Title: ENDOCRINE FUNCTIONS DURING DEEP HYPOTHERMIA IN PEDIATRIC CARDIAC SURGERY

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Introduction: The Technique of deep hypothermia (DH) and circulatory arrest (CA) used during pediatric cardiac surgery produces significant metabolic changes. Detailed knowledge is still lacking in many areas and problems extending into the immediate postoperative period are poorly understood. The present study was designed to follow endocrine functions during the period of cooling, rewarming on cardiopulmonary bypass (CPBP) after circulatory arrest and in the immediate postoperative period.
Methods: Twenty-eight infants, ages 2 weeks to 20 months, undergoing complete surgical repair of congenital heart defects were studies: 8 patients had ventricular septal defects, 3 transpositions of the great vessels, 3 atrial septal defects, 2 atrioventricular canals, 1 tetrology of Fallot and 1 total anomalous venous return. The patients were anesthetized with Ketamine, ventilated with 100% oxygen and received Pancuronium for muscle relaxation. Before initiating CPBP, Methyprednisolone (30mg/kg) and Phentolamine (15mcg/kg) were given intravenously. Phentolamine was reinjected approximately every 15 minutes throughout the cooling on CPBP. In 11 infants pulsatile perfusion was used (group I) and non-pulsatile flow in the remaining 7 patients (group II). Serum glucose (G), insulin (I), growth hormone (GH), calcium (C), and calcitonin (CT) levels were measured after induction of anesthesia, before institution of CPBP, at 25° cooling, immediately before CA, at 25° C rewarming, at 37° C and in group I. 1 hour postoperatively.
Results: Using the student t-test, significant changes were seen in all studied hormones except CT. GH increased continuously throughout the procedure and in the immediate postoperative period, insulin concentration were suppressed but showed rapid rebound during rewarming with return to near control values postoperatively; CT showed practically no variation with temperature changes. Difference between the results of the 2 groups were statistically not significant.
See Table 1
Discussion: Knowledge of endocrine function during and following DH with CA could be helpful in the management of patients subjected to this technique. Our study showed the following results:
1. IN-production was suppressed, resulting in progressive hyperglycemia. IN secretion did not resume until normothermia was reached.
2. Ca++ levels decreased to an extent greater than caused by dilution on CPBP, we postulate that mobilization from bone is impaired. The CT concentrations remained unchanged, but showed an appropriate increase with administration of $CaCl_2$ (20mg/kg) at 32° C.
3. GH levels increased strikingly throughout the procedure to acromegalic levels, suggesting disturbance of hypothalamic functions.

Table 1: Plasma levels (Mean ± SEM)

Hormone	Group I					Group II				
	37°	Pre-CPBP	25°	Pre-CA	25°	37°	1 hr postop	37°	Pre-CPBP	25°
G(mg/dl)	141 ±25	170 ±17	174 ±16	191 ±56	235 ±25	219 ±17	255 ±18	128 ±23	203 ±39	193 ±24
IN(uU/ml)	17.9 ±3.2	15.3 ±2.9	20.9 ±4.2	15.8 ±3.8	18.8 ±5.2	66.8 ±7.5	23.5 ±12.1	10.5 ±2.7	10.7 ±2.9	12 ±4.8
GH(ng/ml)	10 ±0.8	10 ±2.5	22.4 ±6.5	21.1 ±3.8	45.5 ±15.2	65.5 ±15.8	55.5 ±18	6.8 ±0.6	19.4 ±7.9	25.5 ±6.7
CA(mg/dl)	9.5 ±0.6	9.9 ±0.8	7.1 ±0.6	7.1 ±1.4	8.9 ±0.8	11.3 ±0.9	10.6 ±0.8	7.7 ±0.8	8.9 ±0.4	4.6 ±5.1
CT(pg/ml	21.2 ±1.7	23.8 ±1.7	16.4 ±4.5	17.3 ±10	18.4 ±7.3	21.6 ±6.5	18.1 ±3.5	22.9 ±9.6	16.2 ±3.6	21.7 ±2.6

Hormone	Pre-CA	25°	37°
G(mg/dl)	221 ±22	330 ±65	339 ±49
IN(uU/ml)	8.3 ±3.2	17.7 ±8.0	54.2 ±19.7
GH(ng/ml)	22.1 ±3.8	53.5 ±18.2	50.3 ±17.1
CA(mg/dl)	5.3 ±0.9	8.1 ±1.4	7.8 ±0.9
CT(pg/ml	21.8 ±8.7	20.8 ±7.4	28.2 ±2.6

In summary: During DH significant changes were seen in blood glucose, Ca++ and GH levels, suggesting alterations of carbohydrate metabolism and hypothalamic-pituitary functions.