

# Readiness potential in different states of physical activation and after ingestion of taurine and/or caffeine containing drinks

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**Summary.** To investigate the influence of taurine and caffeine containing drinks and physical stress on the cortical movement-preparation, the readiness potentials or "Bereitschaftspotentiale" (BPs), preceding voluntary self-placed pedalling movements, were examined after different states of exhaustion on an ergometer. 15 (13 right-handed) healthy men, aged between 22–30, participated in a randomised, cross over, double-blind, placebo controlled study.

BPs were averaged out of artefact free EEG-segments from more than 90 triggered events, measured at 17 electrodes of the 10:20 system. With increasing effort the BPs were enlarged differently depending on the drink consumed. In placebo trials after exhaustive exercise premovement negative potential curves could be seen even in frontal areas. With caffeine the BPs increased after lower workload, achieving a level, which was reached in the placebo trials only after submaximal physical activation. Furthermore a significant shortening of premovement-brain-potentials in frontal and parietal regions could be seen in the caffeine trials at rest. Taurine admixture seems to inhibit this effects.

**Keywords:** Amino acids – Bereitschaftspotential – Readiness potential – Movement-preparation – Taurine – Caffeine – Exercise

# Introduction

The "Readiness potential" or "Bereitschaftspotential" (BP), (firstly described by Kornhuber and Deecke, 1964), precede initiation of voluntary movements (Kornhuber and Deecke, 1965; Deecke et al., 1976). It was suggested that sensor-, association- and motivation-areas were involved during the information processing in motor systems (Deecke, 1990; Schober, 1987; Deecke and Kornhuber, 1977).

The use of "Red Bull®", a taurine and caffeine containing drink, is wide-spread in sports. Pilot studies have shown an improvement of physical capacity (Geiß et al., 1994) and well-being. Taurine as well as caffeine may act both

at the cardiocirculatory level and the central nervous level. Thereby an influence on physical performance is possibly due to somatic and/or psychophysiological factors. In this study we examined the influence of taurine and/or caffeine containing drinks on the cortical movement preparation in different states of physical stress.

The hypothesis was: if taurine affects the improvement of sensomotor and motivational control of movement programming would it have an influence on the readiness-or "Bereitschaftspotential" as well as on attention and concentration.

## Material and methods

# **Subjects**

15 endurance trained men (343  $\pm$  32 Watt maximal pretest-power (PPO) on an ergometer), 13 of them right-handed (Edinburgh Handedness Inventory, Oldfield, 1971), 26  $\pm$  3 years, 178  $\pm$  5,6 cm, 71,8  $\pm$  3,6 kg participated in this study. After information about the test conditions the subjects gave their written consent.

# Design

48h prior to any test medicaments, alcohol, nicotine were not allowed, 24h prior to the tests stimulants like coffee, tee, chocolate and training were excluded. 3h before the tests a standardized breakfast (bread, butter, mineral water or fruit-tee) was given. 40 minutes prior to the tests one of the test drinks which had the same colour and taste were consumed in a randomized double blind order. The tests were performed isochronous and weekly. In the duration of the 3 trials the probands did not change their usual activities and habits of eating which were recorded in a diary. To exclude the probable influence on recovery time during the measurements to the results, it was standardised.

The applicated testdrinks (500ml) had the following compositions:

- D1: "Placebo" = "Red Bull®" without taurine, without glucuronolactone, without caffeine, with glucose (10,5g), with saccharose (43g)
- D2: "Verum" = "Red Bull®" original drink containing taurine (2g), glucuronolactone (1,2g), with caffeine (160 mg), with glucose (10,5g/l), with saccharose (43g)
- D3: "Control" = "Red Bull®" without taurine, without glucuronolactone, with caffeine  $(160 \,\mathrm{mg})$ , with glucose  $(10.5 \,\mathrm{g})$ , with saccharose  $(43 \,\mathrm{g})$

#### Test

The exercise intensity was determined one week before the trials in a pre-test. Derived from there, the subjects cycled with following intensity and duration (Fig. 1):

- 1. Ten minutes warm-up, lactic acid levels lower than 2 mmol/l (27%  $\pm$  6% of max. power)
- 2. Six minutes with lactic acid levels at 2mmol/l (44%  $\pm$  5% of max. power)
- 3. Six minutes with lactic acid levels at about 3 mmol/l (60% ± 5% of max. power)
- 4. Six minutes with lactic acid levels at about 5 mmol/l (77%  $\pm$  4% of max. power)
- 5. Six minutes with lactic acid levels at about 7 mmol/l (94%  $\pm$  4% of max. power)

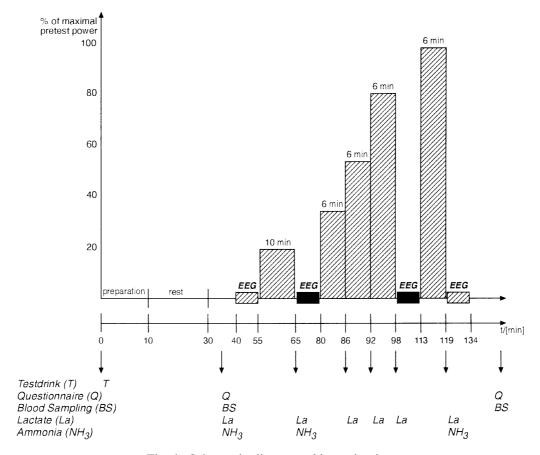
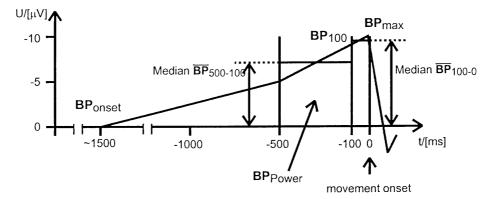


Fig. 1. Schematic diagram of investigation

# Electrophysiological methods

During the EEG measurements the subjects were seated comfortably on the bike ergometer and their feet rested on special switch-blocked pedals which also triggered the movement onset at a threshold force of 171 N (or a torque of 29,07 Nm) (Force/Displacement Transducer, Typ 120T-B, Fa. KYOWA). The kick-force-curves were quantified by DIADEM-Software (University of Paderborn). During the series of kick-movements the eyes were open and fixed to a point. To exclude artefacts from the eye muscles the subjects were instructed and trained to reduce eye-blinks. The BPs, related to voluntary, self placed kick-movements of the right leg, were examined after periods of rest, warm up, submaximal- and maximal stress (see Fig. 1). The EEG was recorded by an electro cap (Electro Cap, Co., USA), as recommended by Blom and Anneveldt (1982). This cap includes 17 electrodes, positioned according to the international 10:20 system (Jasper, 1958), with central electrode (Cz) as physical reference. This electrode cap was not removed between recording periods. EOG and ECG were recorded too. The BP was averaged out of EEG segments triggered by 180 kick-movements. The signals were fed into a battery-powered amplifier and A/D converter, (AC =  $10M\Omega$ ; DC =  $20M\Omega$ / 512 Hz/12 bit) (MediSyst GmbH, Linden, Germany), placed near the subject. The time constant of the amplifier was set at 3s for movement-related cortical potentials (Desmedt, 1977a). Before the recording period an impedance-test (electrode impedances  $<50 \text{k}\Omega$ ) ensured a sufficient signal to noise ratio. An optical fiber transferred the digitalized signals trouble free to the CATEEM-CATERPA-System [(CATEEM = Computer Aided



**Fig. 2.** Schematic BP characterization for frontal, central, as parietal positions. It means: BP- $P_{ower}$  area between BP-waveform (from defined onset to trigger) and baseline;  $BP_{max}$  maximal amplitude of the BP-waveform;  $BP_{100}$  amplitude of the BP-waveform 100 ms before trigger;  $BP_{500}$  amplitude of the BP-waveform 500 ms before trigger; Median  $B\bar{P}_{100-0}$  median averaged BP-amplitude the last 100 ms before trigger; Median Median median averaged BP-amplitude between 500 ms and 100 ms before trigger

Topographical Electro-Encephalometry Measurement) (CATERPA = Computer Aided Topographical Events-Related Potential Analysis)]. The duration of the BP acquisition tooks 15 minutes (3 minutes to prepare and impedance-test and  $4 \times 3$  minutes for the BP measurements). Only artefact free EEG segments (>16) were averaged in the range between 2000 ms before and 1000 or 10 ms after the trigger. The BP-wave measurements were related to a relative baseline (2000 ms to 1700 ms prior the trigger). According to Kristeva and Kornhuber (1980), Kornhuber and Deecke (1965), Cui et al. (1999) we indicated the premovement negative brainpotential waveformes between the first derivation from the baseline (onset) up to the trigger as BP.

Because of artefacts one subject had to be excluded from the evaluation of the BPs. The results were given as brain-map and waveformes in grand average (BPs of all subjects) for *qualitative analysis*. The *quantitative analysis* (BP of each subject) was focused on midline frontal, central, parietal (Fz, Cz, Pz) areas. Characteristical curve parameters were measured like shown in Fig. 2.

## Determination of metabolic measurements

In order to quantity the physical and metabolic strain, heart rate (Polar Sport Tester TM, Fa. Polar, Gross-Gerau, BRD), blood lactate (Laktat für die Sportmedizin® Boehringer Mannheim, EPOS analyzer Eppendorf, Hamburg, BRD) and blood ammonia (ammonia checker distributed in Germany by HEk Pharma Lübeck, BRD) were measured from hyperemized ear lobe capillary blood and amino acids (HPLC-Fluorescence detection, BIO-TEK Kontron instruments, OPA-method) from venous blood as shown in Fig. 1. Influences of the heart rate measurement on EEG recording could be excluded as shown in a pre-test.

# Statistical analysis

For statistical calculation the SPSS-software was used. The significance of the data was validated by MANOVA (parametric analysis of variance) and the T-Test (post-hoc) or by Friedman (nonparametric analysis of variance) and Wilcoxon test (post-hoc), using a significance level of  $p \le 0.05$ . Results are given as mean and standard deviation.

#### Results

The heart rate, blood lactate, -ammonia concentrations showed typical increasing values in ergometer step tests with increasing work loads and were not effected by the testdrinks.

The MANOVA of plasma amino acids showed significant differences between testdrinks only in taurine concentrations [p < 0.001]. The post hoc test detected a significant increased taurine concentration at *rest* and after *exercise* in the *Verum* trial (see Fig. 3).

The quantification of the force-time curves from the trigger pedal mechanism showed no differences between *rest* and *exercise* nor between test trials.

# Qualitative findings

The movement related slow brain potential curves in Fig. 4a–b showed differences between the test drinks. The maps refer to the medium amplitude 100 ms before movement initiation and indicates differences between test trials in regional distribution and magnitude at *rest* and after *exercise*.

At rest with Placebo the map showed an asymmetrical distribution of negative potentials over the right frontal area and a symmetrical distribution from the central to the parietal cortex. The maximum was at the vertex. The negativity was stronger in the Placebo than in the Verum or Control trials. This is supported by the BP wave forms, which were more negative in Placebo trials at frontal, central and parietal areas.

With *Verum*, negativity was lower and less asymmetrically distributed to the right frontal areas. The *Control* situation showed an asymmetrical distribution to the left frontal cortex and the negativity was similar to *Verum* trial (Fig. 4a).

After maximal exercise the map showed more pronounced differences between the trials. In the *Placebo* trial the map showed a widespread symmetry from parietal to central and frontal areas. In the *Verum* trial there was a symmetrically focused negativity on parietal, central and frontal areas. The *Control* trial showed a more parietal negativation. The maximum was at the vertex in all measurements, but in the *Placebo* trial it extended to frontal, and

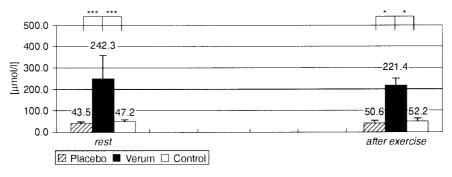
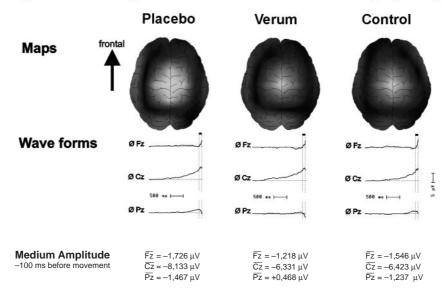
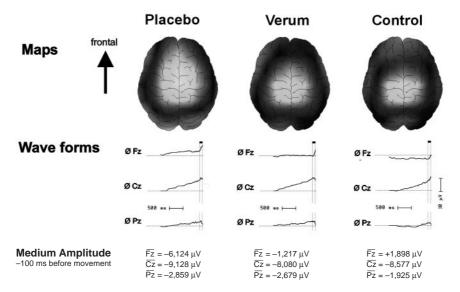


Fig. 3. Taurine concentration  $[\mu \text{mol/l}]$  at rest and after exercise in *Placebo*, *Verum* and *Control* trials

# a) Bereitschaftspotential at Rest in Grand Average (N=14)



# b) Bereitschaftspotential after exercise in Grand Average (N=14)



**Fig. 4a–b.** Grand average of Bereitschaftspotential preceding kick-movements on a bike ergometer at the 3 implied test trials (*Placebo*, *Verum*, *Control*) at rest and after maximal exercise. Numerical data of medium amplitudes averaged from the last 100ms before movement are given additionally. The brain maps are diagrammed in a "glow mode" colour version but in this illustration black/white: negative potentials are defined in blue colours (brighter areas in the center of the brainmaps) and positive potentials in red colours (slightly brightened marginal areas), increasing potentialamplitudes are represented in the brightness of the colours. Wave forms show the grand average brain potentials  $-2000 \, \text{ms}$  (rest) or  $-1500 \, \text{ms}$  (after exercise) prior movement onset related on a relative baseline. The potentials are built up with an inverse scale (upwards = negative, downwards = positive)

in the *Control* trial to parietal areas. This findings were supported by the wave forms. In the *Placebo* trial after *exercise* there was found a characteristic BP-curve in Fz, Cz and Pz while in the *Verum* trial only Cz and Pz showed a BP-curve. In the *Control* situation a BP-curve was only found in Cz position (Fig. 4b).

# Quantitative findings (Table 1)

The MANOVA detected some effects of physical stress in the BP characterization in frontal [Fz500, p = 0,01, Fzmax, p = 0,049], central [Czmax, p = 0,007, Czonset, p = 0,003] and parietal [Pz100, p = 0,035, Pz500, p = 0,025] areas. The Friedman test showed an effect caused by stress in the *Verum* [PzPower, p = 0,007], *Control* [CzPower p = 0,019; Pzonset, p = 0,005], and *Placebo* trial [CzPower, p = 0,038].

Differences caused by the test drink were found in Fzonset, p = 0,009 (*Verum/Control*) and Pzonset, p = 0,005 (*Control/Placebo*) and p = 0,012 (*Verum/Control*) (Table 1).

The BP characteristics indicated incremental physical stress by increasing amplitudes, in the *Placebo* trial at frontal, central and parietal regions significant increases of Fzmax, Fz500, Czmax, CzPower, Pz100, Pz500. The test with *Control* showed a more increased negativity particularly after the *warm up* in the central and parietal cortices, significantly indicated by Czmax, CzPower, Pz100, Pz500. The *Verum* trial showed similar behavior compared to the *Control* condition, but particular in the *warm up* situation with reduced BP amplitudes, so that the BP increase more continuously after incremental physical stress. The increases of BP amplitudes were significant for Fzmax, CzPower and PzPower (Table 1).

The questionnaire did not reveal any significant differences but the subjects stated to feel better with *Verum* at *rest* and after *exercise*. This is represented by the distribution of answers to the question after *maximal stress*: "Are you able to manage another step with increasing intensity?". In the *Placebo* trial 8 subjects felt totally exhausted and only 4 felt able to carry on.

In the *Verum* and *Control* trials 8 and 9 subjects respectively felt able to cycle an increasing step, and only 2 and 1 respectively were exhausted (Fig. 5).

#### **Discussion**

The Bereitschaftspotential preceding a cycling kick-movement was successfully represented under Placebo, Verum and Control conditions after different states of physical activation. As is already well known from event-related potential studies, it starts 1000–1500ms before movement beginning with a bilateral distributed slightly negative shift, which increases progressively. The maximum level is reported to be at the midline close to the vertex (Kornhuber and Deecke, 1965, 1980; Deecke, 1990). Our qualitative and quantitative results agree with previous BP-studies on foot- and leg-movements (Böcker et al., 1994; Boschert and Deecke, 1986; Shibasaki et al., 1981). The BPs in

**Table 1.** Mean and standard deviation of the "Bereitschaftspotential" (BP) at Fz, Cz, Pz electrode positions

positions				
Parameter	Mz	Placebo	Verum	Control
Fzonset	R	$-677.3 \pm 627.3$	$-1019.9 \pm 300.9$	$-378.5 \pm 551.4^{\circ}$
[ms]	W	$-1406.3 \pm 456.5$	$-798.4 \pm 547.4$	$-848.3 \pm 862.4$
	4	$-1266.3 \pm 186.6$	$-695.2 \pm 502.9$	$-767.3 \pm 666.7$
	5	$-1197.1 \pm 234.9$	$-768.4 \pm 657.3$	$-444.9 \pm 593.7$
Fzmax	R	$-5.1 \pm 3.6$	$-4.0 \pm 4.3$	$-6.4 \pm 3.5$
$[\mu V]$	W	$-6.3 \pm 4.2  * **$	$-4.8 \pm 3.0  _{*} _{*}$	$-8.3 \pm 5.6$
	4	$-8.3 \pm 4.0  $	$-6.6 \pm 3.6$ $\rfloor$	$-6.8 \pm 4.0$
	5	$-8.9 \pm 3.9$	$-6.1 \pm 4.3$	$-5.3 \pm 2.9$
$Fz_{500}$	R	$0.3 \pm 2.2$	$0.2 \pm 1.7$	$0.3 \pm 1.4$
$[\mu \tilde{\mathbf{V}}]$	W	$-1.0 \pm 3.2  *  **$	$-0.1 \pm 1.2$	$-1.4 \pm 2.9$
	4	$-1.9 \pm 3.3$	$-0.7 \pm 3.7$	$0.6 \pm 2.4$
	5	$-3.3 \pm 4.1$	$-1.2 \pm 3.5$	$1.6 \pm 4.0$
PzPower	R	$10.0 \pm 12.4$	$8.1 \pm 10.6$	$10.4 \pm 8.7$
$[\mu V^2]$	W	$16.8 \pm 22.7$	$5.0 \pm 2.5$	$15.4 \pm 11.0$
[66 4 ]	4	$20.1 \pm 15.6$	$18.0 \pm 22.0$	$12.1 \pm 8.8$
	5	$37.9 \pm 44.3$	$27.2 \pm 26.0$	$23.5 \pm 39.1$
Cannat		$-1446.1 \pm 295.9$	$-1538.5 \pm 264.4$	$-1444.2 \pm 343.9$ <sub>7</sub>
Czonset	R W	$-1440.1 \pm 293.9$ $-1264.5 \pm 831.3$	$-1338.3 \pm 204.4$ $-1287.0 \pm 863.2$ *	$-1444.2 \pm 343.9$ $-1629.2 \pm 268.1$
[ms]	4	$-1204.5 \pm 851.5$ $-1290.6 \pm 231.0$	$\begin{bmatrix} -1287.0 \pm 803.2 \\ -894.8 \pm 1,031.1 \end{bmatrix}$	$-1029.2 \pm 208.1 \\ -1308.4 \pm 253.7 $ *
	5	$-1290.0 \pm 231.0$ $-1111.8 \pm 783.0$	$-894.8 \pm 1,031.13$ $-1280.5 \pm 218.0$	$\begin{bmatrix} -1308.4 \pm 233.7 \\ -1194.3 \pm 306.1 \end{bmatrix}$
Czmax	R	$-10.3 \pm 6.0$	$-9.6 \pm 5.7$	$-10.0 \pm 6.9$ ]*
$[\mu V]$	W	$-10.4 \pm 5.3$ *	$-10.5 \pm 5.7$	$-12.2 \pm 8.6$ ]*
	4	$-12.5 \pm 6.1$	$-12.0 \pm 6.9$	$-10.7 \pm 7.5$
	5	$-12.4 \pm 7.1$	$-11.6 \pm 8.2$	$-11.8 \pm 7.1$
CzPower	R	$41.6 \pm 58.2$	$33.7 \pm 42.9$ 7	$32.4 \pm 41.0$
$[\mu V^2]$	W	$43.6 \pm 43.6  _{*}$	$43.2 \pm 56.8 \mid *$	$67.3 \pm 88.7  \text{l} **  *  *$
	4	$60.9 \pm 54.5$	$66.1 \pm 72.6$ $^{\downarrow}$	$57.4 \pm 85.4$
	5	$77.4 \pm 97.6^{ m J}$	$65.6 \pm 75.4$	$62.2 \pm 65.0$
Pzonset	R	$-906.8 \pm 479.4$	$-919.3 \pm 552.5$	$-378.2 \pm 247.0^{2})^{3}$
[ms]	W	$-1356.2 \pm 459.3$	$-1131.9 \pm 436.5$	$-1241.6 \pm 585.7$
	4	$-980.4 \pm 446.2$	$-1818.0 \pm 2{,}725.1$	$-791.2 \pm 549.6$
	5	$-1143.2 \pm 231.9$	$-1088.3 \pm 447.6$	$-565.9 \pm 558.3$
$PZ_{100}$	R	$-2.3 \pm 1.5_{7}$	$-2.2 \pm 2.0$	$-1.8 \pm 1.6$ <sub>]*</sub>
$[\mu V]$	W	$-3.2 \pm 3.2$ *	$-3.8 \pm 3.5$	$-4.5 \pm 3.9$ ]*
	4	$-4.6 \pm 4.1$	$-3.7 \pm 4.2$	$-2.7 \pm 3.3$
	5	$-2.3 \pm 4.1$	$-3.4 \pm 5.3$	$-1.8 \pm 4.2$
$PZ_{500}$	R	$-1.7 \pm 1.3_{7}$	$-1.3 \pm 1.5$	$-1.1 \pm 1.2$ 7*
$[\mu V]$	W	$-2.8 \pm 3.7$ *	$-3.0 \pm 3.3$	$-3.7 \pm 3.2$ ]*
n 1	4	$-4.1 \pm 4.0$	$-2.9 \pm 3.2$	$-1.7 \pm 3.0$
	5	$-1.7 \pm 3.7$	$-2.6 \pm 4.7$	$-0.9 \pm 4.2$
PzPower	R	$5.1 \pm 4.0$	5.7 ± 4.7	$8.1 \pm 7.5$
$[\mu V^2]$	W	$14.3 \pm 17.3$	$15.2 \pm 31.3$	$29.3 \pm 37.0$
[h , ]	4	$26.2 \pm 33.5$	$20.6 \pm 33.3$ $+$ $+$	$11.3 \pm 12.1$
	5	$24.6 \pm 30.9$	$20.0 \pm 35.3 \pm 26.2$ $\pm 26.2$	$14.2 \pm 17.7$
		21.0 = 30.7	25.1 = 20.2	11.2 - 1/./

R Rest; W Warm up; 4 submaximal charge; 5 maximal charge. \*<0.05 weak significant; \*\*<0.01 significant; \*\*\*<0.001 high significant (T-test). +<0.05 weak significant; ++<0.01 significant (Wilcoxon).  $^2$ )<0.01 significant between Placebo and Control.  $^3$ )<0.05 weak significant between Verum and Control

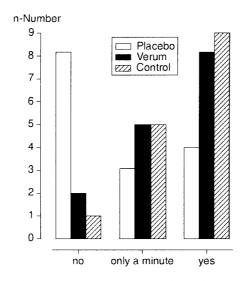


Fig. 5. Question: "Are you able to cycle an increasing step?"

context with either different states of physical activation or with ingestion of taurine and/or caffeine containing drinks have not been previously investigated. Some BP-measurements were enlarged with increasing physical activation (Table 1). In the Placebo trial the BPs are widespread, and are distributed in the frontal areas with higher amplitudes than in the Verum or Control conditions. Biomechanical reasons (different force or speed of the test movement) which attend bigger BPs (Wallenstein et al., 1995) could be excluded in our study.

It would indicate that when exhaustion communised it was necessary to have more volition in movement preparation to attain the same performance. This is indicated by the different expansion of the BP to frontal, central and parietal regions (Fig. 4b Placebo trial) and is in agreement with the assumption that more intention is attended by a larger BP (Rockstroh et al., 1989). In the Control condition compared with the Placebo or Verum trial the BP onset is clearly later (shorter) in frontal and parietal areas. In the Control trial the BP-dimensions reached after warm up was already a level which is needed in the Placebo condition initially after submaximal stress (Table 1. Fz-, Cz-, PzPower). In the Verum trial this "overshoot" does not appear. This may be due to an inhibitory or modulatory effect of taurine (Hashimoto-Kitsukawa et al., 1988). Although a better exercise performance with caffeine was achieved in the study of (Cole et al., 1996) which resulted in a better subjective condition after exercise (Fig. 5). However the standardization of the test trials in our study and the results of metabolic and cardio-circulatory parameters, showed no differences between the testdrinks, which would not support such a caffeine-included effects.

With consideration to the hypothesis, that movement patterns are influenced by emotion and motivation in the frontal areas and that the parietal

areas are involved in coordination of expected sensomotor feedback (Deecke et al., 1985; Roland, 1985), the following interpretation could be discussed.

In the Control trial caffeine influences the continuity of increasing BP with physical stress. In Verum condition taurine prevents the described "overshoot". In the Placebo trial the increasing exhaustion requires a higher mental effort, which involves particularly the frontal regions for producing the same movement performances. With taurine (Verum) an increasing mental effort is not necessary, because the preparation is optimized in directly movement related brain regions. This interpretation is supported by a better subjective condition with taurine in the Verum trial.

#### **Conclusion**

In different states of physical stress the cortical movement programming is influenced by caffeine (Control) and a combination of caffeine and taurine (Verum) in a different manner, this is indicated by a different distribution and magnitude in premovement negative brain potentials.

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