Neurosensory Deficits After Myocardial Infarction

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Abstract: Cardiovascular diseases are extremely widespread and often cause vestibular system dysfunctions. They are related mainly to organic lesions of the brain. To investigate neurootological functional changes, we compared two samples from among our patients, of whom those in group A (42 persons: 92.86% male, 7.14% female) had experienced myocardial infarction within 1 year before our neurootometric investigation and those in group B had undergone infarction 1 year or more before examination (104 patients: 81.73% male, 18.27% female). Considering only the six most important vertigo symptoms experienced by patients, we found 1.48 symptoms per patient in group A and 2.02 symptoms per patient in group B. As regards acoustic symptoms, 45.24% of patients in group A experienced tinnitus and 52.38% reported hearing loss. In patients in group B, 48.08% were affected with tinnitus and 58.65% with hearing loss. Abnormalities in the neurootometric measurements were revealed as follows: in group A, butterfly calorigrams, 80.95%; stepping-test craniocorpography (CCG), 64.29%; and bone conduction audiometry on the right side, 40.48%, and on the left side, 52.38%; in group B, butterfly calorigrams, 78.85%; stepping-test CCG, 61.54%; bone conduction audiometry on the right side, 28.85%, and on the left side, 41.35%.

Key Words: craniocorpography; electronystagmography; hearing loss; myocardial infarction; tinnitus; vertigo; vestibular dysfunction

Human brain consumes some 20–25% of the blood being pumped into the vessels by the heart. Thus, brain function also is affected when the heart does not work properly. As a result, patients suffering from a heart attack or myocardial infarction not only experience pain but later have other neurosensory symptoms from dysfunctions (e.g., in equilibrium and hearing). People who survive a heart attack must undergo extensive rehabilitation, as a risk of a recurrence always remains. Therefore, we have chosen as an issue for this study a comparison between two post–myocardial infarction phases: (1) within 1 year, with patients still exhibiting existing complaints, and (2) more than 1 year after myocardial infarction. All 146 patients in this study underwent postinfarction rehabilitation therapy.

During the last 30 years, major data banks of patients with neurootological complaints both in Würzburg at the Neurootological University Laboratories and in Bad Kissingen at the Neurootological Laboratories of the Forschungsinstitut der 4-G-Forschung have been built up. As a consequence, we were able to select two samples of patients in whom we could compare the patients' subjective complaints with the objective and quantitative findings in a thorough neurootological network analysis.

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PATIENTS AND METHODS

We chose our two sample groups of neurootological patients according to their medical histories: One group consisted of patients who reported experiencing a myocardial infarction within a year prior to our neurootometric investigation (N = 42), and a second group of patients who had experienced a myocardial infarction a year or more prior to our investigation (N = 104).

From all 146 patients, we recorded a thorough neurootological history using the Neurootological Data Evaluation–Claussen (NODEC) system. This systematic questionnaire contains sections for describing vertigo, nausea, hearing disorders, visual disorders, taste and smell disorders, neurological disorders, and other pathological mechanisms (e.g., trauma, cardiovascular diseases, kidney diseases, and diabetes mellitus). All patients were also questioned about any drug treatment and about the use of negative substances (e.g., smoking or drinking habits). A selected number of parameters appear in this article, as the entire body of data is too lengthy to include.

The practice of neurootology includes a large consideration of equilibriometry. From the field of equilibriometry, we applied several tests to all our patients, including polygraphic electronystagmography (ENG) with a five-channel derivation of the patients' ENGs when being investigated for spontaneous nystagmus or experimentally provoked nystagmus. Simultaneously, in all patients a three-channel extremity derivation of electrocardiography (ECG) was recorded, giving us information about the ongoing vegetative reactions during the test (see earlier).

Vestibuloocular tests being recorded by means of the five-channel ENG were monaural caloric tests, with 20 ml of 30° or 44°C water syringed over 30 seconds. The test results were evaluated by means of the Claussen butterfly chart.

The binaural vestibuloocular test was performed on the electronically programmable rotatory chair with a perrotatory supraminimal stimulus and a postrotatory supramaximal stimulus. The polygraphic ENGs were evaluated by means of the L-chart of the rotatory intensity damping test.

If the supraminimal monaural caloric warm response is considered the response to a weaker stimulus and the perrotatory nystagmus response on the rotatory intensity damping test is considered to be the stronger stimulus, a vestibular stimulus response intensity comparison (VESRIC) can be established according to the balancing mechanisms within the equilibrium pathways in the central nervous system. By means of VESRIC, responses of a parallel behavior from recruitment phenomena and from decruitment phenomena can be differentiated.

Regular retinoocular testing was performed by means of an ocular tracking test and by an optokinetic nystagmus test. The results of these tests, however, had to be left out of this article owing to limits of publication space.

For investigating the vestibulospinal pathways, craniocorpography (CCG) was used to record the head and trunk movements in space during standing and stepping. The CCG patterns appear as a radar image of the trails of a human floating through space.

We analyzed hearing function by means of several audiometric tests. The hearing pathways first were investigated by pure-tone threshold audiometry and by determining the discomfort threshold for estimating the acoustic dynamics. Social hearing function was measured by speech audiometry, which also was used especially for the statistics in this study. Tinnitus was masked according to its frequency and intensity.

Further hearing pathway investigations included transiently evoked otoacoustic emissions (TEOAE), acoustic brainstem evoked potentials, and acoustic late or cortical evoked potentials (ALEP). All the data assembled in this study were compiled into a spreadsheet (Microsoft's Excel). On this data bank, our data processing, statistic evaluations, and chart plotting have been performed.

RESULTS

This article focuses on the comparison of neurootological complaints among patients within 1 year after a myocardial infarction (group A) and those within a phase longer than 1 year after a myocardial infarction (group B). The samples contained 42 persons for group A and 104 persons for group B (total, 146 patients). As regards our statistics, we have complete individual descriptions within the patient files. Also, a neurootological data bank, NODEC, has been established that comprises a list and a statistical analysis of the entire sample of 146 patients (Table 1). We found among our patients who had experienced a myocardial infarction a variety of symptoms, which occurred with differing frequency among the groups (Table 2). The results of the vestibuloocular pathway test are presented in Table 3.

For a synoptical overview of the most frequent combination of caloric responses, all the butterfly patterns were transcribed by trinary code and thereafter statistically evaluated for both samples (groups A and B; Fig. 1). From comparisons of the ipsidirectional vestibuloocular responses due to monaural (caloric) and binaural (perrotatory) stimuli, we derived the VESRIC; the most frequent trinary-coded VESRIC patterns are listed in Figure 2. The figure reveals that the vestibuloocular nystagmus function is normal in patients in group A (u_A = 16.67%) and in group B (u_B = 17.31%; see Fig. 2).

Table 1. Biographical Data of 146 Patients with Neurootological Complaints After Myocardial Infarction

	M	F	Age (yr)		Height (cm)		Weight (kg)		Systolic BP (mm Hg)		Diastolic BP (mm Hg)	
	n (%)	n (%)	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
MI <1 yr (n = 42)	39 (92.85)	3 (7.15)	53.54	8.77	172.65	7.66	77.41	9.76	143.75	14.93	85.00	5.77
MI >1 yr (n = 104)	85 (81.73)	19 (18.27)	58.67	9.85	170.15	7.50	74.44	10.79	134.50	17.39	81.66	7.90
Total (n = 146)	124 (84.93)	22 (15.07)										

BP = blood pressure; SD = standard deviation.

Table 2. Symptom Frequencies Noted in Post–Myocardial Infarction Patients in Both Groups

Symptom	Group A Patients (%)	Group B Patients (%)
Vestibular symptoms		
Rocking	40.48	53.85
Lifting	7.14	3.85
Rotating	23.81	36.54
Falling	7.14	31.73
Blackout	38.10	36.54
Instability	30.95	39.42
Vegetative or nausea symptoms		
Sweating	40.48	53.85
Malaise	23.81	29.81
Retching	2.36	3.85
Vomitus	2.38	8.65
Collapse	19.05	12.50
Duration of single vertigo attacks		
Seconds	38.10	50.00
Minutes	35.71	32.69
Hours	2.38	12.50
Days	0	2.88
Visual disturbances		
Loss of acuity	69.05	75.00
Double vision	4.76	3.85
Oscillopsia I	2.38	8.65
Oscillopsia, jerking	0	0.96
Amaurosis	0	3.85
Oscillopsia II	16.67	25.96
Hearing symptoms		
Tinnitus	45.42	48.08
Hearing loss	52.38	58.65
Deafness	7.14	7.69
Ear surgery	7.14	4.81
Humming sound	0	1.92
Whistling sound	4.76	4.81
Noise trauma	9.52	5.77
Cardiovascular background disorders		
Hypertension	21.43	20.19
Hypotension	23.81	26.92
Arteriosclerosis	4.76	0.96
Myocardial infarction	64.29	74.04
≤1 year previous to investigation	100	3.85
>1 year previous to investigation	9.52	100

Table 3. Statistical Evaluation of the Vestibuloocular Caloric Test with Electronystagmography (ENG) and Electrocardiography (ECG) Recording of 146 Patients with Neurootological Complaints After Myocardial Infarction

	Myocardial Infarction <1 yr (n = 42)		Myocardial Infarction ≥1 yr (n = 104)	
	Mean	SD	Mean	SD
Caloric test (frequencies in Hz)				
Sp. nystagmus right	0.43	0.29	0.45	0.41
Sp. nystagmus left	0.45	0.34	0.38	0.31
44°C right	1.2	0.66	1.22	0.65
30°C right	1.34	0.66	1.38	0.75
44°C left	1.29	0.6	1.23	0.62
30°C left	1.42	0.72	1.46	0.79
ECG/min				
Sp. ECG	74.83	10.33	73.75	15.14
44°C right	74.31	9.58	71.71	14.95
44°C left	74.08	9.62	71.67	14.64
30°C right	74.71	9.41	72.01	14.82
30°C left	74.2	9.7	71.73	14.71
RIDT				
ECG rate, sitting position	73.23	10.7	68.13	13.28
Perrotatory ECG left	73.13	10.68	68.97	12.57
Perrotatory ECG right	71.08	11.31	68.52	12.74
Postrotatory ECG right	73.00	12.6	68.0	12.31
Postrotatory ECG left	71.71	11.52	68.97	1.57

RIDT = rotatory intesity-damping test; Sp. = spontaneous.

The vestibulospinal tests that were recorded and evaluated by means of CCG are displayed in Figure 3. Typical findings are underlined by graphic representations.

Some cases represent the individual patterns of the CCGs. Among the CCGs, the stepping-test CCG was clinically the most important for detecting pathology within the vestibulospinal pathways. Of the possible 81 patterns in trinary coding, the 12 most frequent are ranked in Figure 3 for groups A and B. Whereas group A shows only 35.71% normal responses, this increases to 38.46% in group B. Only 5 of 12 patterns match for both the groups, of which pattern "s0200," expressing a pure cerebello-ponto-medullary brainstem disinhibition, is the most frequent single pattern—14.29% in group A and 7.69% in group B.

The audiometric results of the social hearing efficacy test by means of speech audiometry are listed in Table 4. Speech audiometry was evaluated according

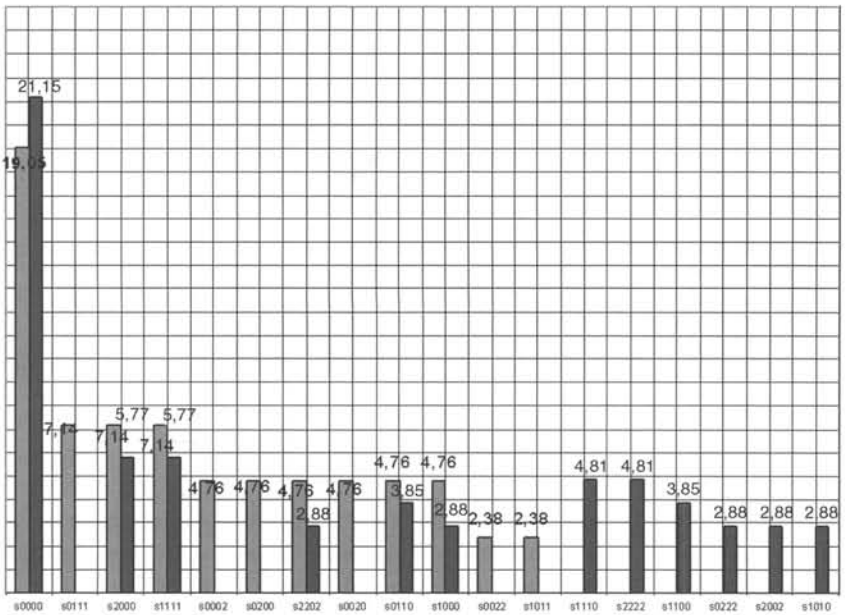

Figure 1. Caloric butterfly test results (%) sorted according to the occurrences of the 12 most frequent patterns, which can be graphed or expressed by trinary coding (0 = normal; 1 = inhibited; 2 = disinhibited) with four digits (position I = right warm; position II = right cold; position III = left warm; position IV = left cold). (Light bars = group A, or R1, ≤1 year post–myocardial infarction [N = 42]; dark bars = group B, or R2, >1 year post–myocardial infarction [N = 104].)

to the three important parameters—recognition of numbers and words and loss of discrimination—for the right ear in 146 patients (groups A and group B) suffering from a myocardial infarction. The parameters were statistically evaluated with respect to mean and standard deviation.

DISCUSSION

It was the aim of this study to elucidate that patients after a myocardial infarction suffer from more than chest pain and reduction of their daily activities owing to restricted blood flow, the experience of shortness of breath, especially during exercise, and tightening pain

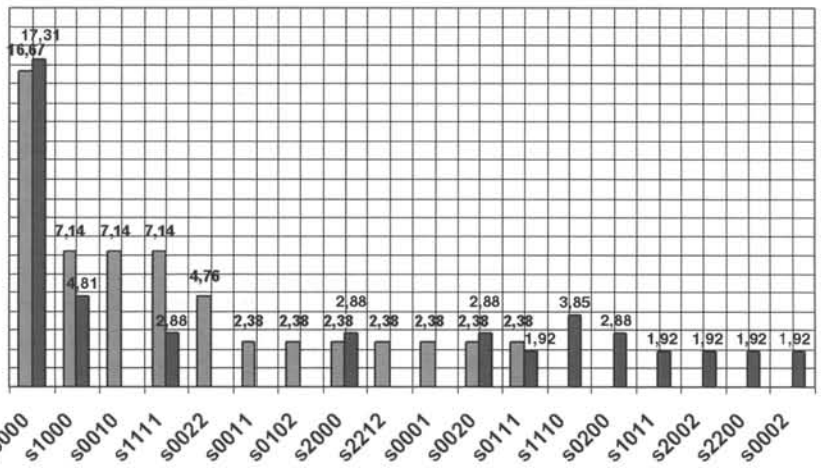

Figure 2. Dynamic vestibular stimulus response intensity comparison (VESRIC) sorted according to the occurrences of the 12 most frequent patterns. Combining the perrotatory part of the rotatory intensity-damping test with the caloric warm response, we can establish the so-called vestibular stimulus response intensity comparison (VESRIC). When combining the two—right-beating nystagmus and the left-beating nystagmus—we arrive at the charts of VESRIC, which can be graphed or expressed in trinary coding (0 = normal; 1 = inhibited; 2 = disinhibited) as four digits (position I = right warm; position II = perrotatory right; position III = left warm; position IV = perrotatory left). The test results can be categorized into three groups, in each of which are three subdivisions. The first group contains the parallel behavior, the second group contains the so-called recruiting phenomena, and within the third group are the so-called decruitment phenomena of VESRIC. (Light bars = group A, or R1, ≤1 year post–myocardial infarction [N = 42]; dark bars = group B, or R2, >1 year post–myocardial infarction [N = 104].)

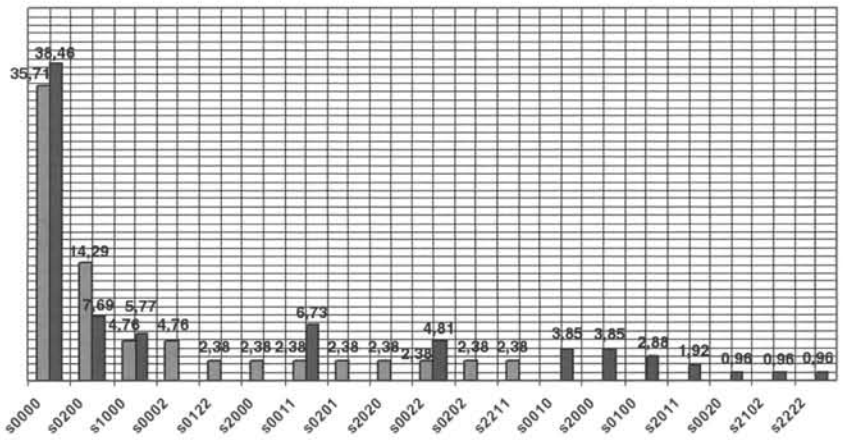

Figure 3. Stepping-test craniocorpography results sorted according to the occurrences of the 12 most frequent patterns, which can be graphed or expressed by trinary coding (0 = normal; 1 = inhibited; 2 = disinhibited) with four digits (position I = displacement; position II = lateral sway; position III = angular deviation; position IV = body spin). (Light bars = group A, or R1; dark bars = group B, or R2.)

in the chest, known as *angina pectoris*. The study also exhibits a wider spectrum of complaints in such patients' neurosensory system, as proved by special history data (NODEC) and by the patients' equilibriometric and audiometric findings (as described).

With respect to pathological anatomy, infarction is an area of coagulation necrosis in heart tissue due to local ischemia resulting from obstruction of circulation to the area, most commonly by a thrombus or embolus. Myocardial infarction is a gross necrosis of the myocardium, as a result of interruption of the blood supply to the area, as in coronary thrombosis. Myocardial infarction may occur when coronary vessels are narrowed or occluded, such that the blood supply to the infarcted heart muscle is seriously impaired.

Congestive heart failure, arrhythmias, aortic or mitral stenosis, carotid hypersensitivity, and other vascular lesions are frequently associated with vertigo or syncopal attacks (or both). Many patients report typical syncopal attacks as giddiness. A syncopal attack must be carefully distinguished from a vertiginous attack by a patient's medical history. Functionally, this is achieved by ECG, which we have performed for more than three decades in our neurootological laboratories in Würzburg and Bad Kissingen.

Syncope is an episodic loss of consciousness caused by vascular insufficiency in the brain. Its management is distinctly different from that of vertigo. Hypoperfusion of the brain, characterized by unconsciousness or by light-headedness, diminished vision, or blackouts, is indicative of a syncope. Conversely, a sensation of motion without external stimuli or a sensation of spinning, whirling, swaying, staggering or, simply, unsteadiness or instability is indicative of vertigo, which now can be verified by ENG, ECG, and CCG. Sometimes, however, typical features of a syncope may be associated with vertiginous symptoms along with central nervous system disorders (e.g., diplopia, dysarthria, focal sensorimotor disturbance, and occipital headache). This unusual combination of features suggests the rare possibility of syncopal attack (ischemia) of the posterior cerebral circulation.

Cardiovascular diseases are extremely widespread and often cause vestibular system dysfunctions. They can be related also to resulting organic lesions of the brain. Vertigo has been shown to occur mainly in patients with disturbances in circulation in the vertebrobasilar blood supply and may be evaluated objectively by various types of vestibular deficiencies (i.e., peripheral or central or both). The cause of vestibular disorders appears to be a vasomotor disturbance mainly in the vertebrobasilar system. A certain correlation is seen between different varieties of nystagmus and a rate of vestibular asymmetry in persons suffering from vertigo due to cardiovascular pathology.

As far as the cardiovascular system is concerned, some researchers question whether individuals with

Table 4. Statistical Evaluation of Speech Audiometry in 146 Patients with Neurootological Complaints After Myocardial Infarction

	Myocardial Infarction <1 yr		Myocardial Infarction ≥1 yr	
	Mean	**SD**	**Mean**	**SD**
Speech audiometry, right				
Numbers	20.12	32.52	23.87	26.25
Words	69.72	16.16	75.22	20.19
Loss of discrimination	14.88	33.93	11.27	25.46
Speech audiometry, left				
Numbers	12.04	12.51	25.03	25.02
Words	70.16	15.66	76.94	20.33
Loss of discrimination	2.04	7.72	12.62	25.74

SD = standard deviation.

cardiovascular diseases are prone to develop sensorineural deafness. Hearing impairment parallels the incidence of coronary heart disease; however, not all cardiac patients develop hearing loss. This could be owing to biological variability from one individual to the other; that is, this phenomenon is likely to have a more complex genetic background. Attacks of vertigo, dizziness, giddiness, blackout, instability, rocking more than rotating, tinnitus, and transient hearing loss can also be considered as early symptoms of coronary insufficiency.

In general, the number of pathological vestibular reactions is much higher in patients who experience myocardial infarction than in patients in other stroke groups. The brain needs to be fed, and when the cardiac power is diminished (e.g., by myocardial infarction), we find various systems with vestibuloocular and vestibulospinal pathological functions, even in speech audiometry, increased discrimination loss, and pooling down of the threshold discrimination loss.

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