



TIROIDE E SCOMPENSO CARDIACO



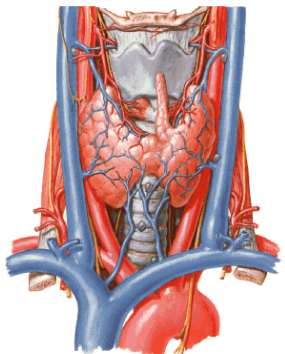
Bari,
7-10 novembre 2013

QUESTIONS & ANSWERS

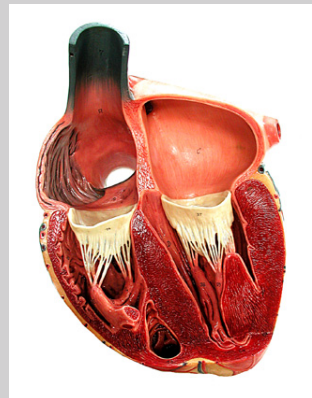
Massimiliano Andrioli

EndocrinologiaOggi, Roma

Endocrinologia, IRCCS, Istituto Auxologico Italiano, Milano



*e*ndocrinologiaoggi



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HYPOTHYROIDISM

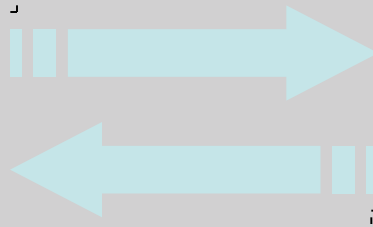


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HYPERTHYROIDISM





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T3 availability controlled by deiodosinases

T3 fundamental for cardiac morphology and performance

Non-genomic effects

Genomic effects: TR ($\alpha 1$, $\alpha 2$, $\beta 1$, $\beta 2$)

TR β required for angiogenesis in cardiac development

“Foetal gene program” (lowT3): \uparrow cells proliferation, \downarrow differentiation

Restored in CHF, hypothyroidism, lowT3



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LOW T3 SYNDROME

20-30% of HF lowT3 in NYHA grado elevato

Low T3 syndrome leads to changes in cardiac function and gene expression similar to hypothyroidism

Low T3 is an adaptative response in HF.

It should be treated? Why? How? New drugs?



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HYPERTHYROIDISM

↑ Cardiac output, ↑ Fc, ↑ preload, ↓ resistance,
hyperdynamic circulation

↓ Renal perfusion, ↑ renin/aldo

↑ Oxygen demand, ↑ energy consumption, ↓ impossible
adequate output during exercise

Prolonged Hyper, hypertrophy, ↓ ventricular performance, ↑ HF



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HYPERTHYROIDISM

Both, overt and subclinical hyperthyroidism, cause HF

Why? Differences?

Increased cardiac mortality in treated hyperthyroid patients

First therapy?



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AMIODARONE

Hypothyroidism

AIT type 1, hyper, pre-existing disease, low I intake, \uparrow flow

AIT type 2, thyreotox, not pre-existing disease, IL6, no flow

AIT & impaired left ventricular ejection: high risk major CV events

Treatment?



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THYROID HORMONE RESISTANCE

Resistance to thyroid hormone (RTH): TR β mutation

TR β gene, chromosome 3, T3-binding isoforms ($\beta 1$, $\beta 2$, $\beta 3$),
hypothalamus-pituitary

TR α gene, chromosome 17, isoforms ($\alpha 1$, $\alpha 2$, $\alpha 3$), heart

Due to α predominance vs β in myocardium, possible
relative sparing of the heart in RTH

Symptoms patients RTH (\uparrow cardiac contractility, \uparrow Fc) similar
to hyperthyroidism

Others hypothyroidism (*Pulcrano JCEM 2009*).

Why? Cardiac condition in RTH?



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Thanks for your answers...