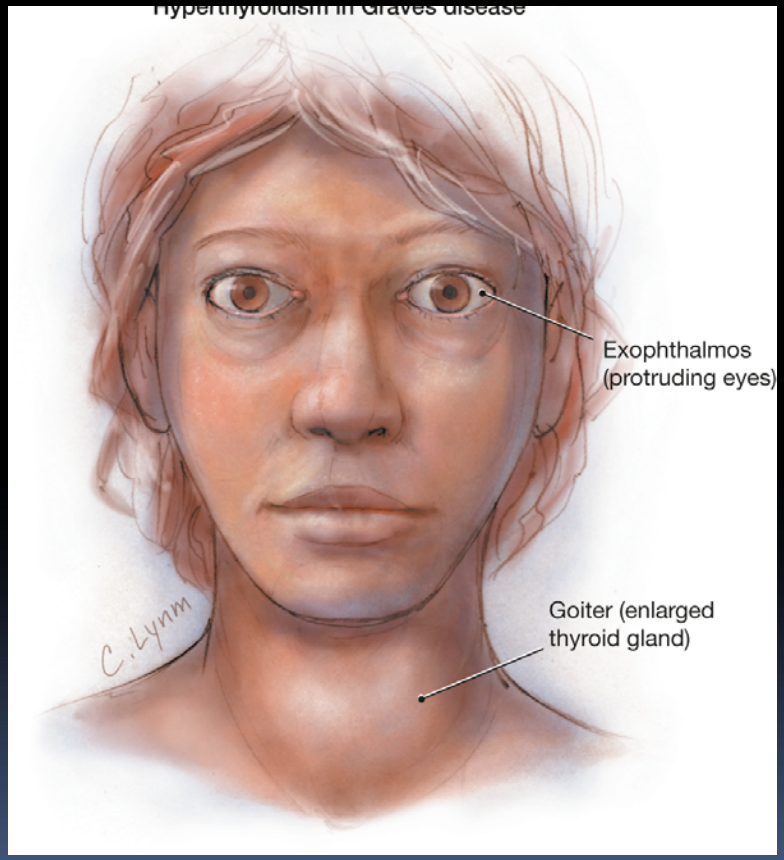
- Hyperthyroidism:
  -Widened pulse pressure
  -Increased arterial stiffness →isolated syst.HTN
  -Low SVR

- Hypothyroidism:
  -Endothelial dysfunction
  -Impaired VSM relaxation →↑SVR→diast.HTN

CARDIOVASCULAR HEMODYNAMICS

- Thyroid hormone effects on the heart and peripheral vasculature include
  - decreased SVR and
  - increased resting heart rate,
  - Increase in left ventricular contractility, and
  - blood volume

HYPERTHYROIDISM



HYPERTHYROIDISM

- Increases in
  - heart rate
  - cardiac contractility,
  - systolic and mean pulmonary artery pressure,
  - cardiac output, diastolic relaxation, and
  - myocardial oxygen consumption
- Reductions in
  - systemic vascular resistance and
  - diastolic pressure

HYPERTHYROIDISM

- CLINICAL SYMPTOMS INCLUDE SYSTOLIC HYPERTENSION, INCREASED LEFT VENTRICULAR MASS,EXERCISE INTOLERANCE, ANGINA PECTORIS AND SYSTOLIC MURMURS
- COMPLICATIONS INCLUDE ATRIAL FIBRILATION WITH ITS RISK OF STROKE AND HIGH OUTPUT HEART FAILURE
- THERE MAY BE CARDIOMYOPATHY WHEN THERE IS SUSTAINED RAPID VENTRICULAR RESPONSE ATRIAL FIBRILATION

Clinical features

- Tachycardia, at rest, during sleep, and exaggerated during exercise.
- Palpitations – tachy/forceful cardiac contractility
- Hyperdynamic precordium.
- Systolic hypertension with widened pulse pressure
- Exertional dyspnea, which is due to respiratory and skeletal muscle weakness

HF in hyperthyroidism

- Paradoxical finding of heart failure in the presence of increased contractility and CO
- Exagerated sinus tachycardia or AF can produce LV dysfunction and HF
- Preexistence of IHD or HCVD
- MVP may cause LA enlargement and AF
- High prevalence of pulmonary HTN which produce same similar signs
- Exercise intolerance and exertional dyspnea may be due to decreased pulmonary compliance or decreased respiratory and skeletal muscle function

Subclinical hyperthyroidism

- Low or undetectable serum TSH with normal T_4 and T_3
- May have no clinical signs or symptoms
- Prevalence increased with age
- Low TSH is associated with increased risk for CV mortality and AF
- Treatment is controversial
- Older patients with MNG or GD should be treated especially if they are deemed to be at risk for CV disease

Subclinical Hyperthyroidism

Factor	TSH (?314#pX2D,	TSH (3140317pX2D,
Age A#98#|hduv	Yes	Consider treating
Age ?#98#z frp rue1		
Heart disease	Yes	Consider treating
Osteoporosis	Yes	Consider treating
Menopausal	Yes	Consider treating
Hyperthyroid sxs.	Yes	Consider treating
Age ?#98 |hduv# dv|p swrp dwlf	Consider treating	Observe

RHYTHM

- Atrial tissue is very sensitive to the effects of thyroid hormone .
- More
  - APCs,
  - non-sustained SVT,
  - VPCs,
- Reduced heart rate variability

ATRIAL FIBRILATION

- PREVALENCE: 2-20%
- INCREASED PREVALENCE WITH AGE(15% IN PTS. OVER 70 YEARS
- ONLY 1% DUE TO OVERT HYPERTHYROIDISM
- TREATMENT OBJECTIVES ARE RATE CONTROL,PREVENTION OF THROMBOEMBOLISM AND RESTORATION OF SINUS RHYTHM
- CHADS2 AND CHA2DS2-VAS SCORE PROVIDE USEFUL GUIDELINES FOR DETERMINING PROPHYLACTIC ANTICOAGULATION

Atrial fibrilation in hyperthyroidism

- 40,628 pts. In danish national registry: 8.3% developed AF; increased risk found with male sex, valvular heart disease, or CHF
- Subclinical hyperthyroidism carry same RR
- Tsh a most in new onset AF

AF in hyperthyroidism: treatment

- Beta adrenergic blockade by beta 1 selective or non selective agents
- Rapid restoration of euthyroid state: atd or radioiodine
- Calcium channel blockers (avoid parenteral as it may lead to hypotension and cv collapse through effects on the smooth muscle cells)
- Digitalis may need higher dose with less predictablle response as there is a higher rate of clearance and decreased sensitivity

AF in hyperthyroidism: prognosis

- Majority revert to sinus rhythm within 2-3 months of succesful treatment with ATD or RI
- If AF persists after chemical euthyroidism is achieved electrical or pharmacologic cardioversion should be attempted
- Majority can be restored to sinus rhythm and will remain so for a prolongued period of time
- Older pts. with AF of longer duration are less likely to revert

AF in hyperthyroidism: anticoagulation

- Risk for systemic embolization
- Advancing age rather than the presence of AF was a major risk
- In younger pts. with hyperthyroidism in absence of other independent risk factors for embolization the benefits of anticoagulation may be outweighed by the risk
- CHADS2 and CHA2DS2VAS provide useful guidelines for determining prophylactic anticoagulation

HYPOTHYROIDISM

Hypothyroidism and the Heart

- Hypertension (Diastolic)
- Diastolic Dysfunction
- Elevated Cholesterol*
- Long Q-T Syndrome
- Serum CK Elevation (*Statin Hazard?)
- Coagulopathy

Hypothyroidism

- Common CV signs and symptoms
- Bradycardia
- Mild hypertension (diastolic)
- Narrowed pulse pressure
- Cold intolerance
- fatigue

Hypothyroidism: CV molecular mechanism

- Decreased expression of sarcoplasmic reticulum Ca^{2+} ATPase
- Increased expression of phospholamban (inhibitor of SR Ca^{2+} ATPase)
- Slowing of the isovolumic relaxation phase of diastolic function

Mechanism

- Myxoedematous deposits within the myocardium.
  - Decreased activity of the sympathetic nervous system.
  - Effects on the myocardium of reduced levels of thyroxine (i.e. reduced inotropy/chronotropy)

HYPOTHYROIDISM

- MOST COMMON CARDIOVASCULAR MANIFESTATIONS ARE DIASTOLIC HYPERTENSION, SINUS BRADICARDIA FAILURE OF THE SINUS NODE TO ACCELERATE NORMALLY UNDER CONDITIONS OF STRESS SUCH AS CAUSED BY FEVER INFECTION OR HEART FAILURE
- OTHERS ARE HEART BLOCK PERICARDITIS PERICARDIAL EFFUSION AND RARELY TAMPONADE
- CAD WHICH MAY BE PRE-EXISTENT OR BE AGGRAVATED BY IT ESPECIALLY AS PVR INCREASES; GREAT CAUTION IS NEEDED IN TREATING THESE PATIENTS (GO SLOW AND GO LOW)
- IN HYPOTHYROID PATIENTS WITH UNSTABLE ANGINA,MAIN LEFT ANTERIOR DESCENDING CORONARY ARTERY DISEASE, TRIPLE VESSEL DISEASE WITH IMPAIRED LEFT VENTRICULAR FUNCTION ANGIOPLASTY OR CABG MERIT CONSIDERATION BEFORE THYROID HORMONE REPLACEMENT
- TYPICAL EKG CHANGES THAT CAN BE SEEN IN HYPOTHYROIDISM ARE SINUS BRADICARDIA, PROLONGUED QTc AND RARELY AV BLOCK

Hypothyroidism: CV risks

- Impaired cardiac contractility with decreased CO and diastolic function
- Increased systemic vascular resistance
- Decreased endothelial derived relaxation factor
- Increased seum cholesterol
- Increased C reactive protein
- Increased homocysteine
- accelerated atherosclerosis
- Increased risk of CAD increased risk of stroke
- Prolongation of QT interval with underlying arrythmias
- Protein rich pericardial and or pleural effusion

Hypothyroidism: Rx in cardiac disease

- In young adults full replacement dose of l-thyroxine of 1.6mcg/kg/d can be started at outset
- In older patients: start low 25-50mcg and go slow increasing dose q 6-8 weeks
- There is predictable improvement in thyroid and and CV functional measures
- Concerns that restoration of the heart to an euthyroid state might adversely affect underlying IHD are largely unfounded
- Patients with atherosclerotic heart disease more often improve rather than worsen with treatment

HYPOTHYROIDISM

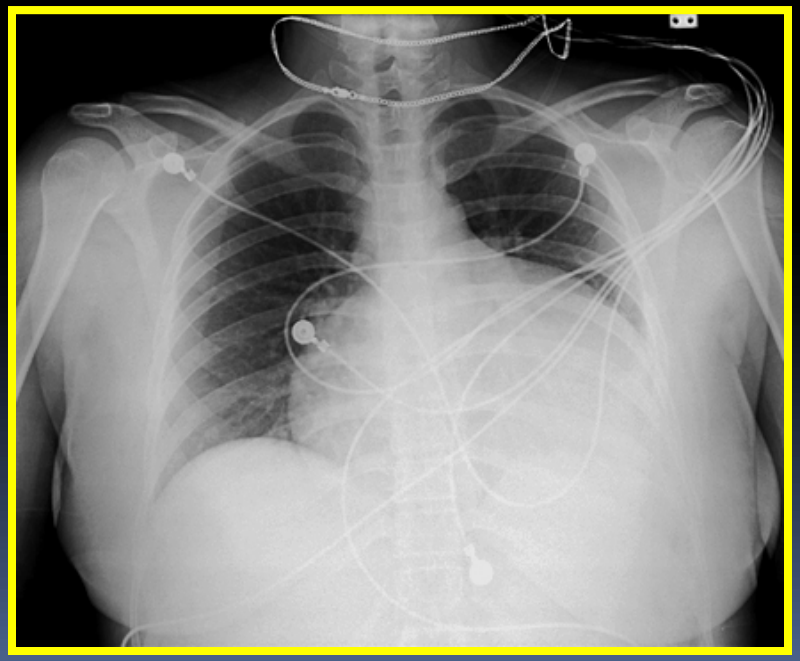
- Major cardiovascular changes
  - decrease in cardiac output
  - decrease in cardiac contractility
  - reduction in heart rate
  - increase in peripheral vascular resistance.
- Others
  - Hypercholesterolemia ,
  - diastolic hypertension,
  - carotid intimal media thickness

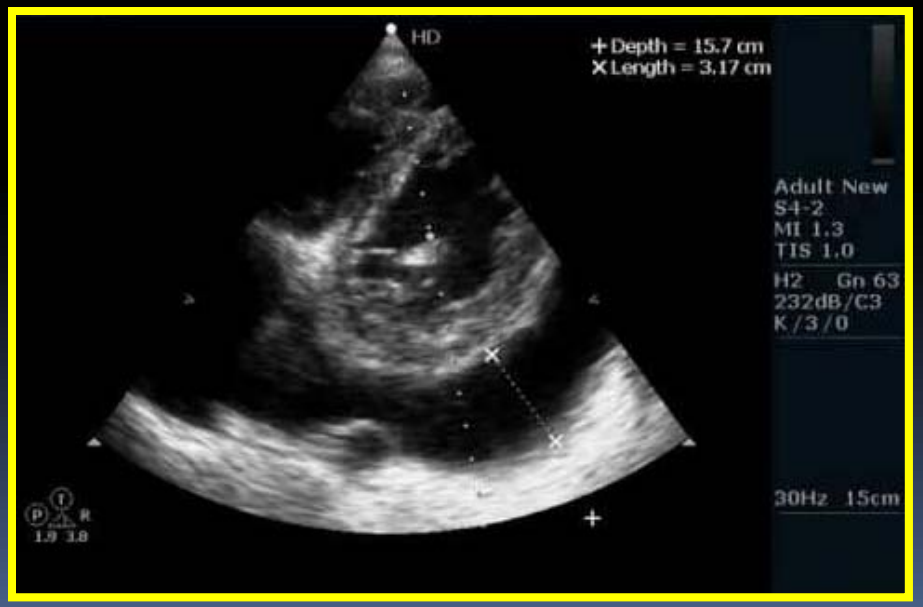
- CLINICAL MANIFESTATIONS —
- Exertional dyspnea and exercise intolerance -due to skeletal muscle dysfunction.
- Cardiac dysfunction with poor contractility, dilatation
- Edema, often nonpitting

Rhythm

- Bradycardia
- Low QRS voltage
- Widespread T-wave inversions (usually without ST deviation)
- QT prolongation-rarely Torsedes
- First degree AV block
- Interventricular conduction delay

PERICARDIAL EFFUSION





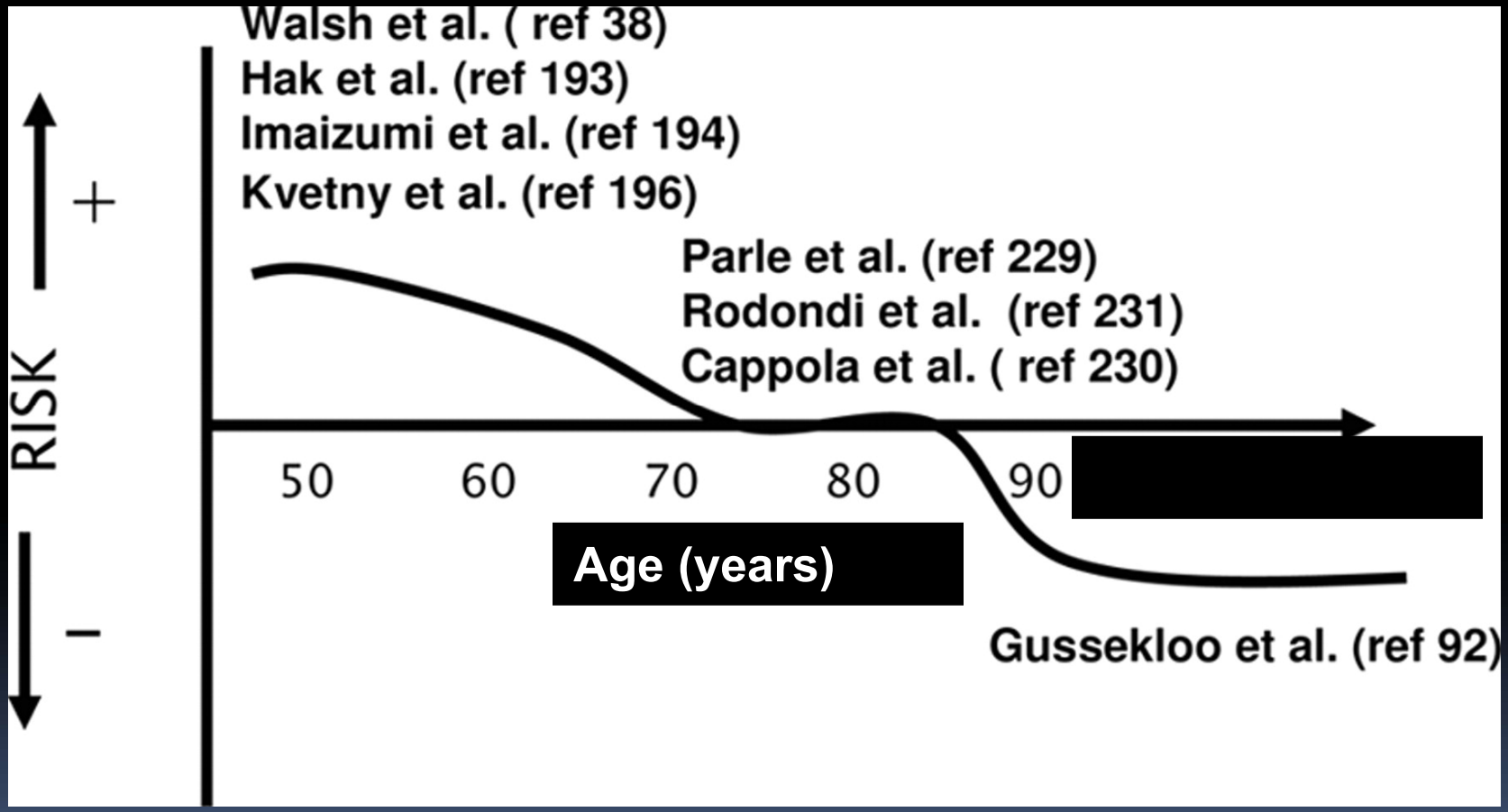
Subclinical hypothyroidism

- Affects 7-10% of older women
- Frequently asymptomatic but many have sxs. of hypothyroidism
- Occasionally associated to increased chol and CRP
- Risk of atherosclerosis, CAD, MI increased
- The benefits of restoration of TSH levels to normal can be considered to outweigh the risk

USE OF LEVOTHYROXINE TO TREAT SUBCLINICAL HYPOTHYROIDISM IS CONTROVERSIAL

Subclinical Hypothyroidism

Impact on Ischemic Heart Disease Events



BIONDI, COOPER ENOCR REV 2008 FROM LADENSON

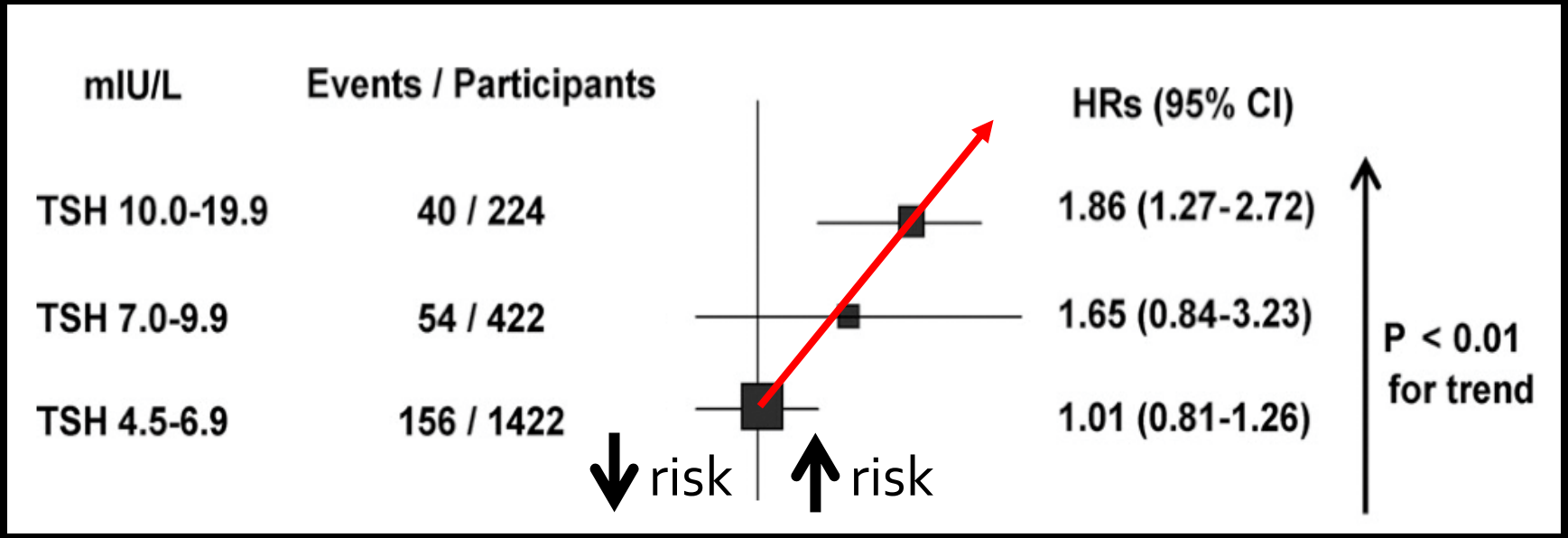
- Study from the U.K. General Practitioners data base showed that treatment of TSH levels between 5 and 10 mIU/mL lowered the incidence of ischemic heart disease events and cardiovascular mortality in patients younger than 70 years.

Best to Date NON RCT--Observational: Benefit of Treatment?

- **Cleveland Clinic**: high risk ASCVD Clinic (TSH 6.1-10 and >10) who were under 65 yrs old and not treated with LT4 had higher all-cause mortality

Arch IM 2012
McQuade, Thyroid 2011

Heart Failure Events by TSH



Until RCTs performed, data favors treating younger, higher TSH values (>10)

Gencer Circulation 2012; 126:1040

SUBCLINICAL HYPOTHYROIDISM METANALYSES CHD and Mortality

- Ten studies evaluating Subclinical Hypothyroidism
  - CHD RR 1.2
    - Older than 65 : LOWER: RR (0.98-1.26)
    - Younger than 65 : HIGHER: RR (1.09.-2.09)
  - Conclusion: May increase risk of CHD, particularly in younger than 65

Ochs, AIM, 2008

Trust trial

- Double blind, randomized placebo controlled, parallel-group trial of 737 pts. over 65 y/o with subclinical hypothyroidism
- ½ pts. assigned to levothyroxine 25 or 50 mcg the other ½ to placebo
- Change in Hypothyroidism Symptom score and Tiredness score at 1 year

- Although there was a change in TSH there was no apparent benefits in older patients with subclinical hypothyroidism in primary or secondary outcomes

Treatment of TSH between 5 and 10? Depends…

R16. Treatment should be considered particularly if they have symptoms suggestive of hypothyroidism, positive TPO antibodies or evidence of atherosclerotic cardiovascular disease, heart failure or have associated risk factors for these diseases.

Grade B, BEL 1; evidence not fully generalizable to stated recommendation and there are no prospective, interventional studies.

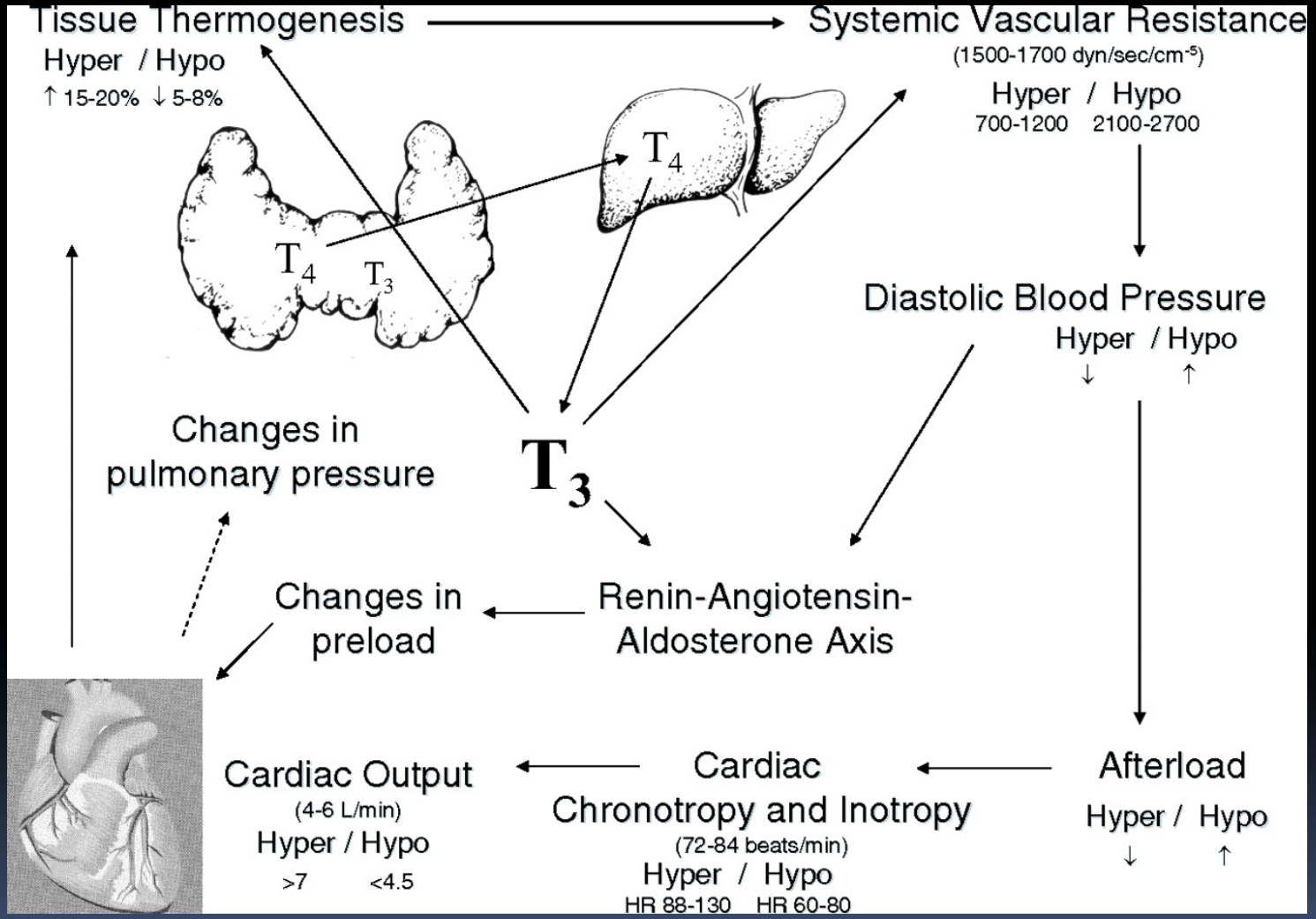
Vanderpump MP et al. 1995 Clin Endo 43:55-68 **(EL2).** Vanderpump MP & Tunbridge WM 2002 Thyroid 12:839-47 **(EL4).** Hollowell JG et al. 2002 JCEM 87:489-99 **(EL1).** Huber G et al. 2002 JCEM 87:3221-26 **(EL2).** McQuade C et al. 2011 Thyroid 21:837-43 **(EL3).** Ochs N et al. 2008 Ann IM 148:832-45 **(EL1).**

Treatment of TSH levels > 10 is recommended

R15. Patients whose serum TSH levels exceed 10 mIU/L are at increased risk for heart failure and cardiovascular mortality, and should be considered for treatment with L-thyroxine.

Grade B, BEL 1; not generalizable and meta-analysis does not include prospective interventional studies.

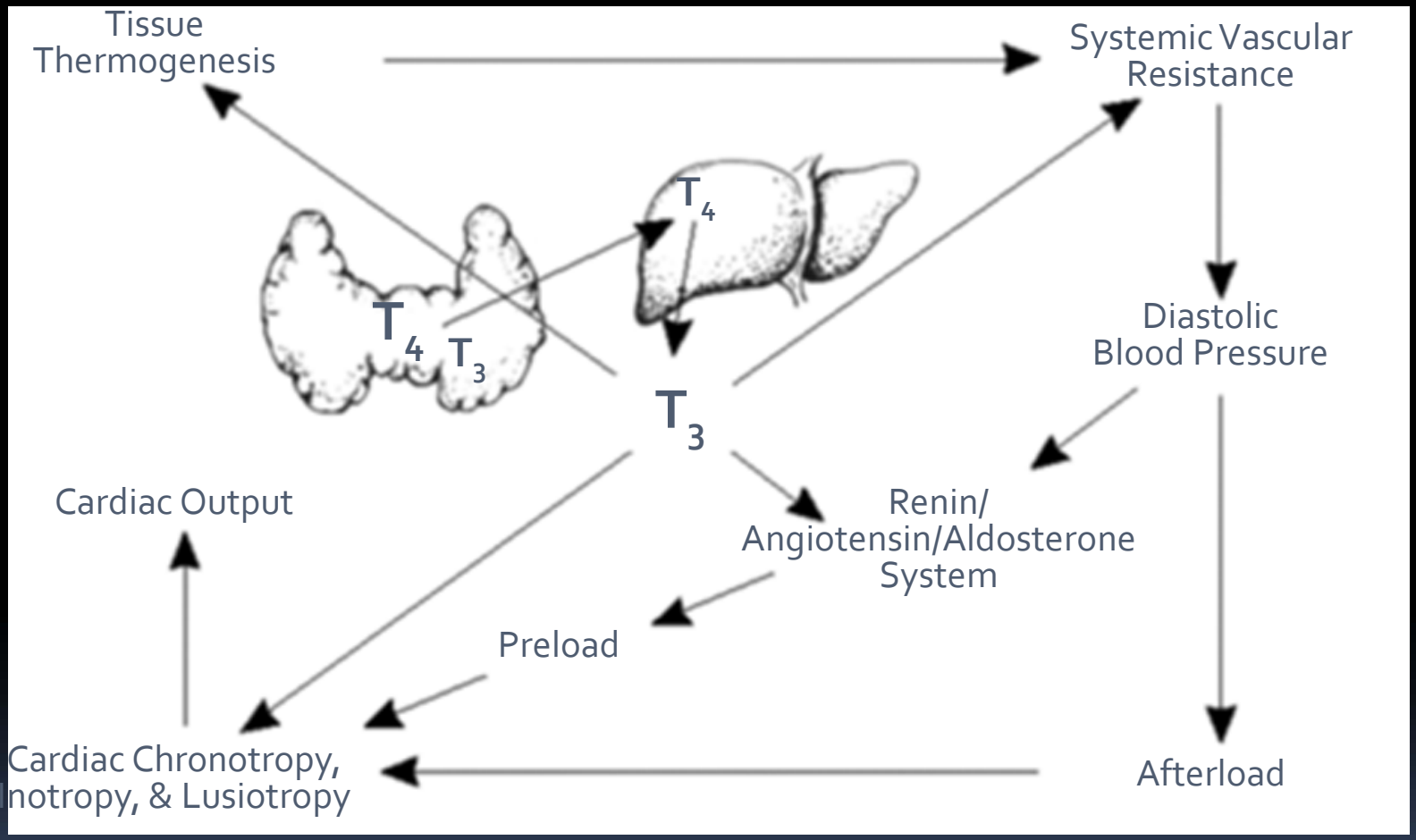
Surks et al. 2004 JAMA 291:228-38 **(EL4).** Rodondi N et al. 2010 JAMA
304:1365-74 **(EL2).** Razvi S et al. 2010 JCEM 95:1734-40 **(EL3).**
Gencer B et a.2012 Circulation Epub before print **(EL1).**



Irwin Klein, and Sara Danzi Circulation. 2007;116:1725-1735

YUN1

Sites of Cardiac Action of Thyroid Hormone



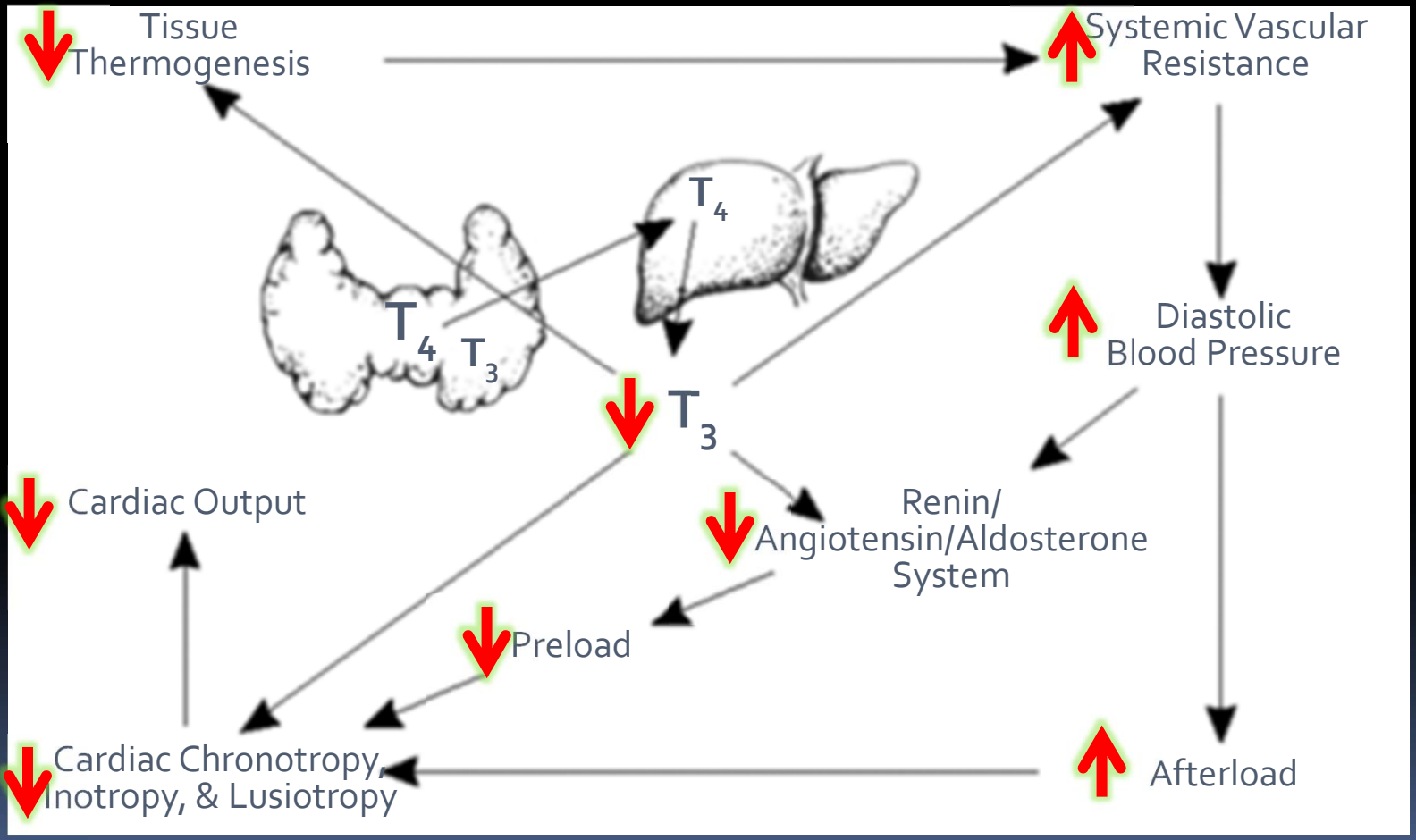
based on Klein and Danzi, In: *The Thyroid* 2004

Slide 60

YUN1 Your User Name, 3/10/2013

YUN2

Sites of Action of Thyroid Hormone on the Heart with Hypothyroidism



based on Klein and Danzi, In: *The Thyroid* 2004

Slide 61

YUN2 Your User Name, 3/10/2013

Thyroid disease and pulmonary hypertension

- High prevalence of pulmonary HTN and AV valve regurgitation in hyperthyroidism
- Effect of TH to ↓ SVR may not occur in pulmonary vasculature
- Primary pulmonary HTN(pulmonary artery pressure ➢25mmhg at rest and ➢30mmhg during exercise)
- Often unknown origin
- A link to thyroid disease has been identified
- Thyroid disease should be considered in Differential Dx. of primary pulmonary hypertension

Thyroid hormone effects on lipid metabolism

- ↓ fractional clearance of LDL by liver

  -Decreased no. of LDL receptors

  -Decreased LDL receptor activity
- ↓ catabolism of cholesterol into bile

  -T3 negatively regulates liver specific enzyme cholesterol 7 alpha hydroxylase

In overt and occasionally subclinical hypothyroidism there is ↑ cholesterol, ↑ LDL and ApoB (90%)

Prevalence of overt hypothyroidism in pts. with hypercholesterolemia is 1.3%-2.8%

Hyperthyroidism: CV signs and symptoms

palpitations	Anginal chest pain
Exercise intolerance	Atrial fibrilation
Exertional dyspnea	Cardiac hypertrophy
Systolic hypertension	Peripheral edema
Hyperdynamic circulation Cardiac output ↑ by By combined effect Blod volume and EF Cerebrovasc. isch sx. In young pts w GD	Congestive heart failure 50-300% of normal of ↑HR, contractility With ↓ in SVR have been reported

HYPERTHYROIDISM: CV signs and symptoms

- Exercise intolerance
- Exertional dyspnea
- Palpitations
- Systolic hypertension
- Hyperdynamic circulation
- Cardiac output ↑ by 50-300% of normal by combined effect of ↑ HR, contractility, blood volume and EF with ↓ in SVR
- Atrial fibrilation
- Anginal chest pain
- Cardiac hypertrophy
- Peripheral edema and CHF
- Cerebrovascular ischemic symptoms have been reported in young patients with GD

Thyroid hormones and Heart Failure

Heart disease and Thyroid function

- Approximately 30% of patients with CHF have low T3 levels
- Reduction of T3 is proportional to the severity of HF
- Reduced serum T3 is a strong predictor of all cause and CV mortality and in fact is a stronger predictor than age,LV EF, or dyslipidemia

- HEART FAILURE LEADS TO DOWN REGULATION OF THE THYROID HORMONE SIGNALING SYSTEM IN THE HEART: DECREASES OF NUCLEAR TR LEVELS IN ADDITION SERUM LEVELS OF T_4 AND T_3 ARE DECREASED
- OVERALL IT APPEARS THAT IN HEART FAILURE A HYPOTHYROID CARDIAC STATE MAY OCCUR DUE TO DECREASED TR LEVELS IN FAILING HEARTS
- DATA FROM CLINICAL STUDIES INDICATE THAT THYROID HORMONE REPLACEMENT IN PATIENTS WITH HEART FAILURE HAS BENEFICIAL EFFECTS ON CARDIAC CONTRACTILE FUNCTION

OTHER CARDIAC CONDITIONS RELATED TO THYROID DYSFUNCTION

- ATHEROSCLEROSIS
- ATRIAL FIBRILATION
- PERCARDITIS, PERICARDIAL EFFUSION AND CARDIAC TAMPONADE
- SINUS BRADYCARDIA AND TACHYCARDIA
- ATRIOVENTRICULAR BLOCK
- TORSADE DE PONTES VENTRICULAR TACHYCARDIA
- LEFT VENTRICULAR SYSTOLIC AND DIASTOLIC DYSFUNCTION
- HEART FAILURE, CARDIOMYOPATHY, HIGH OUTPUT CONGESTIVE STATE MITRAL VALVE PROLAPSE
- ENDOTELIAL DYSFUNCTION
- SYSTOLIC AND DIASTOLIC HYPERTENSION

Summary

- Thyroid dysfunction virtually affects the whole spectrum of cardiovascular hemodynamics
- Thyroid functional abnormalities can cause a range of cardiovascular signs and symptoms and Cardiovascular disease are also associated with derangements of thyroid functions
- Restoration of normal thyroid function often reverses the abnormal cardiovascular hemodynamics

When the Thyroid speaks….the Heart listens”

Thank You