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1

GROWTH HORMONE (GH) LEVELS IN ALLERGIC CHILDREN BEFORE AND AFTER STANDARDIZED EXERCISE. Solé,D.;Spinola-Castro,A.M.;Denardim,O.; Arruda,L.K.P.;Mallozi,M.C.;Naspitz,C.K. Division of Endocrinology, Department of Pediatrics, Escola Paulista de Medicine, São Paulo, Brasil.

Bronchial asthma, among the allergic diseases has been pointed out as a cause of growth failure. In a previous report, our group showed that 10% of asthmatic patients from a private clinic (high social level) had short stature (height < 2,5tile). The factors that could be related to growth impairment in atopic children are unknown despite all the research that had been done. In order to evaluate GH secretion through exercise stimulation test, 17 allergic children were submitted to the following protocol: Exercise in a treadmill, during 5 minutes, with a speed calculated based on stature (V(mph)= 1,16 + 0,02 X Stature). Blood samples were collected at time 0 (before exercise) and 10 minutes after resting. The GH was measured through RIA. The children were separated in two groups based on their stature: A) Stature <10tile B) Stature >25tile. The GH levels didn't show any differences when the two groups were compared (the percentual increment after exercises was 100%). We concluded that the exercise test under appropiate conditions is insatisfactory to distinguish the two population studied. This study points out a possibility to be investigated of anomalous GH pattern secretion in allergic patients as the cause of short stature, that would not be evident when a stimulation test is done, because the allergic patients under a acute stimulus can secrete GH properly.

2

ENDOGENOUS OPIOIDS (EO) MODIFY THE CARDIAC OUTPUT (CO) DISTRIBUTION IN THE HEMORRHAGIC SHOCK IN THE NEWBORN. Espinoza,M.; Riquelme ,R.; Germain,A.; Llanos,A.J. Departamento de Preclínicas, Ciencias Medicas Oriente Facultad de Medicina,Un. de Chile, Un.de Chile, Stgo.Chile.

EO are found in high concentrations in the newborn and they play a role in the circulatory adaptations to shock in adults. Therefore we studied the EO role in cardiovascular responses of 11 chronically catheterized newborn lambs (6-13 day's old) that were submitted to shock. The shock state was produced by withdrawing enough blood to reduce mean arterial pressure to 50% of its basal value during 45 min. Then animals were treatd with the opiate receptor antagonist naloxone (Nlx) (n=6, bolus 1mg/kg + infusion 20ug/kg x min x 45 min) or NaCl 0.9% in equivalent volume (n=5). CO and its distribution (radiolabeled microspheres) was measured in basal conditions (100%), at the end of the hemorrhage (S) and at the end of the infusion. The results were (Mean ± SEM, ANOVA):

	Shock(S)	Infusion(Nlx)	Shock(S)	Infusion(NaCl 0.9%)
CO	61 ± 16*	49 ± 15*	70 ± 17*	47 ± 13*
Heart	126 ± 20*	133 ± 17*	121 ± 20*	64 ± 18* ≠
Brain	140 ± 15*	148 ± 18*	150 ± 30*	67 ± 15*≠
Gut	61 ± 15*	40 ± 10*≠	60 ± 17*	56 ± 14*
Carcass	50 ± 21*	39 ± 12*≠	52 ±14*	45 ±16*

* p < 0.05 vs B.; ≠ p < 0.05 vs S.

These results show that 1) The EO blockade with naloxone keeps the "vital" organs blood flows at the expense of an important decrease in gut and carcass blood flow. 2) EO play a role in the cardiovascular adaptation to hemorrhagic shock modulating the vasoconstriction of the neonatal gut and carcass. G.2183-8733. DIB.U.CH.

3

OXYGEN DELIVERY ($\dot{O}_2$) TO THE FETAL ORGANS DURING ASPHYXIA (A) IN SHEEP. Germain, A.M.;Espinoza,M.; Riquelme,R.; Llanos, A.J. Depato.de Preclínicas, Ciencias Médicas Oriente, Fac.de Medicina, Universidad de Chile.

During the fetal A there is an increase in heart, brain and adrenal blood flows together with a decrease in kidney, gut and carcass blood flows. Since the $\dot{O}_2$ to the fetal organs has not been determinated during a prolong A, we measured the $\dot{O}_2$ during a 40 min reduction of the uteroplacental blood flow (UBF) to 50% of its basal value. In 5 sheep (0.8 - 0.9 of gestation) an infatable occluder was placed around the uterine artery. Catheters were inserted in the fetal femoral vein and artery, carotid artery and jugular vein. Fetal organ blood flows (radiolabeled microspheres) and O_2 content in ascending and descending aorta (Hemoximeter) were measured in basal condition and during a 50% reduction in UBF (+ 20 min and + 40 min). Oxygen delivery to fetal organs was calculated: $\dot{O}_2$=organ blood flow X O_2 content. Results were ($\bar{X}$ ± SEM; n =5 * =p < 0.05, ANOVA)

$\dot{O}_2$ (ml O_2/min x 100g)	Basal	+ 20	+ 40
Heart	21.3 ± 1.1	22.5 ± 0.8	21.6 ± 3.7
Brain	12.2 ± 1.5	9.7 ± 0.9	9.6 ± 1.5
Kidney	15.0 ± 2.1	4.4 ± 1.2*	4.0 ± 1.0*
Gut	5.8 ± 0.9	1.3 ± 0.3*	1.2 ± 0.4*
Carcass	2.2 ± 0.2	0.5 ± 0.1*	0.5 ± 0.1*

These results show that during a prolonged fetal asphyxia $\dot{O}_2$ to the "vital" is maintained at the expense of a reduction of $\dot{O}_2$ to the "non-vital" organs. The 75% reduction of $\dot{O}_2$ to the "non-vital" organs probably produces tissue hypoxia that could explain the damage observed in these organs during asphyxia. Grant 2183 - 8733, DIB, Universidad de Chile.

4

MATERNAL HEMODYNAMIC CHANGES AND FETAL GROWTH RETARDATION. Rosso,R.;Donoso,E.;Espinoza,R.; Fernández,C.;Braun,S.;Godoy,R. Depts.of Pediatrics; Obstetrics and Gynecology; and Cardiovascular Diseases Medical School; Pontifical Catholic University;Santiago, Chile.

Based on previous studies we have postulated that blood volume expansion during pregnancy is a key physiological adjustment since it makes possible the increase in cardiac output and utero-placental blood flow, necessary to sustain normal fetal growth. This idea was tested in non-smoking, non-hipertensive gravidas who by ultrasound scanning were found to carry either a normal size fetus ("control") (15 cases) or a small-for-date fetus ("experimental") (15 cases). At weeks 37 - 40 of gestation measurements of plasma volume, using the Evans blue method, and cardiac output, using echocardiography, were performed in all subjects while in a left lateral position. At delivery the newborns were examined to establish adequacy of weight for gestacional age and to rule out congenital malformations and infections. Plasma volume was 3120 ± 484 (S.D) ml in controls and 2717 ± 217 ml in experimentals (p <0.01). Cardiac output was 6127 ± 868 ml/min in controls and 5282 ± 891 ml/min in experimentals (p<0.01). In both groups combined plasma volume was significantly correlated with cardiac output (r= 0.45; p<0.01) and both plasma volume and cardiac output were significantly correlated with birth weight (r= 0.47; p <0.01 and r=0.42; p <0.01, respectively). The data support the hypothesis that inadequate maternal plasma volume expansion my lead to fetal growth retardation. (Supported by grant 116/85 from the Dirección de Investigación, Universidad Católica de Chile).