Similarities on the permissive role of tissue oxygenation on compression- and contraction-induced hyperaemia

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Objective: Skeletal muscle vasculature exhibits a rapid dilatation in response to mechanical stimulation (e.g., muscle compression, MC), that leads to a transient hyperemia. This characteristic, called mechano-sensitivity, is considered to play an important role in the initial phase of functional hyperemia. The compression-induced hyperaemia was recently shown to progressively decrease in spite of continuing stimulation, the extent of attenuation being correlated with the increase of tissue oxygenation in the relevant muscles.
Aim of the present human study is to test the hypothesis that contraction-induced hyperaemia shares the same dependence on prior mechanical stimulation as compression-induced hyperaemia.

Methods: In 10 healthy subjects hemodynamic changes are assessed in response to: a short mechanical compression (MC) at a supra-systolic pressure (150 mm Hg) delivered to the lower leg by means of a customized pneumatic device, an electrically-stimulated contraction (ESC; pulse duration: 500 μs; frequency: 20 Hz; total train duration: 0.5 s) of the calf muscles, and a combination of both stimuli separated by 25-s of pause. Hemodynamic monitoring includes near infrared spectroscopy, detecting tissue oxygenation and blood volume in lateral gastrocnemius muscle, as well as simultaneous echo-Doppler measurement of blood flow at femoral artery.

Results: Single MC and ESC elicited comparable hyperaemic responses (41.0 ± 15.5 and 41.9 ± 12.4 ml, respectively) and transiently increased local tissue oxygenation from 65.7 ± 1.1 to 77.9 ± 4.7 % (P b 0.05) and from 65.9 ± 1.0 to 78.7 ± 3.9 % (P b 0.05), respectively. After the 25 s blood flow was returned to basal level while tissue oxygenation was about at its peak (ranging from 77.9 to 79.4% in the different conditions, P b 0.05). Irrespective of whether this condition was caused by prior ESC or MC the hemodynamic response to MC or ESC delivered at this time was considerably attenuated (by 66 to 89% in the different conditions, P b 0.05).

Conclusions: The contraction-induced hyperemia shares the same dependence on mechanical pre-conditioning that characterizes the compression-induced hyperaemia, supporting the idea of a common mechano-sensitive mechanism, inactivated by increased tissue oxygenation. This mechanism may play a role in limiting hyper-perfusion, thus preserving local homeostasis and systemic resources.