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TREINAMENTO FÍSICO AERÓBIO REVERTE O EDEMA PERIFÉRICO, RESTAURA O TÔNUS MIOGÊNICO E O REMODELAMENTO VASCULAR EM ARTÉRIA TIBIAL CAUDAL DE RATOS COM INSUFICIÊNCIA CARDÍACA

Tese apresentada ao Programa de Pós-graduação em Fisiologia Humana do Instituto de Ciências Biomédicas da Universidade de São Paulo, para obtenção do Título de Doutor em Ciências.

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RESUMO

PAULA, S. M. **Treinamento físico aeróbio reverte o edema periférico, restaura o tônus miogênico e o remodelamento vascular em artéria tibial caudal de ratos com insuficiência cardíaca**. 2017. 121f. Tese (Doutorado em Fisiologia Humana) Instituto de Ciências Biomédicas, Universidade de São Paulo, São Paulo, 2017.

A insuficiência cardíaca (IC) é uma síndrome que apresenta sintomas como fadiga e intolerância ao exercício, além de sinais como edema periférico (EP). Alterações referentes à intolerância ao exercício têm sido atribuídas a modificações da musculatura esquelética (ME) e a alterações de fluxo sanguíneo, o qual se encontra atenuado na IC durante o exercício. Tem-se relatado ainda prejuízo na reatividade vascular das artérias que irrigam a ME, porém não há consenso quanto à resposta dependente do endotélio, ou quanto à constrição miogênica (CM). O prejuízo na CM acarretaria uma falha na perfusão causando aumento na pressão hidrostática capilar, levando ao surgimento de EP. Diversos estudos têm associado complicações cardiovasculares às mudanças estruturais e mecânicas arteriais, contribuindo para o agravamento da IC. Como o treinamento físico (TF) amplifica a função endotelial e a CM em indivíduos e animais saudáveis e tem reconhecido benefício sobre o sistema cardiovascular na IC, testou-se a hipótese de que a IC induz disfunção na reatividade vascular e no remodelamento em artéria tibial caudal (ATC) de ratos, e que o TF é capaz de melhorar esses parâmetros e reverter sinais como o EP. Ratos Wistar foram submetidos à oclusão da artéria coronária descendente anterior ou falso operado (SHAM). Após 4 sem., os animais foram caracterizados como IC por meio do ecocardiograma (FE≤30%) e da quantificação do BMP plasmático (≥0,3±0,02 ng/mL), e, em seguida foram divididos em sedentários (SHAMs e ICs) e treinados (SHAMt e ICt, esteira, 5 vezes/ sem., 8 sem.). O grupo ICs apresentou uma redução da distância percorrida no teste de esforço escalonado e o TF aumentou esse parâmetro em ambos os grupos. O volume da pata, avaliado por hidropletismometria, foi maior nos ICs e o TF reverteu o mesmo para o nível observado no SHAMs. Apesar da vasodilatação à acetilcolina não estar modificada na ATC dos grupos avaliados, a CM foi deflagrada a partir de 100 mmHg nos SHAMs, abolida nos ICs, amplificada nos SHAMt e restaurada nos ICt. O inibidor da ROCK (Y-27632, 1 μM) reduziu a CM em todos os grupos estudados. Ao se avaliar a mobilização de cálcio nas ATC, observou-se aumento da contração induzida por noradrenalina (10 μM) em ausência de Ca^{2+} extracelular nas ATC de ICt, indicando um aumento na cinética do cálcio intracelular (Ca^{2+}_i), sem alteração no influxo de Ca^{2+}. A expressão proteica da razão SERCA-2/Fosfolambano e da ROCK II foi aumentada nas ATC dos ICt. As ATC dos ICs apresentaram aumento do diâmetro interno (D_i) sem alteração da área de secção transversa; o TF foi capaz de restaurar o D_i para o nível observado nos SHAMs. Confirmando a presente hipótese, ATC dos ICs apresentam CM prejudicada e um remodelamento eutrófico para fora que contribuem para a geração do EP. O TF foi efetivo em restaurar a CM e o remodelamento vascular em ATC, revertendo conjuntamente o EP. Os presentes resultados também sugerem que os ajustes na CM induzidos pelo TF estão associados a uma melhora na mobilização do Ca^{2+}_i e na sensibilização das proteínas contráteis.

Palavras-chave: **Insuficiência cardíaca. Treinamento físico. Edema periférico. Resposta miogênica. Cinética do cálcio. Sensibilização ao cálcio.**

ABSTRACT

PAULA, S. M. **Aerobic physical training reverses peripheral edema, restores myogenic tone and vascular remodelling in caudal tibial artery of rats with heart failure.** 2017. 221 p. Ph. D. Thesis (Human Physiology) - Instituto de Ciências Biomédicas, Universidade de São Paulo, 2017.

Heart failure (HF) is a syndrome that presents symptoms such as fatigue and exercise intolerance, as well as peripheral edema (PE). Exercise intolerance is associated with changes in skeletal muscle (SM) and in blood flow regulation, which is attenuated in HF during acute exercise. In addition, an impairment of vascular reactivity in arteries that feed SM has also been described, but there is no consensus regarding endothelium-dependent or myogenic constriction (MC) responses. It is well known that impairment of MC would cause, under perfusion, an increasing hydrostatic pressure at capillary levels, leading to the onset of PE. Several studies have associated cardiovascular adjustments to structural and mechanical arterial changes, contributing to the worsening of HF. As physical training (PT) enhances endothelial function and MC in healthy individuals and animals and has been recognized for its beneficial effects on the cardiovascular system in HF, it was hypothesized that HF induces dysfunction in vascular reactivity and remodelling in caudal tibial artery (CTA) of rats, and that PT is able to improve these parameters and revert PE. Male Wistar rats were submitted to occlusion of coronary artery or false operated (SHAM). After 4 weeks, the animals were characterized as HF by echocardiogram (ejection fraction ≤30%) and quantification of plasma BNP (≥0.3 ± 0.02 ng/mL), and then divided into sedentary (SHAMs and HFs) and trained (SHAMt and HFt, treadmill, 5 times/ week, 8 weeks). The HFs group presented a reduction of the distance covered in the maximum exercise test and the PT increased this parameter in both groups. Paw volume, measured by hydroplethysmometry, was higher in the HFs and the PT reverted. Although vasodilation-induced by acetylcholine was not modified in the CTA among groups, MC was triggered at 100 mmHg in SHAMs, abolished in HFs, amplified in SHAMt and restored in HFt. ROCK inhibitor (Y-27632, 1 µM) reduced MC in all groups studied. Calcium (Ca^{2+}) mobilization was evaluate in CTA, noradrenaline (10 µM)-induced contraction in the absence of extracellular Ca^{2+} was higher in HFt, indicating an increase in intracellular calcium (Ca^{2+}_i) kinetics, without changes in Ca^{2+} inflow. SERCA-2/Phospholamban ratio and ROCK II protein expression were increased in the CTA of the HFs. CTA of HFs showed increase of the internal diameter (D_i) without cross section area alteration; PT was able to restore D_i to SHAMs level. In line with the present hypothesis, CTA of HFs has impaired MC and an outward eutrophic remodelling that contribute to the PE. PT was effective to restore MC and vascular remodelling in CTA and to reverse the PE. In addition, MC adjustments induced by TF are associated with an improvement in Ca^{2+} mobilization and contractile proteins sensitization.

Keywords: Heart failure. Physical training. Peripheral edema. Myogenic response. Calcium kinetics. Calcium sensitization.

1 INTRODUÇÃO

As doenças cardiovasculares representam a principal causa de morte e incapacidade no mundo (World Health Organization - WHO, 2013). No Brasil, segundo dados do Departamento de informática do Sistema Único de Saúde, aproximadamente 31% das mortes por doenças cardiovasculares resultam diretamente de infarto do miocárdio (RIPSA, 2013), o qual foi a principal causa de óbito no ano de 2014 no município de São Paulo, segundo dados da Secretaria Municipal de Saúde de São Paulo (TABNET, 2015).

O infarto do miocárdio (IM) é caracterizado por um desequilíbrio entre a demanda e o suprimento de oxigênio para o miocárdio (LEE; CANNON, 2005). Com base em fatores etiológicos, o IM pode ser classificado em espontâneo (IM tipo 1), o qual está relacionado à ruptura da placa aterosclerótica levando à embolia com necrose miocitária subsequente, ou em secundário decorrente de desequilíbrio isquêmico (IM tipo 2), o qual é observado em casos de aterosclerose fixa à parede arterial reduzindo o fluxo coronariano. Ainda o IM pode ser decorrente de vasoespasmo coronariano e/ ou disfunção endotelial, além de outras causas menos comuns (LEE; CANNON, 2005; THYGESEN et al., 2012). Como o coração não possui estoque de oxigênio e depende quase inteiramente do metabolismo aeróbio para prover suas altas taxas de consumo energético, após segundos de oclusão coronariana, a tensão de oxigênio cai rapidamente e ocorre disfunção ventricular esquerda (KERN, 2005).

A perda tecidual decorrente do IM e a disfunção do ventrículo esquerdo (VE) iniciam um processo chamado remodelamento ventricular (BOLOGNESE; CERISANO, 1999), caracterizado por alterações da geometria ventricular, as quais podem influenciar no surgimento da insuficiência cardíaca (IC) (ANQUENOT et al., 1992).

Clinicamente, a IC pode ser definida como uma síndrome na qual os pacientes apresentam sintomas típicos, como dispneia, edema pulmonar, hepático ou de membros e intolerância aos esforços físicos, e sinais como pressão venosa jugular elevada, crepitação pulmonar e batimento do ápice de VE deslocado, resultado das anormalidades estruturais e funcionais cardíacas que levaram à falha do coração em fornecer oxigênio a uma taxa compatível com os requisitos da metabolização tecidual (MCMURRAY et al., 2012).

Nesse sentido, efeitos da IC podem repercutir em diversos órgãos e tecidos como é o caso dos vasos sanguíneos, relatado em diversos estudos avaliando relaxamento dependente do endotélio mostrando prejuízo (COUTO et al., 2015; BEN DRISS et al., 2000; VARIN et al., 1999) ou manutenção da resposta vasodilatadora (BEN DRISS et al., 2000) bem como prejuízo da resposta contrátil (BEM DRISS et al., 2000; PEREIRA et al., 2005), o

que sugere relação entre a função vascular e a evolução da IC. Outro fator de regulação do fluxo sanguíneo que pode estar afetado na a IC é a resposta miogênica, como o aumento da constrição miogênica observado em artéria cerebral posterior (YANG et al., (2012) e em artéria mesentérica (GSCHWEND et al., 2003), o que sugere um distúrbio da autorregulação do fluxo sanguíneo e da regulação da resistência vascular periférica. Além das alterações na reatividade vascular, levanta-se também a questão no presente estudo sobre a presença de modificações na estrutura e mecânica vascular (remodelamento vascular) presentes na IC, uma vez que alterações hemodinâmicas, além da ativação do sistema renina-angiotensin-aldosterona (SRAA), estresse oxidativo e elevação das concentrações plasmáticas de endotelina estão presentes na IC (LEVINE et al., 1982; MCMURRAY et al., 1992; SCHIFFRIN, 2005).

Sabe-se que o treinamento físico (TF) aeróbio na IC resulta em melhora de sintomas como a intolerância ao exercício (HAMBRECHT et al, 1995; PIÑA et al, 2003), além de melhorar a função endotelial (HAMBRECHT et al., 2003; KEMI et al., 2013; VARIN et al., 1999). Porém ainda é desconhecido o efeito do TF na resposta miogênica e estrutura e mecânica vascular associados à IC após IM.

7 CONCLUSÃO

Concordante com a hipótese do presente estudo, o TF aeróbio foi capaz restaurar o prejuízo da constrição miogênica das artérias dos animais IC, amplificar a mesma das artérias dos animais SHAM, sem alterar a vasodilatação dependente do endotélio e a vasodilatação ao nitroprussiato de sódio. Os ajustes na constrição miogênica induzidos pelo TF estão associados a uma melhora na mobilização de Ca^{2+} pelo retículo sarcoplasmático, além da participação de via de sensibilização ao Ca^{2+} via ROCK. Em associação ao restauro da constrição miogênica, o TF promoveu a reversão do remodelamento para fora nas artérias tibiais caudais, associado a uma redução do volume da pata dos IC. Assim, efeitos benéficos tanto funcionais quanto estruturais observados podem vir a ser uma combinação de fatores a contribuir para a melhora do controle da perfusão muscular esquelética bem como reduzir sintomas característicos da IC como a intolerância ao exercício e sinais como o edema periférico.

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