Title: **ALMITRINE INDUCED PaO_2 INCREASE PERSISTS IN ABSENCE OF PULMONARY PRESSURE INCREASE**

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Introduction: Almitrine (A) improves gas exchange in ARDS patients. Several authors suggest that the increase in pulmonary arterial pressure (PAP) induced by A, could improve VA/Q ratio by pulmonary vessels recruitment. The aim of the study was to analyse the role of the pulmonary hypertension on PaO_2 increase.

Materials and methods: 7 patients mechanically ventilated (FiO_2 = .3), were studied in the early post-operative period after coronary bypass graft surgery. Criteria for inclusion were: normal right ventricular function; cardiac index (CI) > 2.5 l/min/m^2; no pulmonary, renal and hepatic failure; no cardio or vasoactive drugs. Arterial and mixed venous blood gases (radial and Swan-Ganz catheters) were sampled, and pulmonary and systemic hemodynamic parameters were measured after the following periods: 1/ T0 = 1 hr of hemodynamic, ventilatory and temperature stability; 2/ T1 = 15 min of lower body positive pressure (LBPP) increase by a G-Suit (35 mmHg on the legs, 20 mmHg on the abdomen) which was maintained during T2 and T3 periods; 3/ T2 = 15 min of A infusion (2-4 mcg/kg/min); 4/ T3 = recovery period during 90 min ; 5/ T4 = 15 min of A infusion (at the same doses) with LBPP decrease adapted to obtain a similar PAP than T3. Diuresis was compensated during the protocol. Statistics were performed by Wilcoxon test.

Results:

	PaO_2 (mmHg)	$P\bar{v}O_2$ (mmHg)	$\overline{PAP}$ (mmHg)	CI (l/min/m2)	MAP (mmHg)
To	108±33	33±5	14±4	3.3±.7	82±11
T1	109±31	33±6	17±4 #	3.4±.7	88±14
T2	128±32 #*	35±5	21±5 #*	3.6±.9	90±14
T3	111±32	34±6	16±4	3.4±.7	88±12
T4	125±33 #*	35±4	17±4 #	3.4±.8	85±15

#: p<.01 vs T0; *: p<.01 vs T1

MAP = mean arterial pressure;
$P\bar{v}O_2$ = oxygen venous pressure.

Discussion: PAP increase by LBPP did not improve PaO_2. A significantly increased PaO_2 in the same extent when PAP was increased (T2 vs T1) or maintained unchanged (T4 vs T1), suggesting that A induced PAP elevation is not necessary to improve PaO_2.

TITLE: EFFECT OF BRANCHED CHAIN AMINO ACIDS (BCAA) ON DIAPHRAGM FATIGUE IN VITRO

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Nutritional support improves respiratory muscle function; BCAA specifically increase respiratory drive. This study investigates the acute effect of BCAA on isometric force production, fatigue and recovery in isolated, directly stimulated, rat hemidiaphragms in the absence of systemic and neurohumoral interactions.

Four groups of hemidiaphragms were equilibrated with either Krebs Ringer Buffer solution(KRB) alone(as paired controls, n=18), with KRB plus a balanced mixture of amino acids of five times plasma concentration(CAA, n=6), an equimolar mixture of leucine, isoleucine, and valine at the same total amino acid concentration as in CAA(BCAA, n=6), or KRB and CAA+BCAA mixture(CAA+BCAA, n=6). Hemidiaphragms were stimulated directly under complete neuromuscular block. Fatigue was induced by 10 min stimulation with 30 trains/min of 5 Hz at 50 % duty cycle. The isometric tensions elicited by single and tetanic(10 to 100 Hz) stimuli were measured at baseline(B), after 2 hours of equilibration, and 0, 10, 30, and 60 minutes(T6) after induction of fatigue.

As illustrated in figure, tension difference (% of B in experiment - % of B in paired control) at T6 was significantly different in BCAA and CAA+BCAA at all frequencies compared with control, and there was significant difference ($p<0.05$) between CAA and either BCAA or CAA+BCAA at 100 Hz stimuli.

We conclude that, although the mechanism is unclear, branched chain amino acids have an ameliorative effect on fatigue of isolated rat diaphragm.