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" EFFECTS OF CONDITIONING, DECONDITIONING AND RECONDITIONING ON CATECHCOLAMINE RESPONCES OF ADULT MALES"

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ABSTRACT:

Catecholamines are well known for the role they play in the adaptive processes both at rest and in response to acute stress. Under normal conditions, stress is considered as a primary factor in catecholamine secretion. Indeed, a marked increase of plasma catecholamine concentrations is often observed in response to various stressors such as physical exercise. Twenty four residential untrained male subjects (age between 22-24 years) were selected for the study and repeated measures design was applied. The effects of conditioning programme for 8 weeks, followed by 4 weeks of deconditioning and 6 weeks of reconditioning treatment were imparted and catecholamine response of adult males analyzed. 4 weeks of deconditioning significantly increased noradrenaline responses of adult males, 6 weeks of reconditioning significantly decreased adrenaline response of the subjects of the present study whereas the other conditions were unaffected.

Key words : Conditioning, Deconditioning , Reconditioning and Catecholamine

INTRODUCTION :

Nervous and endocrine systems are important mediators of the body's physiological adjustment to a variety of physical, environmental and behavioural stressors. Catecholamines are well known for the role they play in these adaptive processes both at rest and in response to acute stress. Under normal conditions, stress is considered as a primary factor in catecholamine secretion.[1] Indeed, a marked increase of plasma catecholamine concentrations is often observed in response to various stressors such physical exercise,[1-8] or in response to various non-exercise-related factors such as insulin-induced hypoglycaemia[9] as well as after stimulation with hypoxia,[10,11] acidaemia,[11] glucagons[11] or caffiine.[12]. The term 'catecholamines' is composed of several components that are all derived from an amino acid, e.g. tyrosine. The principal components are adrenaline (epinephrine) and noradrenaline (nore- pinephrine). Their synthesis takes place at two levels: (i) sympathetic nervous fibre extremities for noradrenaline; and (ii) chromaffin cells of the adrenal medulla for both adrenaline and noradrenaline [13,14]. Therefore, noradrenaline is considered as a neurotransmitter and a hormone, and adrenaline only as a hormone. Since the adrenal medulla is under sympathetic nervous system control, we often talk about the sympathoadrenal system.

Therefore noradrenaline and adrenaline are considered, respectively, as indexes of the sympathetic nervous system activity and the adrenal medulla activity.

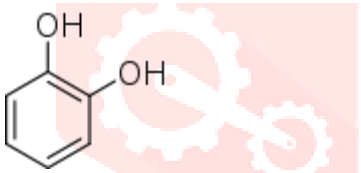
A **catecholamine** (/ˌkætəˈkoʊləmiːn/; abbreviated **CA**) is a monoamine neurotransmitter, an organic compound that has a catechol (benzene with two hydroxyl side groups next to each other) and a side-chain amine [15]. Catechol can be either a free molecule or a substituent of a larger molecule, where it represents a 1,2-dihydroxybenzene group. Catecholamines are derived from the amino acid tyrosine, which is derived from dietary sources as well as synthesis from phenylalanine [16]. Catecholamines are water-soluble and are 50% bound to plasma proteins in circulation.

Included among catecholamines are epinephrine (adrenaline), norepinephrine (noradrenaline), and dopamine. Release of the hormones epinephrine and norepinephrine from the adrenal medulla of the adrenal glands is part of the fight-or-flight response [17].

Tyrosine is created from phenylalanine by hydroxylation by the enzyme phenylalanine hydroxylase. Tyrosine is also ingested directly from dietary protein. Catecholamine-secreting cells use several reactions to convert tyrosine serially to L-DOPA and then to dopamine. Depending on the cell type, dopamine may be further converted to norepinephrine or even further converted to epinephrine [18].

Various stimulant drugs (such as a number of substituted amphetamines) are catecholamine analogues. Catecholamines cause general physiological changes that prepare the body for physical activity (the fight-or-flight response). Some typical effects are increases in heart rate, blood pressure, blood glucose levels, and a general reaction of the sympathetic nervous system. Some drugs, like tolcapone (a central COMT-inhibitor), raise the levels of all the catecholamines. Catecholamine is secreted into urine after being broken down, and its secretion level can be measured for the diagnosis of illnesses associated with catecholamine levels in the body [19]. Urine testing for catecholamine is used to detect pheochromocytoma.

Two catecholamines, norepinephrine and dopamine, act as neuromodulators in the central nervous system and as hormones in the blood circulation. The catecholamine norepinephrine is a neuromodulator of the peripheral sympathetic nervous system but is also present in the blood (mostly through "spillover" from the synapses of the sympathetic system). High catecholamine levels in blood are associated with stress, which can be induced from psychological reactions or environmental stressors such as elevated sound levels, intense light, or low blood sugar levels.



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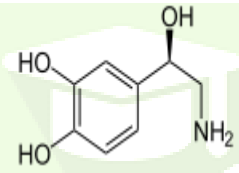
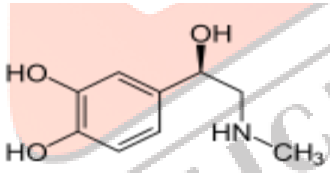


Epinephrin



Norepinephrin

Extremely high levels of catecholamines (also known as catecholamine toxicity) can occur in central nervous system trauma due to stimulation or damage of nuclei in the brainstem, in particular, those nuclei affecting the sympathetic nervous system. In emergency medicine, this occurrence is widely known as a "catecholamine dump".

Extremely high levels of catecholamine can also be caused by neuroendocrine tumors in the adrenal medulla, a treatable condition known as pheochromocytoma.

High levels of catecholamines can also be caused by monoamine oxidase A (MAO-A) deficiency, known as Brunner syndrome. As MAO-A is one of the enzymes responsible for degradation of these neurotransmitters, its deficiency increases the bioavailability of these neurotransmitters considerably. It occurs in the absence of pheochromocytoma, neuroendocrine tumors, and carcinoid syndrome, but it looks similar to carcinoid syndrome with symptoms such as facial flushing and aggression [20, 21]. Acute porphyria can cause elevated catecholamines [22].

Catecholamines act by using membrane receptors.[23] There are at least two adrenergic receptor sites (α and β). These two last receptors are also divided into subtypes α1, α2, β1 and β2 [24]. Noradrenaline primarily activates α-receptors and adrenaline activates primarily β-receptors, although it may also activate α-receptors (for review see Garcia-Sainz[25]). Stimulation of α-receptors is associated with constriction of small blood vessels in the bronchial mucosa and relaxation of smooth muscles of the intestinal tract. β-Receptor activation relaxes the bronchial smooth

muscles, which cause the bronchi of the lungs to dilate. In addition, β-receptor stimulation also affects the heart and causes an increase in the rate and force of contractions (table-I).
The activation of the sympathoadrenal system may induce several physiological effects on the body (for review see Hanoune[26]). Catecholamines have been shown to stimulate respiratory, cardiac, metabolic and thermoregulatory functions. These results were obtained by using different methods, such as the adrenal medulla ablation in animals, pharmacological sympathectomy, adrenaline and noradrenaline infusion, α- or β-blockades and marked adrenaline. Therefore, all these methods have some limits that must be taken into account when interpreting results.

Catecholamines act well simultaneously at several levels to permit the realization and/or the prolongation of physical exercise. For example, during prolonged exercise, catecholamines play a major role in oxygen and energetic substrates transportation to active muscles. Hence, studies using β-blockade report a decrease of endurance capacity and maximal oxygen uptake ($\dot{V}$ O2max). This decrease in performance is often explained by the action of catecholamines at the metabolic and haemodynamic levels. In fact, when using β-blockade to inhibit catecholamine secretion, Laustiola et al.[27] reported a decrease of heart rate and blood flow pressure both at rest and in response to exercise. This inhibition also reduces $\dot{V}$ O2max, plasma glucose, plasma free fatty acid, plasma glycerol and blood lactate concentrations.[28] It is also demonstrated that catecholamines influence physical performances in response to maximal or supramaximal exercises by regulating muscular glycogenolysis[29] and hepatic glycogenolysis.[30-32] Therefore, it is clear that a high capacity to secrete these hormones represents an advantage in competitive sports.[1,5,33].

Table I. Main cardio-respiratory and metabolic effects of catecholamines

Efects	Adrenoreceptors	Physiological effects	Responses
Cardiovascular and respiratory effects			
atria and ventricles	β 1	↑ Contractility	↑ Cardiac output
Sinotrial node	β 1	↑ Conduction velocity ↑ Heart rate	↑ Conduction velocity ↑ Heart rate
Arteries renal	α	Vasoconstriction ↓ Local blood flow	Vasoconstriction ↓ Local blood flow
Splanchnic	α	Vasoconstriction ↑ Systemic arterial pressure	Vasoconstriction ↑ Systemic arterial pressure
Skeletal muscles	β 2	Vasodilation	↓ Arterial pressure ↑ Local blood flow
Veins	α 2	Vasoconstriction	↑ Blood return to the heart ↑ Cardiac output
Lungs Airway smooth muscles	β 2	Relaxation Bronchodilation	Relaxation Bronchodilation
Metabolic effects			
Liver α1 or β2	Liver α1 or β2	↑ Glycogenolysis ↑ Glyconeogenesis	↑ Blood glucose
Muscle	β2 β2	↑ Glycogenesis ↑ Glucose utilization	↑ Blood lactet ↑ Blood glucose
Pancreas	α2 β2	↓ Insulin secretion ↑ Insulin secretion	↑ Blood glucose ↓ Blood glucose
Adipose tissue	β1 β2 β3?	↑ Lipolysis	↑ Free fatty acids
	α2	↓ Lipolysis	↓ Free fatty acids

At rest and in response to exercise, catecholamine concentrations are influenced by several factors such as exercise characteristics, training status. However, data concerning the training status and gender influence on adrenaline and

noradrenaline responses to exercise at the same absolute and/or relative intensity remain conflicting. In fact several studies concerning catecholamine concentrations in response to exercise, did not report any effects of both endurance[34 and sprint[35] training. In addition, transversal studies did not find differences between trained and untrained subjects.[36] Incontrast,some studies reported higher post-exercise adrenaline concentrations in endurance-[1,37,38] and sprint-trained[4] subjects compared with untrained subjects or in anaerobic-trained subjects compared with aerobic-trained subjects.[39] Other studies also reported higher significant noradrenaline concentra retions in response to exercise after endurance training[40,41] or when comparing endurance-trained subjects to untrained subjects.[38] The training effect on catecholamine responses has already been reviewed[1].
However, it can be noted that this review focused on the effect of training on adrenaline responses to exercise. In the present article, we added new data about the effect of conditioning, deconditioning and reconditioning on the catecholamine response of adult males.

METHODOLOGY :

Twenty four residential untrained male subjects (age between 22-24 years) were selected for the study. Repeated measured design was applied for the study and 't' tests [42] were applied for statistical purpose. The statistical analysis was tested for significance at 0.05 level of confidence.

Height : Height of the subjects was measured with the help of stediometer and it was recorded in centimeters.
Weight: Weight of the subjects was measured with the help of electronic weighing machine and it was recorded in kilogram.
Body Mass Index (BMI): Body mass, weight in kg / height in m^2 [43] .

Height Weight and body mass index were measured of the subjects before conditioning protocol of eight weeks, at the end of conditioning programme, at the end of four weeks deconditioning and at the end of six weeks reconditioning of the subjects i.e. four times the data were collected from the subjects. Catecholamine were estimated clinically by collecting Urine in 24 hours for four different phases of the study [44]. Table-2 represents the conditioning protocol of the subjects.

Table-2. Conditioning protocol of the subjects:

Treatment	Duration	Daily schedule	Weekly plan	Nature of activity
Conditioning	8 weeks	135 minutes in the morning and 90 minutes in the evening	5 days in a week	Warming up, continues run, lite apparatus drills, free hand exercises etc. Heart rate=140 btpm
Deconditioning	4 Weeks	Rest	Rest	Rest
Reconditioning	6 Weeks	135 minutes in the morning and 90 minutes in the evening	5 days in a week	Warming up, continues run, lite apparatus drills, free hand exercises major games etc. Heart rates =140 btpm

RESULTS AND DISCUSSION:

TABLE -3. Mean and standard deviation of the demography of the subjects

Variables	No.	Pre Condition Test		Post Condition Test		Decondition Test		Recondition Test	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Wt	24	60.75	6.7	59.81	5.4	59.84	5.23	59.43	5.32
Ht	24	167.22	3.29	167.22	3.29	167.22	3.37	167.30	3.40
BMI	24	1.67	0.11	1.66	0.08	1.66	0.08	1.65	0.08

Table-4. Pair 't' test value of the subjects

Variables	Pre Vs Post test	Post Vs Decond test	Decond Vs Recond test
Wt	1.57	0.17	1.75
Ht	-	-	1.00
BMI	1.10	0.55	0.47

Table 3 and 4 reveal that height, weight and Body Mass Index of the subjects of the study remains unaffected following conditioning protocol of eight weeks, at the end of conditioning programme and at the end of four weeks deconditioning. As Body Mass Index is depended upon the height and weight of the subjects, the insignificant changes may be due to the insignificant change of height and weight of the sample of the study.

TABLE -5. Mean and standard deviation Catechomanime

Variables	No.	Pre Condition Test		Post Condition Test		Decondition Test		Recondition Test	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Adrenaline (mg/ml)	24	38.85	18.83	39.27	11.07	39.27	11.07	28.11	10.08
Nor-Adrenaline (mg/ml)	24	75.52	26.74	113.45	102.31	74.05	73.71	94.44	68.38

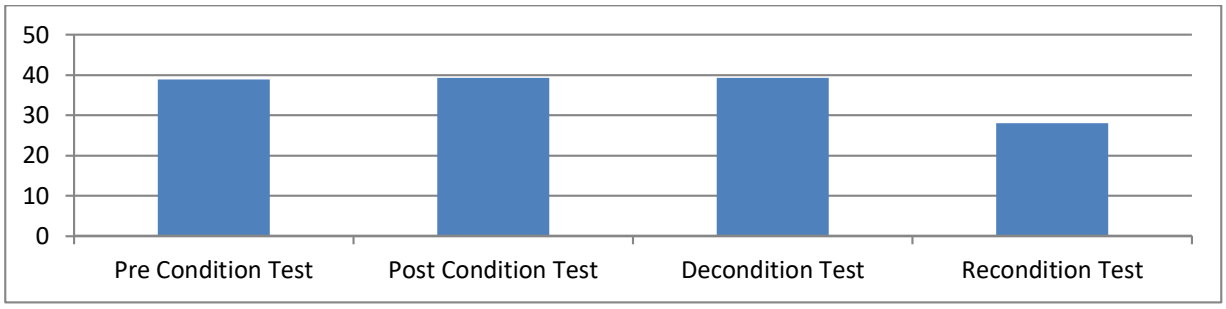


Fig-1. Graphical representation of Adrenaline (mg/ml) responses

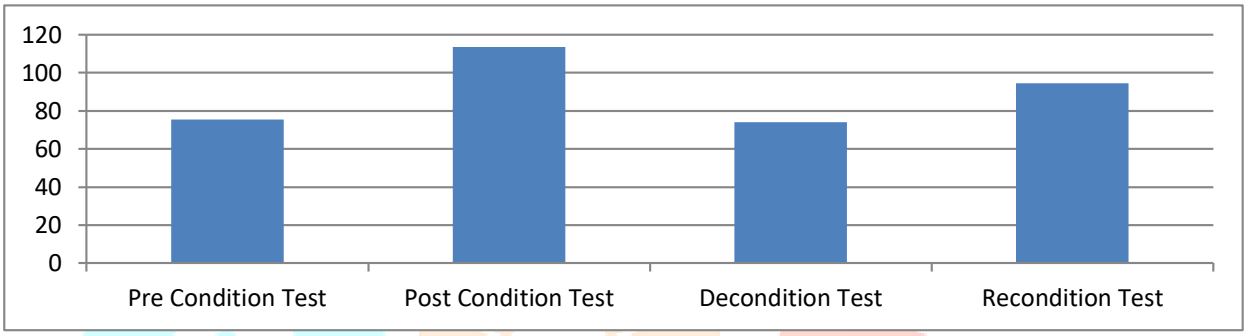


Fig-2. Graphical representation of Noradrenaline (mg/ml) responses

Table-6. Pair 't' test value Catecholamine

Variables	Pre Vs Post conditioning test	Post Vs Decond test	Decond Vs Recond test
Adrenaline (mg/ml)	0.13	-	3.78 **
Nor-Adrenaline (mg/ml)	1.95	2.13*	0.89

* Significant at 0.05 level (2.069)
** Significant at 0.01 level (2.807)

Table -5 and 6 represents the Catecholamine values of different treatment protocols. The adrenaline responses Following deconditioning and reconditioning were significantly decreased (p >.01) and other conditions remained unaffected. Figure-1 represent the graphical presentation of adrenaline responses following three different treatment protocols. The noraderenaline responses were significantly increased (p>.05) after reconditioning and remained unchanged during other conditions. Figure-2 represent the graphical presentation of noradrenaline responses following three different treatment protocols.

Glucocorticoids are released from the adrenal cortex in response to the stress of exercise. Of these, cortisol accounts for approximately 95% of all glucocorticoid activity. Cortisol has catabolic functions that have greater effects in type II muscle fibres [45] About 10% of circulating cortisol is free while ~15% is bound to albumin and 75% is bound to corticosteroid-binding globulin. In peripheral tissues, cortisol stimulates lipolysis in adipose cells and increases protein degradation and decreases protein synthesis in muscle cells, resulting in greater release of lipids and amino acids into circulation, respectively. Because of its major role in tissue remodelling, acute and chronic changes of cortisol during resistance training is often examined.

Resting cortisol concentrations generally reflect a long-term training stress. Chronic resistance training does not appear to produce consistent patterns of cortisol secretion as no change,[46, -52] reductions [53-57] and elevations[58] have been reported during normal strength and power training in men and women, and during short-term overreaching. In animals, cortisol concentrations have ex-plained a substantial amount of the variance observed in muscle mass changes[59]. Thus, it appears that the acute cortisol response may reflect metabolic stress, whereas the chronic changes (or lack of change) may be involved with tissue homeostasis involving protein metabolism.

CONCLUSSIONS :

Catecholamines act well simultaneously at several levels to permit the realization and/or the prolongation of physical exercise. For example, during prolonged exercise, catecholamines play a major role in oxygen and energetic substrates transportation to active muscles.

Within the limitations of the study the following conclusions were drawn :

1. 8 weeks of conditioning remained unaffected for adrenaline and noradrenaline i. e. catecholamine responses of adult males.
2. 4 weeks of deconditioning remained unaffected for adrenaline responses of adult males.
3. 4 weeks of deconditioning significantly increased noradrenaline responses of adult males.
4. 6 weeks of reconditioning remained unaffected for a noradrenaline response of the subjects of the present study.
5. 6 weeks of reconditioning significantly decreased adrenaline response of the subjects of the present study.

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