

## The Effect of High Intensity Training on Resting Cardiovascular Hormones of Elite Wushu Sanda Athletes

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**Abstract:** *This work had used radioimmunoassay to determine 9 male elite Wushu Sanda (a self-defense oriented Chinese wushu) athletes' (24.30±4.20yr, 170.30±3.8cm, 68.30±4.20kg) peaceful level of cardiovascular hormones, include endothelin(ET), calcitonin gene related peptide(CGRP) and atrial natriuretic peptide(ANP). During and after 3 months of high intensity training, the level of ET increased gradually towards the end it significantly increased by two points of measurement. The results suggest that increased plasma catecholamine and hypertensin secretion and/or anoxic and damage of skeleton and cardiac muscle would have caused enhanced ET level; At the beginning of high intensity training CGRP level decreased gradually and parallel with the training the CGRP level significantly decreased. It suggested that the effect of long-term training was different from short-term exercise. Parallel with the intensity training, ET/CGRP ratio was increased. It points to a mismatching of the secretion of ET and CGRP. The level of ANP increased gradually and towards the end by points of measurement it was significantly higher than pre-training. The results suggest that the effect of high intensity training on ANP was significant; Another result showed that there was a significant negative correlation between ET and CGRP, a significant negative correlation between CGRP and ANP, was a significant negative correlation between ET and ANP; This work suggests that the changes of hormone were not related to blood concentration.*

**Key words:** *Wushu Sanda peaceful state ET CGRP ANP*

### Introduction

It was considered that the wushu sanda (a self-defense oriented Chinese wushu) is the most opposable event (full-contact) in which the training and /or competing intensity reached high level. Therefore it does not matter what the physical capacity, performance techniques, tactics of competition or mentalities are in daily training and competition session. There was no long period of Wushu Sanda training, but it has the most effective potential and will have been an event in the 2008 Beijing Olympic

games. Some indices such as blood lactate (La), serum blood urea nitrogen (BUN), creatinine (Cre) and serum enzymes were used to evaluate and control the Wushu Sanda exercise volume in the last decade. For a long time, it has been said that the heart has only a function of pumping blood, but in the recent years, studies have shown that the heart plays important role of secreting hormones such as Endothelin (ET), Calcitonin Gene Related Peptide (CGRP) and atrial natriuretic peptide (ANP). The studies concluded that cardiovascular hormones modulate the functions of the heart<sup>(1,2)</sup>. However, the changes of cardiovascular hormone do not seem to have been studied by the numbers in Wushu Sanda athletes. This has prompted us to study these hormones in elite athletes during the intensity-training season.

### Materials and Methods

Nine male elite Wushu Sanda athletes(24.30±4.20yrs,170.30±3.8cm,68.30±4.20kg)from Inner Mongolia Wushu Sanda team who were selected to attend the 8th National Games of Peoples Republic of China, gave their written consent to participate in this study after being informed of risks and stress associated with the research.. They showed no clinical signs of overtraining, had normal diets and did not take any homeopathic (hormonal) drugs during this study.

In this study, the short periodic training method was introduced during training season. We decided 4 weeks for a training cycle in which the pre 3 weeks was the intensity training phase, the 4th week acting as a minus load period (according to the physical conditions of athletes to adjust training in order to allow them recover for the following season). During the intensity training weeks, 11 training courses were arranged every week. In courses of Tuesday and Friday the maximum training intensity was conducted while Monday, Wednesday and Saturday the training intensity was less intense, Thursday acted a flexible day according to athletes' physical conditions. Every training course was for the period of 2 hours. In the intensity training cycle the educational observation methods and the Rating Perceived Exertion (RPE) according to Borg (1970)<sup>(3)</sup> were carried out to modulate and control the exercise volume.

We the doctors for the Inner Mongolia Wushu Sanda team performed a thorough history and clinical examination on all subjects prior to the investigations. Data from this examination and routine blood chemistry and urinalysis revealed no abnormalities. All subjects were involved in a full-season study and were given blood samples. Blood samples were collected 5 times before a national tournament and the 8th National Games of Peoples Republic of China (11 May, 29 May, 15 June, 15 July and 12 August). Before the first blood samples were taken, athletes were given no exercise intensity for a week. The physiological data such as heart rate and blood pressure were obtained previous of sampling day (pre-training and post-training) and sampling day (at rest), the blood samples were taken on the sampling day.

This study used <sup>125</sup>I labeled radioimmunological methods. Venous blood samples were taken between 07:00 am and 08:00 am. During standardized conditions such as fasting and resting samples in a sitting position for at least ten minutes resting from dormitory next morning of intensity exercise. Marked

a glass tube for each blood sample, in the glass tubes the added EDTA-Na<sub>2</sub> 30.0  $\mu$ l and trasyolol 40.0  $\mu$ l and 2.0 ml blood, mixed and centrifuged at 4°C 3000 rpm for 10 min. Plasma was withdrawn after centrifugation and stored at -20°C until analyzed. Before analysis ET, CGRP and ANP, the deposited plasma was melted with ice water and centrifuged at 4°C 3000rpm for 5 min, on the upper level. The RIA kits were from Beijing Dongya Medical Biotech Co. Ltd (ET (DY-03), CGRP (DY-04)), and Beijing North Institute of Biological Technology (ANP (RH-51-100)). The coefficient of inter group and extra group variability was, ET as 6.9% and 9.4%, CGRP as 6.9% and 8.8%, ANP as 8.9% and 10.4%, respectively. This study also used Hematimeter (Aitaik CA-300, Japan) to test athletes' hemoglobin (Hb), Hematocrit (HCT); Urine analyzer (Mini-4210, Japan) and electrocardiograph (NIHON KOHODEN Cardiofax, Japan) Lactate analyzer (YSI-1500, US) to test urine protein, lactate and ECG, respectively. The athletes were also required to periodically report to the laboratory before and after training. We felt that these safeguards insured that all subjects followed the training.

All measures are expressed as mean $\pm$ SD. One-way analyses of variance (ANOVA) with repeated measures were used to determine the significance of the effect of high intensity training on changes of cardiovascular hormones. Specific mean differences were identified using a Newman-Keuls post-hoc test. Correlations of indexes were calculated and used Fisher's formula to evaluate the significance. Significances of differences are denoted as  $p < 0.05$  and  $p < 0.01$ , respectively.  $\chi^2$  was used to determine the differences of individual in whose urine the protein appeared. Significances of differences are also denoted as  $p < 0.05$  and  $p < 0.01$ .

## Result

1. The heart rate, systolic blood pressure, urine protein and RPE for the Wushu Sanda athletes on previous day of sampling (pre-training and post-training) and the sampling day are presented in table 1. Neither heart rate nor urine protein significantly changed during the training and testing periods. Systolic blood pressure of previous day of sampling significantly increased ( $p < 0.05$ ) on 12 August compared with 11 May and 29 May, the samplings taken on 15 June, 15 July, and 12 August were significantly higher than those on 11 May, 29 May and 15 June ( $p < 0.05$ ). The rating of perceived exertion of the previous day of sampling significantly increased ( $p < 0.01$ ) on 15 July and 12 August compared with 11 May, 29 May and 15 June, and the sampling day was significantly enhanced ( $p < 0.01$ ) compared with other testing points.

2. Resting ET levels were significantly increased ( $p < 0.05$ ) on 15 July compared with 11 May, 29 May and 15 June, and which also significantly increased ( $p < 0.01$ ) on 12 August compared with 11 May, 29 May, 15 June and 15 July (table 2).

3. Opposite to ET changes, the mean values of resting CGRP levels decreased gradually. CGRP levels were significantly decreased ( $p < 0.05$ ,  $p < 0.01$ ) on 15 June, 15 July and 12 August compared with 11 May, 29 May, 15 June and 15 July (table 2).

Table 1. Physiological and urine variables on the previous day of sampling and sampling day

Variables	11 May	29 May	15 June	15 July	12 August
<b>Pre-training (previous day of sampling)</b>					
Heart rate(bts · min <sup>-1</sup> )	59±8	58±7	57±8	58±8	58±9
Systolic blood pressure(mmHg)	120±10	120±11	125±12	135±12	132±10 <sup>#</sup>
Urine protein (≥0.05g · L <sup>-1</sup> ) <sup>a</sup>	0	0	0	2	2
Rating of perceived exertion	8.5±0.4	8.7±0.6	8.6±0.4	9.5±0.5 <sup>**#SS</sup>	10.6±0.4 <sup>***#SS&amp;&amp;</sup>
<b>Post-training (previous day of sampling)</b>					
Heart rate(bts · min <sup>-1</sup> )	----	173±11	173±10	176±11	178±11
Systolic blood pressure(mmHg)	----	196±21	197±23	193±23	195±21
Urine protein (≥0.05g · L <sup>-1</sup> ) <sup>a</sup>	----	1	0	2	3
Rating of perceived exertion	----	19.2±0.4	19.3±0.3	19.2±0.4	19.4±0.3
<b>At rest (sampling day)</b>					
Heart rate(bts · min <sup>-1</sup> )	60±8	59±7	60±9	59±8	60±8
Systolic blood pressure(mmHg)	118±12	120±11	130±12 <sup>*#</sup>	129±10 <sup>#</sup>	132±12 <sup>*#</sup>
Urine protein (≥0.05g · L <sup>-1</sup> ) <sup>a</sup>	0	0	0	1	1
Rating of perceived exertion	8.3±0.5	8.2±0.4	8.3±0.4	8.5±0.3	10.8±0.5 <sup>***#SS&amp;&amp;</sup>

\* : p<0.05 \*\* : p<0.01 denotes significantly different from 11May

# : p<0.05 ## : p<0.01 denotes significantly different from 29May

\$ : p<0.05 \$\$ : p<0.01 denotes significantly different from 15June

& : p<0.05 && : p<0.01 denotes significantly different from 15July

a : quantity of individual in whose urine the protein appeared protein≥0.05g · L<sup>-1</sup>

Table 2. The changes of plasma variables during intensity training

Variables	n	11 May	29 May	15 June	15 July	12August
ET(pg · ml <sup>-1</sup> )	9	45.36±5.18	46.36±4.39	46.87±5.12	50.34±5.02 <sup>*#S</sup>	55.69±4.40 <sup>***#SS&amp;&amp;</sup>
CGRP(pg · ml <sup>-1</sup> )	9	17.33±0.87	16.38±0.87	16.05±0.99 <sup>*</sup>	15.78 ±0.90 <sup>**</sup>	13.49±0.86 <sup>***#SS&amp;&amp;</sup>
ET/CGRP	9	2.62±0.34	2.85±0.39	2.94±0.42	3.27±0.38 <sup>*#S</sup>	4.15±0.38 <sup>***#SS&amp;&amp;</sup>
ANP(pg · ml <sup>-1</sup> )	9	104.24±40.70	194.92±43.84 <sup>**</sup>	174.11±45.28 <sup>**</sup>	188.68±43.82 <sup>**</sup>	236.50±44.60 <sup>***S&amp;</sup>
HCT(%)	9	45.63±4.01	48.50±4.30	45.36±4.35	46.52±4.45	45.43±4.68
Hb(g · dL <sup>-1</sup> )	9	16.30±1.40	15.60±1.30	15.90±1.24	14.80±1.60	15.60±1.35
La(mmol · L <sup>-1</sup> )	9	1.50±0.40	1.60±0.30	1.50±0.40	1.60±0.30	1.50±0.30

\* : p<0.05 \*\* : P<0.01 denotes significantly different from 11 May

# : p<0.05 ## : p<0.01 denotes significantly different from 29 May

\$ : p<0.05 \$\$ : p<0.01 denotes significantly different from 15June

& : p<0.05 && : p<0.01 denotes significantly different from 15July

4. Table 2 also showed that by carrying out intensity training, the ratio of ET/CGRP had a trend of going up gradually like the ET changes and the last two sampling points (15 July and 12 August) the differences showed significance.

5. ANP levels were significantly enhanced ( $p < 0.01$ ) from early days of intensity training (29 May), after that time it was continuously, significantly higher than pre-training (15 May) ( $p < 0.01$ ). The last sampling point (12 August) the ANP level was significantly higher than the second, and third points from the bottom (15 June and 15 July) (table 2).

6. The coefficient between ET and CGRP showed a significant negative correlation ( $r = -0.9646$ ,  $p < 0.01$ ); CGRP and ANP also showed a significant negative correlation ( $r = -0.8752$ ,  $p < 0.01$ ); and ET and ANP showed a significant positive correlation ( $r = 0.7856$ ,  $p < 0.01$ ).

7. During the intensified training period there are no changes of athletes' Hb concentrations, HCT percentage, La concentration (table 2) and ECG.

## Discussion

The present study provides evidence that successive intensified training both elevate systolic blood pressure and RPE. Urine protein was less responsive, but significant increase was observed in systolic blood pressure during the intense training period. Although the Wushu Sanda athletes in this study showed no decrement in performance, together with the coach's subjective assessment, it was suggested that some of athletes did not tolerate the increased training intensity. An increase in resting heart rate has been suggested as a good indicator of over-training<sup>(6)</sup>. Dressendorfer et al (1985)<sup>(9)</sup> reported 10beats·in<sup>-1</sup> increases in the resting heart rate of distance runners during repeated days of a 500 km run, which they attributed to cardiac fatigue. However, heart rate did not sufficiently increase in the present study to draw any conclusions on the relationship between increased training stress, RPE and endocrine indexes.

Intensity training alters functional control of the coronary circulation. Up to now, ET is known that the most intense contractive effect on blood vessels, and has the most long-term actions. In addition, ET can also promote some neuro-endocrine functions. Some peptides are released with stress. Recent work has approached this area using ex vivo coronary arterial preparations (proximal coronary arteries, near-resistance arteries, resistance arterioles) isolated from exercise-trained animals and contracting independently of confounding in vivo influences. The combined results of these studies indicate that training-induced alterations in vascular control mechanisms do not occur uniformly throughout the coronary vascular tree<sup>(6)</sup>. Shi et al (1999)<sup>(4)</sup> from the radioimmunoassay measured the 75 untrained young men and athletes' plasma ET and CGRP. Their results showed that the basic levels of plasma ET and CGRP in athletes were significantly higher than those in the ordinary young men. But some authors reported changes of ET levels that were different in high loading exercise. Appenzeller et al (1992)<sup>(7)</sup> examined effects of different exercise stress at low and moderate altitudes and after heat stress on beta-

endorphin and endothelin in the human circulation. Their study concluded that ET, a powerful vasoconstrictor, is increased independent of type of exercise, duration and moderate hypoxia. We have not any clues to consult the meaning of Wushu Sanda athletes' plasma ET, but in our study, during intensity training, Wushu Sanda athletes' ET level had an enhanced tendency, but the last two sampling points (15 July and 12 August), changes were marked significant ( $p < 0.05, p < 0.01$ ). In basic theory, on the one hand, in acute exercise, intensifying body sympathetic nerve excitement enhanced the secretion of adrenal gland to elevate plasma catecholamine and hypertension levels which activate ET concentration in plasma; however, while prolonged intensity exercise, the body tissues, especially skeleton and cardiac muscle, they in anoxic, damaging positions, also to be factors of enhancing ET level. In normal conditions, ET has no pathological effects, but while tissue ischemia and anoxic, endotheliocyte released plenty of ET to contract vessel and furthermore ET proliferate endotheliocyte to work a vicious spiral. It is told that the over-training elevates the plasma ET level, this training effect on ET sensitivity lasted for a long time. This is in agreement with above discussion.

CGRP, the bioactivated peptide, was found by genetic engineering. Up to now, CGRP is the most powerful substance to dilate vessels in the body. Brooks et al (1990)<sup>(8)</sup> reported that in the sprint study times fold increase in plasma adrenaline (AD) occurred with the peak concentration observed after short time recovery sprints. The maximum noradrenaline (NA) concentration also occurred after sprints. No changes in CGRP were detected in response to the exercise. Their results indicated that there was no evidence for a role of CGRP in the control of the cardiovascular system after brief intermittent maximal exercise but has differences between men and women in performance and hormonal responses. Jin Qiguan et al (1998)<sup>(9)</sup> used the SD rats to establish overload training model based on swimming training. Their results indicated that overload training may promote the secretion of ET while restrain the secretion of CGRP, which may cause a secreting dislocation of ET and CGRP, this would have been the pathophysiological mechanism for hypertension. The performance of cardiovascular hormones is a poor understanding field in nonperiodic (running and swimming is periodic event) events such as Wushu Sanda. However, our results showed that the early stages of intensity training (29 May), although no significant differences appeared ( $p > 0.05$ ), the changes of plasma CGRP level was lowered gradually. With continuing intensity training, we watched the CGRP level maintains to decrease ( $p < 0.05, p < 0.01$ ). It is reported that the long-term exercise reduces CGRP level by enhancing plasma catecholamine secretion<sup>(10)</sup>. It was also said there are ET and CGRP dislocation, which was caused by long-term intensity training. Our study also showed that with carrying out intensity training, the ratio of ET/CGRP tended to of increase gradually to like the ET changes and the last two sampling points (15 July and 12 August) the differences showed significance ( $p < 0.05, p < 0.01$ ). It is clear that the effect of ET is contrary to the process of CGRP. This study found that there was a negative correlation between ET and CGRP changes ( $r = -0.9646, p < 0.01$ ). It points to the mismatching of the secretion of ET and CGRP in high load

exercise. Further study needs to look at this dislocation of ET and CGRP changes to determine whether or not it would have been a main factor of pathophysiological mechanism for hypertension.

ANP intervenes in the cardiovascular adjustments to exercise<sup>(11,12)</sup>. In Brooks' study the peak ANP concentration occurred immediately post-exercise<sup>(8)</sup>. Chang Yun et al (1998)<sup>(12)</sup> reported that there were differences in secreting area and releasing level of cardiac hormones in different kinds of the athletic heart. The cardiovascular hormones from the athletic hearts were mainly secreted from atria tissue while those of static athletic heart were mostly secreted from ventricular tissue. Changes of ANP level in endurance athletic heart was more significant than in static one. Their study leads to the conclusion that endocrine changes of endurance athletic hearts have essential significance for the enhancement of myocardial contractility, aerobic endurance and reserve. The endocrine changes of static athletic heart play an important role for regulating myocardial hypertrophy. This study showed that at early days of intensity training ANP level was enhanced significantly ( $p < 0.01$ ) (29 May), after that time it was continuously significantly higher than pre-training (15 May) ( $p < 0.01$ ). At the last a sampling point (12 August) ANP level was significantly higher than the second, third points from bottom (15 June and 15 July). The result suggest that long term high intensity training would have a affect on athletes' hearts in that it enhances its secretion, meanwhile the performance of catecholamine and other factors also tamper with change of ANP levels. Authors described in literature the increase in plasma a-ANP was correlated with alterations in plasma cGMP, plasma lactate, hematocrit, and body weight<sup>(13)</sup>. In our study CGRP and ANP was showed significant negative correlation ( $r = -0.8752, p < 0.01$ ); and ET and ANP was showed significant positive correlation ( $r = 0.7856, p < 0.01$ ). But there was no change in hematocrit, and then we concluded that the changes of cardiovascular hormones were not related to blood concentration.

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