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LETTER TO THE EDITOR

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Somatosensory evoked potential in panic disorder

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Panic disorder has been an issue of permanent interest to many specialists, especially psychiatrists, and recently to neurologists and neurophysiologists as well. Knott and Lapierre (1986), Kukumberg et al. (1994) described significant changes of brain stem auditory evoked potentials (BAEP) during i.v. sodium lactate provocation and also in the interparoxysmal period and after hyperventilation. Beckerman et al. (1986) noted similar findings of BAEP in central apnoea syndrome, which, according to Klein's (1993) theory of failure of the suffocation alarm system, is considered to be a reversed entity to panic disorder.

The locus coeruleus seems to be the site of panic attack initiation, but a major role in panic attack pathogenesis is played by the respiratory centres of the brain stem, which are hypersensitive (panic disorder) or hyposensitive (central apnoea) to carbon dioxide.

To confirm the involvement of the brain stem in the pathophysiology of panic disorder, we investigated somatosensory evoked potentials (SEPs). SEP of the median nerve was investigated bilaterally using the apparatus Madaus Electronic 15 GmbH, Co KG, Freiburg, Germany in 15 untreated right-handed patients with panic disorder, with or without agoraphobia according to DSM-III-R, (12 women, 3 men, average age 37.7 ± 4.3 years) and 20 healthy right-handed persons (12 women, 8 men, average age 34 ± 8.5 years). In the patients the results revealed a significant bilateral shortness of mean latency of P17 (Student t-test, $P < 0.01$) and mean interpeak latencies of N13 – P17 and P14 – P17 ($P < 0.01$) (Table 1).

Generators of the wave P17 are located in the medulla oblongata, presumably up to the pons Varolii, and the dysfunction in panic disorder was also related to the brain stem. These results are qualitatively reversed to BAEP findings which showed prolongation of the III-V interpeak interval (Knott and Lapierre 1986) as an expression of deranged function. Shortness of parameters of SEP was described at increased neuronal excitability due to hypocalcaemia (Kanda et al. 1988). Durlach (1988) reported Mg intracellular depletion in normocalcaemic tetany, mitral valve prolapse and in panic attack. This aspect should be considered when studying biochemical relationships and implications of these clinically overlapping affections.

Changes of two modalities (auditory and somatosensory) of brain stem evoked potentials might express not only functional but also structural disorders in predilection regions of the brain stem, resembling findings of selective metabolic and haemodynamic changes of temporo-limbic areas in panic disorder (Nutt and Lawson 1992).

Due to the steadiness of the parameters of brain stem evoked potentials their changes are signals of disorder and

Table 1 The mean latencies and the interpeak latencies of the median somatosensory evoked potentials (SEPs) in the patients with panic disorder (PD) and the control group

	Patients with PD ($n = 15$)		Control group ($n = 20$)	
	sin	dx	sin	dx
SEPs waves				
N9	9,6 + 0,7	9,7 + 0,7	9,7 + 0,6	9,9 + 0,6
N13	13,1 + 0,8	13,3 + 0,9	13,4 + 0,5	13,6 + 0,5
P17	16,2 + 0,8*	16,3 + 0,8*	17,0 + 0,7	17,2 + 0,8
P14	14,3 + 1,0	14,3 + 0,9	14,6 + 0,4	14,7 + 0,5
N20	18,5 + 1,0	18,8 + 0,8	18,7 + 0,5	19,0 + 0,6
P25	22,2 + 1,7	22,3 + 1,7	22,9 + 1,0	23,1 + 1,6
Interpeak latencies				
N9–N13	3,4 + 0,4	3,5 + 0,4	3,3 + 0,4	3,4 + 0,4
N13–N20	5,5 + 0,3	5,4 + 0,4	5,3 + 0,5	5,6 + 0,4
N13–P14	1,2 + 0,4	1,1 + 0,5	1,3 + 0,4	1,1 + 0,4
N13–P17	3,1 + 0,5*	3,1 + 0,6*	3,6 + 0,5	3,6 + 0,4
P14–P17	1,9 + 0,6*	1,8 + 0,46*	2,3 + 0,8	2,5 + 0,5
P14–N20	4,2 + 0,6	4,2 + 0,4	4,1 + 0,4	4,5 + 0,4
P17–N20	2,3 + 0,6	2,4 + 0,4	2,4 + 0,6	2,0 + 0,5

*The significant shortening of the mean latencies and the interpeak latencies of the SEPs ($P < 0.01$ Stutent's test)

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may prospectively be used to advantage as a screening diagnostic method.

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