P10.08: PULMONARY ARTERY CALCIFICATION IN RACEHORSES

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To link to this article: https://doi.org/10.1016/j.artres.2009.10.139

Published online: 14 December 2019
Objective: to test whether 1,25(OH)2D3 (active vitamin D) modifies contractility of proximal resistance vessels, dose-dependently.

Methods: Male Wistar rat mesenteric arteries were investigated by wire myography, as a method to assess vessel tone, using a modified UMR CNRS 6214/INSERM 771, Angers, France

Results: KPPS-induced contraction was unaffected by 1,25(OH)2D3, but slightly decreased after 3 h incubation in control and 1,25(OH)2D3 groups (generally n = 5 arteries each). After 10 min NA-induced contraction at 10-7 M, a small dose response occurred (controls 192±22%; vitD 10 nM 183%, 100 nM 169%), but after 3 h incubation with 100nM 1,25(OH)2D3, contraction decreased at 3x10-6, and at 10-5 M to 118.6±10.3%, compared with controls (mean ± SE: 145.4 ± 13.9%). While differences were individually ‘significant’ (p < 0.04, Wilcoxon test), 2-way ANOVA demonstrated clear vitD (F(3,80) 6.3, p = 0.001) and NA effects (F(3,80) p < 0.000), without interaction. Ach-induced relaxation (at 10-4 to 10-3 M) after 30 min incubation was not enhanced by any 1,25(OH)2D3 dose. After 3 h, higher concentration NA (10-6 to 10-3 M) induced contraction. Paradoxically, 100 nM 1,25(OH)2D3 marginally increased contractions (105.2±4.8%; control 91.7±4.7%), not individually ‘significant’ but by 2-way ANOVA, both vitD & Ach dose effects were (F(3,80) 6.6, p < 0.001).

Conclusion: To our knowledge, these are the first vitD experiments on proximal resistance vessels. 100 nM vitamin D may decrease NA-induced contraction but paradoxical endothelial effects may underline its variable in vivo actions.

P10.07

A PPG MEASUREMENT SETUP AND PULSE WAVE ANALYSIS FOR ARTERIAL STIFFNESS

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The light energy absorption of whole blood in the visible and infrared range is partly caused by the oxidized and reduced haemoglobin. The measurement principle can be applied for photoplethysmography (PPG). Our PPG measures the blood flux in human vessels with means of red and infrared light absorption. The absorption of light varies with the oxygen concentration and amount in blood. The PPG device has phase sensitive detection electronics which was found to be a good solution for the measurement. The small absorption signal occurs simultaneously at two different wavelengths, 660 and 940 nm. In practice, the PPG waveforms, called pulse waves, can be rapidly and simply acquired by a PIN photodiode which measures the transmission of red and infra-red LED light through the forefinger and the second toe simultaneously. The waveforms are characteristics for the young person but different for the elderly person. The four template waveforms are in the consideration for waveform analysis and we get the accurate results enough.

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PULMONARY ARTERY CALCIFICATION IN RACEHORSES

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Vascular calcification (VC) has been sporadically reported in horses, but little is known regarding cause, pathogenesis and clinical significance. We hypothesized that in horses, structural and molecular changes may occur during VC that are comparable to human and mouse models. We surveyed Thoroughbred and Standardbred racehorses (n = 101) for the prevalence, distribution and severity of VC. Histopathological, ultrastructural imaging and energy dispersive X-ray elemental analyses were used to examine the lesions. Immunohistochemistry for cell markers (smooth muscle α-actin, SM22a and Sox9) was performed in selected samples from control (n = 10), mildly (n = 10), and severely (n = 10) calcified arteries. Results showed that calcification of the tunica media of mainly the pulmonary artery branches, was present in 82% of horses, and both breeds and genders were similarly affected. Lesions appeared as white-to-yellowish, hard, gritty plaques of variable size. Microscopically, elastic fibers were thin, fragmented and calcified, and surrounded by dense collagen matrix, as described for Mönckeberg sclerosis. Elemental analysis of the calcified areas was consistent with hydroxyapatite mineral. No immunoreactivity for the smooth muscle cell markers, smooth muscle α-actin and SM22a was observed in cells found at the calcification site. Many of these cells had a chondrocytic phenotype appearance and showed immunoreactivity for Sox9, a chondrocyte marker.

Arterial calcification in horses share histopathological features with arterial medial calcification in humans and may result in similar physiological abnormalities such as vascular stiffness. The occurrence of VC in young racing horses indicates the need to investigate its pathogenesis and potential clinical implications.