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Correlation of cerebrovascular disorder and anxiety: The Kecskemet study

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Abstract. In order to test the hypothesis that anxiety is a risk factor for cardiovascular disease, specifically stroke, we simultaneously measured anxiety and cerebral vascular alternation, using a computer-based system, "Cerberus." Sixty nine psychiatric patients (including an alcoholic subgroup) were selected as subjects for measurements conducted in Kecskemet, Hungary. The five-item short form of anxiety test (STAI) was administered twice during the same session. Between each test, brain pulse waves were recorded by rheoencephalogram (REG). A REG peak time above 180 milliseconds was considered a cerebrovascular alteration (modified after Jenkner). Data were sorted into two groups: low anxiety (N=10) and high anxiety (N=10). Significant differences were found between cardiovascular risk factors (p< 0.001), REG peak time (p<0.043), and heart rate (p< 0.045). Six subjects showed cerebrovascular alteration in the high anxiety group, and two in the low anxiety group. For the two anxiety groups, there were no significant differences in body mass index, cardiovascular sympathetic-parasympathetic balance, age and symptoms of transient ischemic attack. The correlation of REG and age was significantly different only for the alcoholic subgroup (Szalay et al, 2007). These data support the hypothesis that a correlation exists between cerebrovascular disorder and anxiety in the studied population.

1. Introduction

Since arteriosclerosis is not a mono-causal disease, the detection of the presence of a single risk factor cannot accurately predict the development of the disease. A complex, multi-factorial approach can produce a more exact diagnosis. We utilized a device, "Cerberus," a computer-based system developed for primary stroke prevention [1]. The Cerberus system incorporates all known risk factors for arteriosclerosis/stroke and includes a short form of an anxiety test (STAI) [2]. The STAI Form Y is the definitive instrument for measuring anxiety in adults. It clearly differentiates between the temporary condition of "state anxiety" and the more general and long-standing condition "trait anxiety." Vegetative balance was also determined [1,3,4]. Vegetative balance is the measurable status of both the sympathetic nervous system (ergotroph) and the parasympathetic nervous system (trophotroph). To estimate a patient's cerebral circulation, the Cerberus system uses a non-invasive, continuous technology known as REG [5,6]. In this study we tested the hypothesis that anxiety is a

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risk factor for cardiovascular disease, specifically stroke, by measuring psychiatric patients for anxiety and cerebral vascular alteration.

2. Methods

The Cerberus system involves the measurement of pulsatile electrical impedance in the brain (REG), detailed elsewhere [1]. A/D sampling rate was 275 Hz. A one minute REG recording was used for signal averaging, triggered by the ECG R wave. The left and right side REG data were averaged for each person. REG peak (anacrotic or rising) time was measured and evaluated as a symptom of cerebrovascular alteration [7]. A short form of STAI was used containing five "state anxiety" items. Cerberus software calculated the ergotroph/trophotroph ratio (modified vegetative balance by Kerdo and Sipos), in which: 1- (diastolic blood pressure /heart rate) x 100 [1]. Values above 100 indicate ergotrop; values below 100 are considered a trophotroph vegetative state. Body mass index (BMI: kg/m²) was also calculated by Cerberus. Student t-test in Excel (Microsoft, Redmond, WA) was used for statistical evaluation; P< 0.05 was considered significant.

2.1. Subjects

Three patient groups were combined (drug abuse, alcohol abuse, depression; N: 42 male, 27 female). The alcohol abuse patients, middle-aged inpatients identified as having alcohol problems for more than ten years (N=44, mean age: 41), were participating either in detoxification (Dg [8]: F10.30, F10.40) or abstinence treatment programs (Dg: F10.10, F10.20, F10.80). Age varied from 27 to 54 years for males and from 27 to 56 years for females. The drug abuse patient group included two subjects with drug dependency (F12.20, F13.20) and four subjects with a diagnosis of abusive disorders (F11.10, F18.10, F19.10) (N=6; mean age: 31). The depressive subjects (N=6, mean age: 51) (F32.00, F32.20, F333.20) were non-psychotic. In the drug patient group, the youngest and oldest subjects were males (19 and 49 years of age). The male/female ratio for alcoholic patients was 32:12; for drug patients, 4:2; and for depressive patients, 1:5; mirroring the general tendency of gender distribution for these mental disorders.

2.2. REG

REG electrodes were placed on the fronto-temporal area, within the electrode cap; localization: Fp1-F7; Fp2-F8, according to 10-20 system of EEG electrode placement. Each REG electrode was a 15-mm diameter tin disc attached to traditional EEG electrodes (ECI Electro-Cap Electrode System, Electro-Cap International, Eaton, OH). Electrode gel was used to improve skin-electrode contact. The REG sclerosis standard used was based on Jenker's work [7]. A REG peak time longer than 180 msec was considered sclerotic in our evaluation, see figure 1.

3. Results

Significant differences were found between cardiovascular risk factors (p< 0.001) and REG peak time (p< 0.043). Although the second anxiety level (following a one minute polygraphic recording) was typically lower than the first, this difference was not significant. In the high anxiety group, six subjects (60 %) showed cerebral vascular alteration; in the low anxiety group, two subjects (20 %) showed cerebrovascular alteration. For the two anxiety groups, there were no significant differences in body mass index, cardiovascular sympathetic-parasympathetic balance, age and symptoms of transient ischemic attack The correlation of REG and age was significantly different only for the alcoholic subgroup, detailed elsewhere [9].

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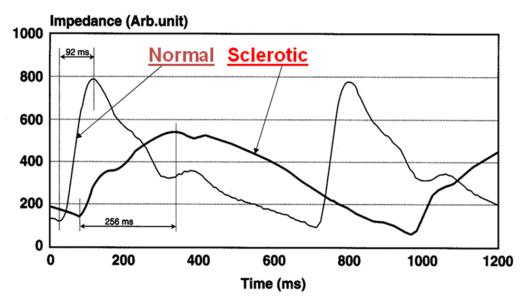


Figure 1. Typical REG curve of a healthy (normal) and a sclerotic person. Note the difference in peak time of REG curves for normal patient (92 milliseconds) and sclerotic patient (256 milliseconds). This difference has pathological meaning, independent of heart rate [10].

Table 1. Comparison of low and high anxiety groups. We found significant differences between low anxiety groups (upper part of table) and high anxiety groups (lower part of table) in cardiovascular risk factors (red column) and REG peak time (yellow column). Both groups consisted of 10 subjects.

| Low anxiety | (N=10) | | | | | | | | | | | |
|-------------|----------|--------|-----------|-----------|-----------|---------|---------|-------|-----------|-------|-------|----------|
| | Male | Female | Kerdo ind | TIA sympt | Risk Fact | Anxiety | Anxiety | Age | REG pk tm | HR | ВМІ | |
| | n | n | | n | n | 1 | 2 | У | ms | 1/min | kg/m2 | |
| | 8 | 2 | 98,90 | 0,90 | 0,50 | 9,00 | 8,30 | 39,40 | 98,53 | 81,00 | 23,30 | mean |
| | | | 9,67 | 1,37 | 0,53 | 1,94 | 2,16 | 7,49 | 59,38 | 14,53 | 2,63 | sd |
| | | | | | | | | | 2 | | | >180 REC |
| | | | | | | | | | | | | |
| ligh anxiet | v (N=10) | | | | | | | | | | | |
| | 6 | 4 | 100,00 | 3,31 | 1,92 | 19,69 | 18,92 | 43,23 | 134,81 | 88,38 | 22,77 | mean |
| | | | 10,37 | 3,47 | 0,86 | 0,48 | 1,89 | 11,76 | 66,25 | 12,02 | 4,26 | sd |
| | | | 0,66 | 0,08 | 0,001 | 0,00000 | 0,00000 | 0,18 | 0,043 | 0,45 | 0,88 | t-test P |
| | | | | | | | | | 6 | | | >180 RE |
| otal: 69 | 42 | 27 | | | | | | | | | | |

4. Discussion

In the literature, there are many supportive findings of a correlation between psychological and somatic factors[11]. The wider spectrum of somatic and psychological factors correlates with our recent findings. In another study we conducted involving 546 subjects, we found significant correlations among somatic and psychologic variables. Cerebrovascular alteration (REG-Doppler-and symptoms of transient ischemic attack) was in a bridge position between measured somatic variables and psychological variables [12]. These results are consistent with our recent findings that anxiety should be considered as a cardiovascular and stroke risk factor. The potential anatomical substrate of this relationship is the vascular endothelium, which plays an essential role in regulation of cerebral blood flow--a fine tuned mechanism. Although the measurement of cerebrovascular reactivity by REG has been known for decades, it was recently documented that REG reflects cerebral blood flow autoregulation [13]. This fact explains findings where cerebral blood flow changes were detected by REG during transcendental meditation [14] and eye opening and closing [15].

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5. Conclusion

On the basis of previous and recent findings, we suggest that anxiety should be considered as a stroke risk factor. The potential anatomical substrate of this relationship is the vascular endothelium, which plays an essential role in regulation of cerebral blood flow. These results confirm a correlation between anxiety and sclerotic alteration of coronary and carotid arteries. Spielberger and others observed relationships among anxiety, anger and cardiovascular risk many years ago. Here, we present new evidence supporting this observation.

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