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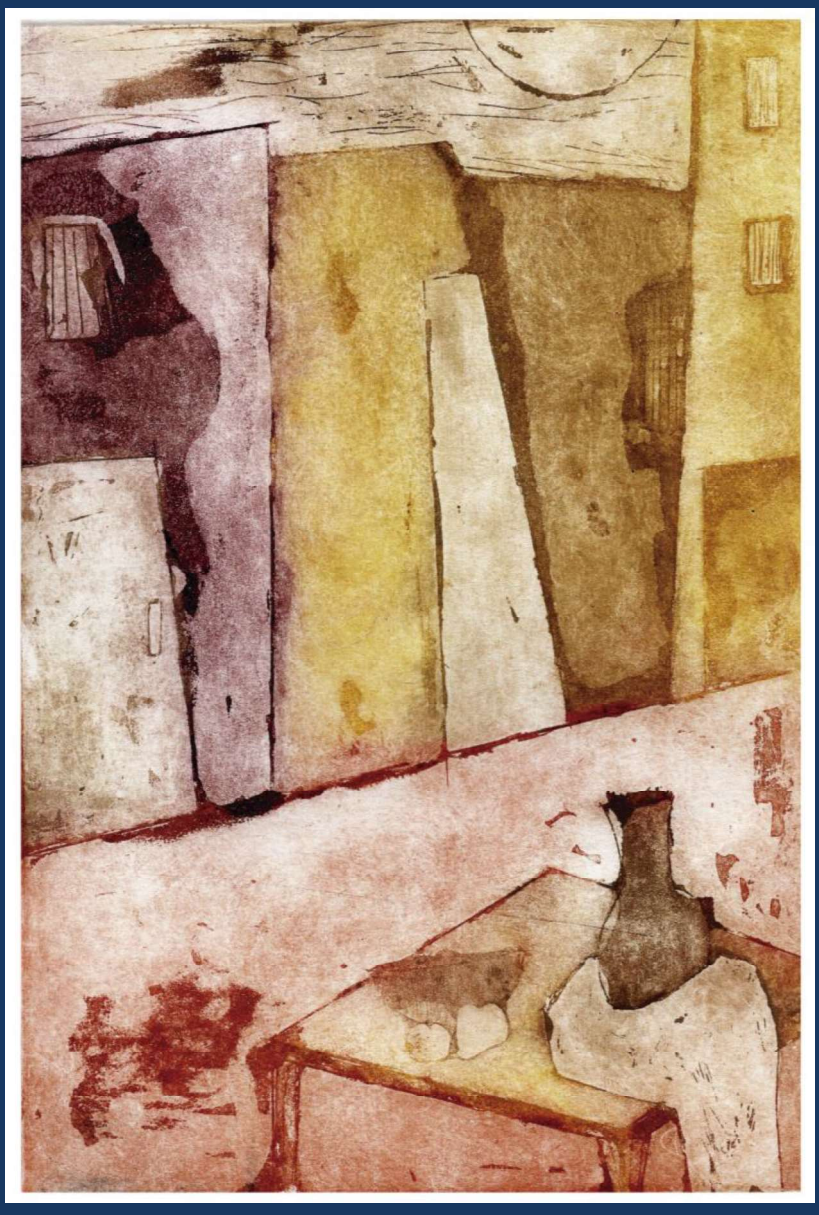

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La Tapa (Ver pág. 4)
Atardecer en la tarde
Antonella Ricagni

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(NANOMED-ar)

VI Reunión Científica Regional de la Asociación Argentina de Ciencia y Tecnología de Animales de Laboratorio (AACyTAL)

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to a decrease in plasma cholesterol levels (Cho) and has a dose-dependent effect on blood viscosity. The objective was to analyze the effect of the infusion of Lc on the lipid profile and hemorrheological parameters in patients with hypercholesterolemia. Eleven patients of both sexes (6 females, 5 males) were studied, prior to signing an informed consent. All received envelopes with dried extract of leaves and stems of Lc (2.6 gr each) and instructive to prepare the infusion to be ingested three times a week, for two months. Determinations made in blood: Total Cho, ChoHDL and ChoLDL, triglycerides (TG), hepatogram (glutamic-oxalacetic transaminase (GOT) and glutamic-pyruvic transaminase (GPT) (all by autoanalizer), relative blood viscosity (with rotational viscometer), and erythrocyte rigidity index (RI) (by filtration method), at 0 (Baseline) and 60 days (post-treatment with Lc). Results: Mean ± SEM, n= 11, B: baseline, TLc: post-treatment with Lc, *p<0.05 vs. B, ns= no significant vs. B (t-Test for paired data). Cho (mg %): B: 257 ± 11; TLc: 241 ± 8*. ChoLDL (mg %): B:168 ± 13; TLc: 158 ± 10*. ChoHDL (mg %): B: 70 ± 7; TLc: 67 ± 7, ns. Triglycerides (mg %): B: 114 ± 18; TLc: 106 ± 15, ns. Relative blood viscosity (centipoisse): B: 3.5 ± 0.3; TLc: 3.6 ± 0.4, ns. RI: B: 7.8 ± 1.1; TLc: 8.1 ± 1.2, ns. GOT (IU/l): B: 19 ± 2; TLc: 19 ± 1, ns. GPT (IU/l): B: 20 ± 2; TLc: 18 ± 2, ns. No differences between sexes. In the patients evaluated, with the analyzed dose, there were no changes in the hemorrheological parameters or alterations in transaminases. Besides, we obseved a decrease in both Cho (-10 %) and ChoLDL (-8.4 %), suggesting that an increase in the dose of Lc could lead to a marked decrease in both parameters, which would support the Lc treatment as an important tool in lipid lowering.

0185 - ACUTE TREATMENT WITH TRIIODOTHYRONINE (T3) ATTENUATES POSTISCHEMIC MITOCHONDRIAL INJURY: A RESPONSE ASSOCIATED WITH ENHANCED AMP-ACTIVATED PROTEIN KINASE (AMPK) ACTIVATION

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Abstract/Resumen: Recent studies have provided evidence that acute treatment with T3 could enhance the recovery of ischemic myocardium through the preservation of mitochondrial function and the improvement of energy substrate metabolism. To this respect, our previous results showed that T3 enhanced the activation of AMPK, a key enzyme that regulates the cellular energy metabolism, during Is-Rs, which was prevented by AMPK pharmacological inhibitor, Compound C (CC; 10 µM). During reperfusion, T3 increased contractile function recovery, mitochondrial ATP production and tissue ATP, effects that were reverted by CC. The aim of the present study was to investigate the effects produced by the acute treatment with T3 (60 nM) and AMPK inhibitor, in mitochondria of isolated rat left atria subjected to 75 min simulated ischemia (Is)–75 min reperfusion (Rs). ANOVA, followed by Tukey's test was used, n= 8/group. The results showed that mitochondrial ultrastructure, analyzed by electron microscopy, was better preserved in the atria subjected to Is-Rs in the presence T3, effect that was reverted by CC. Moreover, calcium retention capacity (CRC), defined as the amount of Ca^{2+} required to trigger a massive Ca^{2+} release by isolated mitochondria, was increased by acute treatment with T3, effect that was reverted by CC (Is-Rs: 77 ± 9, Is-Rs+T3: 114 ± 12*, Is-Rs+T3+CC: 82 ± 8 nmol/mg protein; *p<0.05). As accumulating evidence suggests that the phosphorylation and inhibition of GSK-3ß; acts as a master switch to limit the mPTP opening, improving mitochondrial recovery function, the relation between phosphorilated/total GSK-3ß was assessed. Results showed that T3 increased this relation, which was prevented by CC (Is-Rs: 1.5 ± 0.2, Is-Rs+T3: 2.2 ± 0.1*, Is-Rs+T3+CC: 1.6 ± 0.2 AU; *p<0.05). The results suggest that AMPK is involved, at least in part, in the protective effects exerted by T3 at mitochondrial level in the myocardium subjected to ischemia-reperfusion, contributing to mitochondrial structure and function preservation.

0244 - ANGIOGENESIS REGULATION: DIFFERENT MECHANISM OF ACTION ELICITED BY PROGESTERONE (PG) AND MEDROXYPROGESTERONE ACETATE (MPA)

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Abstract/Resumen: In atherosclerosis, the generation of microvessels within the plaques represents a survival option for damaged tissue but would also be associated with the instability of the plaque. The risk/benefit of hormone replacement therapy using natural or synthetic progestins such MPA as an alternative to prevent cardiovascular diseases in menopausal women is controversial. The aim of this work was to evaluate the mechanism of action by which Pg and MPA regulate angiogenesis. Tube formation assay and endothelial cell (EC) culture derived from murine aorta were used to evaluate angiogenesis. Total tube length of vessel segments was quantified using ImageJ software. Both progestogens significantly enhanced the number of tube structures (26 %; 46% above control, 100 nM Pg; 100 nM MPA respectively, p<0.05). Firstly, we tested the participation of Pg receptor (PgR). Pre-tre atment of EC with RU486, an antagonist of PgR, completely inhibited the proangiogenic effect of Pg and MPA. Considering that VEGF is the main regulator of angiogenesis, we neutralize its action with a VEGF antibody (a-VEGF). Besides, we used the compound genistein to block the tyrosine kinase activity of VEGF receptor (VEGFR). The presence of a-VEGF or genistein abrogates the proangiogenic action of Pg. Meanwhile, the effect of MPA was not modified. Nitric oxide synthase (NOS) is involved in VEGFR downstream signaling pathway. In the presence of L-NAME, a NOS inhibitor, the stimulation of tube formation induced by Pg was blunted. Meanwhile, MPA action was not affected. The proangiogenic action of Pg was not altered by the presence of platelet-rich-plasma (PRP)-derived plasma. Instead, the MPA action was potentiated (29 % vs. 100 nM MPA, p<0.05). We demonstrated that 100 nM Pg markedly increased VEGF synthesis (39 % vs. control, p<0.05). In contrast, MPA (100 nM) did not affect VEGF production. In conclusion, both progestogens promote angiogenesis with a slight different mechanism of action elicited by each steroid.

0301 - IDENTIFICATION OF CARDIOPULMONARY DISEASES BY THE PERFUSION INDEX IN THE 6-MINUTE WALK DISTANCE TEST

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Abstract/Resumen: The distance walked in the 6-minute walk distance test (6MWD) identifies healthy subjects (HS) from patients with cardiopulmonary diseases, but it can be influenced by factors independent of the underlying pathology, such as musculoskeletal disorders, frailty and lack of training. The perfusion index (PI) measures the pulsatile force at the control site and is an indirect assessment of peripheral perfusion, providing a more reliable evaluation of the response to the 6MWD. The objective of this study was to assess the ability of PI to detect patients with heart failure (HF, n= 37), pulmonary hypertension (PHT, n= 36) and chronic obstructive pulmonary disease (COPD, n= 93) from HS (n =36) in the 6MWD. O_2 saturation (O2Sat), heart rate (HR), the Borg scale (BS) and PI