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"CO_2PING": ACUTE MOUNTAIN SICKNESS, STRESS AND THE ROLE OF THE RIGHT CEREBRAL HEMISPHERE

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Abstract

Introduction: The concept of stress has been introduced in psychology and psychosomatic medicine in order to describe the response of biological systems to different types of strain and pressure. Mild oxygen deprivation (hypoxemia) is a vital physical stressor which triggers an alarm reaction. The adrenal hypothalamus-medulla system is being activated along the fight-or-flight stress axis. Research has shown that the right cerebral hemisphere is predominant in orchestrating the primary alarm reaction.

Aim of the study: This study aimed to evaluate the effect of the personality dimension trait-anxiety (ANXT-factor) on processing the stressor hypoxemia under field conditions.

Methods: Prior to the expeditions to Imja Tse (n=14) and Chulu West Peak (n=14) an extensive anamnesis of each participant was written and the individual ANXT-factor was determined using the STAI. After having crossed the threshold-altitude (2500 m), the Lake Louise Score was applied (evening and morning protocol) to quantify symptoms of AMS. A sum score ≥3 was considered symptomatic. Additionally heart rate (HR) and peripheral oxygen saturation (SpO_2) were daily measured by pulse oximetry.

Results: In the Chulu-group ANXT-percentile and LL-AMS-sum score were highly related (r=0.82, t=5 which exceeds 4.318, the critical value of t for $P<.001$). In the Imja-team r=0.74 ($P<.01$; evening protocol). HR and likewise SpO_2 had insignificant relations with ANXT and AMS-scores.

Conclusions: The study indicates a direct link between AMS and the personality dimension trait-anxiety. The symptoms of acute mountain sickness are to be considered as the neuro-psycho-somatic stress response to mild oxygen deprivation orchestrated by the right cerebral hemisphere. This article presents a stress-model based on those findings.

Key words: *hypoxemia, mild AMS, vegetative nervous system, neurotic trait, stress model*

Introduction

Stress is a complex endeavour aimed to re-establish the pre-existing neuropsychosomatic balance. Burdens and strains are answered by the organism by responses trying to adapt to the challenges of everyday life. The mechanisms of stress management may include physical, emotional, behavioural or cognitive processes and are to be regarded as coping measures in the sense of habitual personality traits that are relatively stable over time. Stress is triggered by threatening conditions and active agents called stressors that can be of physical nature like coldness, heat, noise or toxic substances (e.g. cigarette smoke, drugs, pesticides). Psychical factors, for instance certain attitudes, anticipations, anxieties and apprehensions may equally function as emotional stressors. In the neurochemistry of the stress response adrenal hormones (epinephrine, norepinephrine, corticosteroid hormones) are being activated. Somatic stress parameter primarily engage vegetative reactions of the sympathetic nervous system encompassing cardiovascular, respiratory, gastrointestinal, renal and endocrine changes.

In the course of the 20th century various stress theories have been developed to explain the relationship between stressor and stress reaction. According to Walter Bradford Cannon's acute stress response or alarm model [1] the organism reacts under certain conditions, "quick as lightning", by means of fight-or-flight readiness. Hans Selye broke the effects of stressors into three stages: alarm, resistance, exhaustion. In 1936, he described this staged response as general adaptation syndrome, or GAS [2]. Selye published in 1975 a model dividing stress into eu- and distress [3]. Where stress enhances function (physical or mental) it may be considered eustress. However, persistent stress that is not resolved through coping or adaptation, deemed distress, may lead to anxiety or withdrawal (depression) behaviour.

In the model of Henry & Stephens [4], according to the stressful situation, specific neurophysiological reactions are distinguished: along the fight-or-flight stress axis a threatening situation causes an activity boost (arousal) in the sympathetic, adrenal medulla system that is followed by a release of the hormones epinephrine and norepinephrine. Anger and annoyance leads to a rise in norepinephrine and testosterone. Loss of control (defeat, subordination) however triggers along a second stress axis the hypophysis, adrenal cortex system, causing a rise in cortisol and a drop in testosterone. Inhibition (of the recticular activation system) and depression are the consequences.

Stressors can be classified in: physical (1st), emotional (2nd) and cognitive (3rd order). Acute oxygen deprivation (HYPOX) means a serious threat to an organism. HYPOX must be regarded as a vital stressor of the 1st order. A number of coping mechanisms are automatically activated by it. HYPOX produces an acute stress reaction with the aim of guaranteeing or restoring adequate oxygen- and energy supply of primarily the brain. This aim is sought after by means of cerebral autoregulation. According to the *Selfish Brain Theory,* the brain covers its own, comparably high energy requirements with the utmost of priorities when regulating energy fluxes in the organism, usually at the cost of other organ systems. The brain behaves selfishly in this respect [5].

In agreement with the stressor category different brain regions are involved in the process of reducing the effects of a given strain. A stressor of the 2nd order will activate the amygdala and hippocampus, while a cat.-I type stressor like HYPOX is going to trigger an acute, strictly unconditioned thalamic-hypothalamic reaction. Increased sympathetic activity and endocrine changes are the effect.

Functional Asymmetry

Possessing more white substance, the right cerebral hemisphere (RCH) is usually a little larger and heavier then the left hemisphere (LCH). Further on, the temporal lobes show pronounced asymmetry which corresponds heavily to the functional asymmetry of the thalamus in the diencephalon.

The valence hypothesis determines that the LCH is dominant for positively, the RCH for negatively tinged emotions, feelings and body sensations. The RCH's predominance concerns rather basal aspects like autonomous reactions (e.g. blood pressure) on emotional stimuli and stressors. Work by Otto et al. clearly proves that the RCH is becoming activated by aversive conditions such as pain [6-8]. Patients with lesions of the RCH tolerate pain longer than controls when compared to patients with left hemispheric damage. Compared to the left thalamus, the right thalamus is more intensely activated by negative experiences. The amygdala, a limbic structure in the anterior part of the temporal lobe and centre of the "inborn"-affective functions, shows a comparable asymmetry. Stress factors lead to a clearly more intensive activation (respectively overreaction) of the right sided amygdala [9].

HYPOX counts as a strong stressor. In the context of *Project Silverpyramid* [10] it was scrutinized whether under hypoxia the EEG suits as a predictive marker for AMS. At 3450 m and 5050 m Feddersen et al. examined 32 subjects with 24 electrodes applied to the scalp according to the international "10-20" system [11]. Those twelve mountaineers that had signs of AMS at 5050 m, showed a significant increase of power spectral delta activity in the right temporal region (electrode T4) under subacute hypoxemic conditions at 3450 m. The other subjects (non-AMS) showed a decrease of power spectral delta activity in the right temporal region. Additionally in AMS-vulnerable persons significantly higher mean blood flow velocities of the right middle cerebral artery (ACM) were recorded ranging from 51 cm/s at baseline (100 m) to 58 cm/s at 3450 m and 71 cm/s at 5050 m sea level

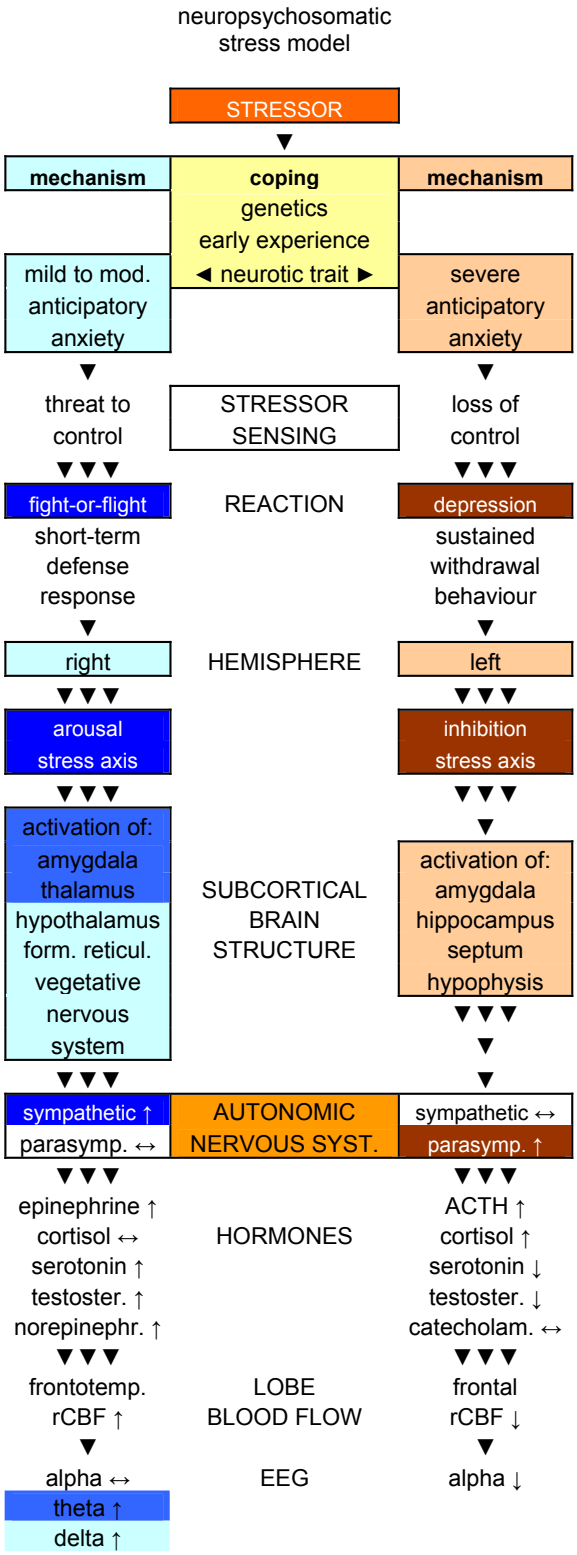

Figure 1. Neuropsychosomatic stress model (NPSS model; R. Waanders)

while no progressivity was found in the left ACM [12]. A quantification of regional cerebral oxygen saturation (rSO_2) by means of transcranial near infrared spectroscopy revealed a reversion of the normoxic "LCH > RCH" model: under hypoxemic conditions initially oxygen saturation was higher in the blood vessels of the right cerebral hemisphere [13]. After adaptation to altitude was accomplished, which took usually some 24 to 48 hours, the "LCH > RCH"-pattern had normalized which means that the left cerebral hemisphere recorded the higher rSO_2 again.

Individual AMS-vulnerability

The brain's response to hypoxemia consists of RCH activation. Besides the anterior temporal lobes the central mechanism comprises the deep, limbic structures of thalamus-hypothalamus, corpus amygdaloideum (amygdala) and hippocampus. Activation of right cerebral structures that are part of the fight-or-flight stress axis will result in vegetative respectively neuropsychosomatic symptoms (Fig. 1). At high altitude these symptoms are known to us as acute mountain sickness (AMS).

People differentiate in their vulnerability regarding the 1st order stressor HYPOX, the individual AMS-susceptibility is not purely of a physical nature. Neuropsychological factors that apply to the emotional, affective structure play an important, probably dominant role in the development of AMS. Top of the list marks the so called anxiety-predisposition or trait-anxiety (ANXT). People with a strong ANXT-personality factor react earlier and altogether more intense to the stressor HYPOX by contrast with individuals having a less pronounced trait-anxiety factor [14, 15].

Methods

The above cognizance lead to the following work hypothesis: The stronger the individual ANXT-factor (measured by a standardized clinical questionnaire; see below), the more pronounced the number and intensity of AMS-symptoms triggered by hypoxemia that acts as a stressor will be.

Prior to the expeditions to Imja Tse (6189 m, Khumbu, Nepal; [10]) and Chulu West Peak (6419 m, Damodar Himal, Nepal, [16]) an extensive anamnesis of each participant was written and the individual ANXT-factor was determined using the STAI (raw score and percentile, German norm data, State-Trait-Anxiety-Inventory; [17]). All subjects were without psychiatric history. They were informed about the study goals and gave their written consent.

During the ascent of Imja Tse 14 Europeans respectively ditto 14 mountaineers during the ascent of Chulu West Peak were scrutinized regarding their acclimatization. After having crossed the threshold-altitude (2500 m), the Lake Louise Score was applied daily twice (evening and morning protocols) to quantify symptoms of AMS [18]. This scale has a range of 0 to 29 and includes both self- and external evaluations. Mountaineers with a sum score ≥3 were considered symptomatic. Additionally heart rate (HR) and peripheral oxygen saturation (SpO_2) were daily measured by pulse oximetry. In cases of urgent need a more comprehensive medical examination could be done.

After having returned again below the threshold-altitude, the average Lake Louise morning sum score and the average evening sum score of all protocols was calculated for each individual as well as the combined morning-evening score.

Results

Four women and 10 men from Austria and Germany make up the Imja Tse-group which is part of *Project Silverpyramid*. Concerning the ANXT-factor of these participants, the median raw score is 32 (50th percentile), the arithmetic ANXT-mean (± 1 SD) is 33.63 ± 7.26 (48th percentile; Tab. 1a) with a minimum

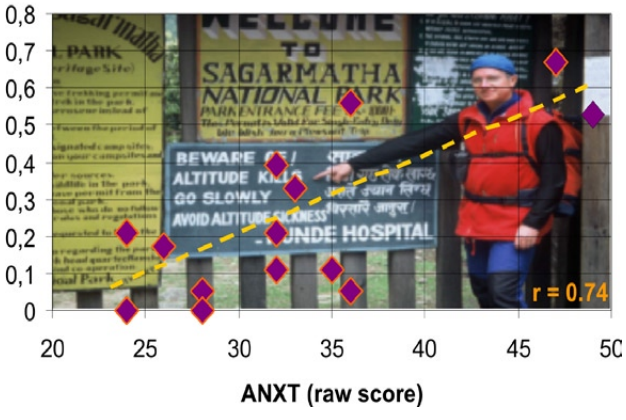

Figure 2. *AMS-Score Imja-Team (PSP-2002). ANXT-factor (raw score) versus LL-AMS-sum score of the Imja Tse-team. Y-axis shows mean values of the AMS-evening protocol.*

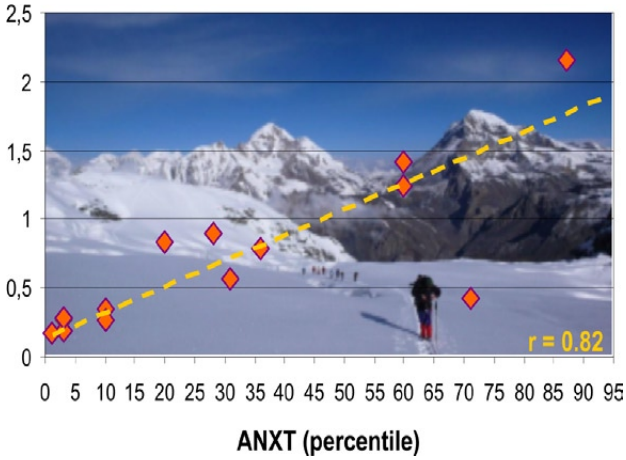

Fig. 3. AMS-Score (CHULU WEST-2008). *ANXT-factor (percentile) and LL-AMS-sum score of the Chulu West-team. Y-axis shows mean values of the AMS-evening-morning protocol (picture by D. Neumann).*

Table 1. *Correlations between ANXT-factor and LL-AMS-score, heart rate and SpO_2 in Imja Tse Team (n=14) and Chulu West Team (n=14)*

Tab. 1a							
Imja Tse	ANXT-raw score	ANXT-percentile	ANXT-T-value	Chulu West	ANXT-raw score	ANXT-percentile	ANXT-T-value
MED	32.00	50.50	49.50	MED	28.00	24.00	43.00
MW	33.63	47.88	49.19	MW	29.36	30.21	42.43
SD	7.26	26.56	8.29	SD	7.73	28.69	10.32
MIN	24.00	6.00	35.00	MIN	21.00	1.00	28.00
MAX	49.00	92.00	64.00	MAX	46.00	87.00	62.00
Tab. 1b							
Imja Tse	AMS-evening	AMS-morning	AMS-Δ	Chulu West	AMS-evening	AMS-morning	AMS-both
MED	0.19	0.29	0.24	MED	0.38	0.64	0.50
MW	0.24	0.35	0.30	MW	0.54	0.96	0.70
SD	0.22	0.27	0.20	SD	0.58	0.76	0.57
MIN	0.00	0.00	0.00	MIN	0.00	0.27	0.17
MAX	0.67	0.88	0.78	MAX	2.29	2.64	2.15
r =	0.74*	0.13*	0.49$^{\Delta}$	r =	0.72*	0.69*	0.79*
							0.82* (PR)
Tab. 1c							
Imja Tse	HR evening	SpO2 evening		Chulu West	HR both	SpO2 both	
MED	78.55	88.20		MED	81.00	91.00	
MW	81.25	88.41		MW	82.04	89.89	
SD	8.73	2.39		SD	10.10	7.25	
MIN	64.30	83.70		MIN	62.00	73.00	
MAX	95.70	91.80		MAX	110.00	99.00	
r =	-0.14*	-0.04*		r =	-0.27*	-0.40*	
r =	0.20$^{\square}$	-0.21$^{\square}$		r =	0.24$^{\blacklozenge}$	-0.52$^{\blacklozenge}$	

* correlation between variable and ANXT-raw score resp. -percentile (PR)
$^{\Delta}$ values based on arithmetic AMS-sum score: (morning+evening protocol)/2
$^{\square}$ correlation between variable and evening-AMS-sum score (n=14; 19 measured values)
$^{\blacklozenge}$ correlation between variable and both AMS-sum scores (n=13; 3 measured values)

raw score of 24 (6th percentile) and a maximum of 49 (92th percentile). In the Imja-team between ANXT-raw score and LL-AMS-sum score r = 0.74 ($P < .01$; evening protocol; Fig. 2); in the morning r = 0.13 (Tab. 1b).

The Chulu West-group comprises 6 women and 8 men from Austria and Germany. Their median ANXT-raw score is 28 (24th percentile), the arithmetic ANXT-mean (± 1 SD) is 29.36 ± 7.73 (30th percentile; Tab. 1a) with a minimum raw score of 21 (1st percentile) and a maximum of 46 (87th percentile). The correlation between ANXT-raw score and LL-AMS-sum score is 0.72 (evening protocol; Tab. 1b) respectively 0.79 (mean morning-evening protocol). ANXT-percentile and LL-AMS-sum score have the highest correlation coefficient (average morning-evening protocol; r = 0.82, t = 5 which exceeds 4.318, the critical value of t for $P < .001$ with v = 14 - 2 = 12 degrees of freedom; Fig. 3) indicating a direct link between both variables.

In both teams HR and likewise SpO_2 correlate insignificantly with ANXT-values and LL-AMS-scores (for details see Tab. 1c). For the Chulu-team r = -0.27 (HR and ANXT) respectively r = -0.40 (SpO_2 and ANXT). Similar values are found for HR and AMS (r = 0.24), and SpO_2 and AMS (r = -0.52).

Discussion

Acute Mountain Sickness is generally seen by doctors and climbers as a kind of "sickness" caused by oxygen deprivation that directly leads to an organic

hypoxic state. However, the existing sickness concept that is dominated by physiological interpretations of hypoxia leading to brain swelling is not well able to explain the huge inter-individual variety in reactions to (mild) oxygen deprivation and thus the so called individual vulnerability to AMS! It should be mentioned, that brain swelling was never proven for early stages of AMS. Here, a different and somewhat alternative, if you like novel model and explanation for AMS is presented: AMS is thereby not so much caused by hypoxic brain swelling as is argued by for instance Roach and Hackett in their review [19], it is more over the result of a hypoxemia induced stress reaction on the basis of the pre-existing individual emotional tuning.

As early as the nineteen sixties and -seventies some authors presumed that AMS is the natural response of the body to certain processes in the CNS. Slater und Roth thought that stress was responsible for the symptoms of AMS [20]: "The principal symptoms of AMS are generally indistinguishable from the normal responses to mental or physical stress, which, in turn, are variably dependent on many (psychological) factors such as motivation, expectation and personality". Heath and Williams concluded that these psychological aspects without a doubt participate in the development of AMS, however they could not imagine that on the basis of personality factors (alone) one could make predictions regarding the behaviour and the reactions of sea-level individuals at high altitude [21]. Olive and Waterhouse examined 17 participants of a British Himalaya-expedition using 3 standardized personality-questionnaires [22]. They were not able to ascertain an apparent interrelation between the test scores and a list of selected clinical symptoms like headache, nausea or Cheyne-Stokes breathing. However individual personality factors had an influence on the self-assessment regarding the magnitude of AMS. Individuals with a diagnosis of AMS furthermore had higher values on the neuroticism-factor (N-scale after H. J. Eysenck). This factor represents "stability up to instability", the N-dimension of personality is rather closely coupled to the congenital amount of instability of the autonomic nervous system. The N-scale thereby represents likewise a "negatively tinged emotional tenor", showing a significant correlation with the individual anxiety score [23].

A more or less straight association between negative affects and AMS has been proven again and again from the start of the 1990s. Shukitt-Hale et al. report on the effects of mood changes and Crowley et al. of negative moods on the development of acute mountain sickness [24, 25]. Several French researchers go even further. They found out that "individuals susceptible to AMS are significantly more anxious than those who were not susceptible" and concluded that anxiety – as measured by Spielberger's State-Trait-Anxiety Inventory [17] – is an adequate psychological predictor of AMS [14, 15, 26]. In a chamber study Hildebrandt et al. could prove that under HYPOX, subjects having a more pronounced trait-anxiety, report the symptoms of AMS intensified [27]. After just 4 hours in the hypobaric chamber, the correlation between the LL-AMS-sum score and the individual ANXT-factor was 0.61 ($P < .01$).

Under field conditions significant correlations varying from 0.74 (between ANXT and LL-AMS-evening protocol on Imja Tse) till 0.82 (ANXT-percentile and LL-AMS-sum score on Chulu West Peak) were calculated in this study, while the interrelation between the ANXT-factor and SpO_2 was clearly less pronounced or in case of HR did hardly exist. These data advocate a highly significant impact of the individual's personality dimension trait-anxiety on the development and intensity of AMS. Brain swelling on it self, as is often argued [19], is not the "AMS-producing factor" as the study by Dubowitz et al. [28] clearly shows. They found comparable amounts of swelling in all subjects: those susceptible to AMS and those not! So again, hypoxic brain swelling can't explain the individual differences, anxiety trait does.

According to the neuropsychosomatic stress model in figure 1 the stressor HYPOX activates a number of coping mechanisms: even before possible symptoms of AMS are recognized in fittingly vulnerable persons subacute hypoxemia will lead to an increase in blood flow velocity of the right ACM [12], as well as to a significant rise in power spectral delta activity in the right cerebral temporal region (electrode T4; [11]). HYPOX poses a potential threat for a „Selfish Brain". Along the fight-or-flight stress axis HYPOX causes an activity boost (arousal) in the sympathetic, adrenergic and serotonergic medulla system. In this manner adequate energy and oxygen supply to the brain is being ensured, usually at the cost of other organ systems [5].

Within the stress-coping model arousal (vigilance) is defined as a class of coping mechanisms, that are deployed in threatening situations with the purpose of reducing unstableness (loss of control) or of limiting its further increase. Dependent on the pre-existing "arousal-setting" of the brain which corresponds to the individual amount of trait-anxiety, the stressor HYPOX triggers a fittingly intense coping measure aimed at warranting adequate oxygen supply. The symptoms of acute mountain sickness are the consequence, or in other words: AMS is at first instance a form of stress, is the result of a "Selfish Brain" that by means of "CO_2PING" measures tries to prevent the potentially detrimental effects of hypoxia (cerebral edema; organic psychosyndrome; neurologic/neuropsychologic deficits). At the same time the intensity of this "CO_2PING" is linked to a fair amount to the neurotic personality dimension: The data strongly suggest that the more

pronounced the individual ANXT-factor, the stronger the RCH-activation of the fight-or-flight stress axis and (thus) the AMS-symptomatic.

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