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Psychophysiological Correlates of Suicidal Behavior in Depression

A Preliminary Study

Key Words

P300 Contingent negative variation Depression Suicide

Abstract

P300 and contingent negative variation (CNV) were recorded in depressive inpatients with and without history of suicide attempt. The results show a significant reduction of both P300 and CNV in patients who had attempted suicide as compared with patients who had not. Moreover, a significant correlation was found between the suicidal risk scale and CNV amplitude. Psychophysiological and biochemical implications are discussed.

Introduction

P300 and contingent negative variation (CNV) have been widely applied in psychiatry, especially in depression, schizophrenia and dementia [1, 2]. Concerning depressive disorders, abnormalities of CNV amplitude and duration as well as reduced P300 amplitude have been reported [3, 4]. Moreover, the decrease in CNV amplitude has been related to the severity of depressive symptomatology [5] and to the central catecholaminergic deficit [6]. Unfortunately, the suicidal dimension of the depressive illness has never been investigated in these studies. Recently, our group reported that depressive patients with a history of suicide attempt exhibited a significantly lower growth hormone (GH) response to apomorphine than patients who had never attempted suicide [7]. Among others [8, 9], this study supported a dopaminergic deficit in relation to suicidal behavior. Since P300 and CNV are known to be modulated by central neurotransmitters, particularly catecholamines [2, 6, 10], we hypothesized a difference in these psychophysiological measures between depressive patients with and without a history of suicide attempt.

Method

The study was conducted in 7 DSM-III-R depressive inpatients admitted consecutively because of suicide attempts to the Psychiatric Unit of the University Hospital André-Vésale, Montigny-le-Tilleul, Belgium. The sample comprised 4 men and 3 women, with a mean age of 39.4 years (SD = 9.8 years). The patients were matched for age and gender with 7 depressive inpatients without a history of suicide attempts. All patients had at least a score of 18 on the 17-item Hamilton Depression Scale. Past history of suicide attempt was assessed by the suicidal risk (SR) scale [11]. All patients were free of medical illness, evidence of which was provided by clinical examination, history, EKG, EEG, chest X-ray, and routine laboratory tests.

Psychophysiological recording was conducted at the end of a drug-free period of at least 1 week. The subjects were sitting in a sound-attenuated room. For the P300 paradigm, a series of 100 auditory stimuli was presented with a variable interstimuli interval, which comprised between 2 and 4 s. According to the classic oddball

Table 1. Comparison of clinical and psychophysiological data between depressive patients with and without history of suicide attempt

	Suicide attempt (n = 7)	No suicide attempt (n = 7)	F.	p
SR scale	17.1±3.7	8.0±4.2	18.3	0.001
Hamilton depression				
scale	24.8 ± 3.2	23.0 ± 2.5	2.01	0.17
CNV amplitude	-14.7 ± 4.5	-20.9 ± 4.6	4.7	0.04
CNV RT	420 ± 425	294 ± 240	1.7	0.21
P300 amplitude	6.6 ± 5.3	16.6 ± 8.3	5.0	0.04
P300 latency	347.2 ± 258	335.1 ± 368	1.4	0.24
P300 RT	607.5 ± 256	435.8 ± 368	1.7	0.21

RT = Reaction time.

paradigm, 85% of the stimuli (frequent) were tones of 2,000 Hz, 75 dB, and 50-ms duration. The other 15% (rare) were tones of 1,000 Hz, 75 dB and 50-ms duration. The subjects were asked to press a button for the rare stimuli. P300 amplitude was measured as the difference in voltage between the baseline and the higher point between 250 and 650 ms after the stimulus.

The CNV paradigm consisted of a single reaction time preceded by a warning stimulus 1 s earlier. The intertrial interval was randomized from 5 to 10 s. The CNV was obtained from 32 artefact-free trials as inspected by electrooculogram tracing. CNV amplitude was measured as the voltage difference between the baseline and the average of the 200 ms preceding the imperative stimulus.

The EEG was recorded using silver-silver chloride disc electrodes attached with collodion at Cz, using left earlobe for reference and right forehead for ground. All sites were cleaned with acetone and abraded to maintain resistance below $3 \, \mathrm{k}\Omega$. EOG was recorded from above the left eye. Amplifier gains were set at 10,000, with a band pass of 0.1–30 Hz, and digitized at 250 samples/s for 3,000-ms epochs (of which the first 1,000 ms were prestimulus activities).

Results

As expected, SR scores were higher in patients with a positive history of suicide attempt (table 1). However, no difference was apparent between the two groups in the Hamilton Depression Scores.

Mean CNV and P300 amplitudes were significantly lower in patients with suicide attempts as compared to patients with a negative history (table 1). In contrast, P300 latency and P300 or CNV time reaction did not significantly differ between the two groups (table 1).

A significant correlation existed between CNV amplitude and SR scale (r = 0.63, p < 0.05). For the Hamilton

Depression Scores, the relationship did not reach the level of significance (r = 0.43, p = 0.07).

No significant relationship existed between P300 amplitude and SR scale or the Hamilton Depression Scores (r = -0.30, NS, and r = -0.24, NS).

Discussion

The present study shows a significant reduction of both P300 and CNV amplitude in depressive patients who had attempted suicide as compared with those who had not. These results could be interpreted both from a psychophysiological and a psychopharmacological viewpoint.

First, slow potentials have been integrated in a model providing a synthesis from different theories [12]. This model combines two concepts: cerebral potentiality and cerebral performance. Cerebral potentiality refers to the psychophysiological state necessary for any cerebral processes. Cerebral performance covers the input, decoding, storage, and output of information processing. Moreover, cerebral potentiality is necessary for cerebral performance.

According to this integrative model, CNV amplitude represents the cerebral potentiality, and its resolution cerebral performance. Therefore, our results suggest that patients with a history of suicide attempt present a lower cerebral potentiality than patients without previous suicidal attempts. Moreover, CNV amplitude is correlated with the SR scale. A possible explanation for these findings is that suicidal behavior appears in patients with less resources to cope with their environment, as indicated by less cerebral potentiality. This reduced cerebral potentiality could reflect the concepts of helplessness and hopelessness described in depression [13, 14].

Similarly, P300 amplitude has been associated with cerebral performance (information processing). The cognitive disturbances in a view of the future, the world, and the self as described in depression [15], involve a perturbation of cerebral performance. Our results show that disturbances of information processing were more severe in patients with suicidal attempts, but were not influenced by the severity of depression.

Second, from a psychopharmacological point of view, a large body of findings shows that neurochemical systems modulate P300 and CNV amplitude [6, 10]. For example, dopaminergic agonists like methylphenidate enhance P300 amplitude in hyperkinetic children and normal subjects [16], whereas dopaminergic antagonists like flupentixol reduce P300 amplitude in normal subjects [17]. In

the same manner, CNV amplitude is modulated by dopaminergic agents [18], and correlated with GH response to appropriate, a dopaminergic agonist [3].

Recently, many studies have shown that dopaminergic function is impaired in depressive patients with previous suicide attempts [8, 9, 19]. Our group reported a lower GH response to apomorphine in patients with suicide attempts, as compared with patients without a suicidal history [7]. Therefore, the reduction of P300 and CNV amplitudes could reflect the dopaminergic dysfunction linked to suicidal behavior in depression.

Taken together, the results of this preliminary study show that patients who attempt suicide exhibit a reduction of both P300 and CNV amplitudes as compared with patients with a negative history. These results support the dopaminergic hypothesis of suicide behavior in depression. Moreover, they could represent an objective assess-

ment of helplessness and hopelessness which play a important role in depression, and more particularly for the suicidal behavior [14].

Several limitations in the present study should however be acknowledged, particularly those related to the limited number of patients as well as to the absence of a distinction between violent and nonviolent suicide attempts. Therefore, further studies with larger samples will be necessary to confirm and extend these preliminary results.

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References

- 1 Roth WT, Duncan CC; Pfefferbaum A, Timsit-Berthier M: Applications of cognitive ERPs in psychiatric patients. Electroencephalogr Clin Neurophysiol Suppl 1986;38:419-438.
- 2 Charles G, Hansenne M: Le potentiel lent P300: intérêts cliniques dans trois pathologies et neurobiologie: une revue. Encéphale 1991; 18:225-236.
- 3 Timsit-Berthier M, Mantanus H, Ansseau M, Devoitille J-M, Dal Mas A, Legros J-J: Contingent negative variation in major depressive patients; in Johnson R, Rohrbaugh JW, Parasuraman R (eds): Current Trends in Event-Related Potentials Research. Amsterdam, Elsevier, 1987, pp 761-771.
- 4 El Massioui F, Lesèvre N: Psychomotor retardation in depressed patients: An event-related study. Neurophysiol Clin 1988;70:46-55.
- 5 Papart P, Ansseau M, Devoitille J-M, Mantanus H, Timsit-Berthier M: Contingent negative variation and the severity of depression; in Stefanis CN, Soldatos CR, Rebavilas P (eds): Psychiatry: A World Perspective. Amsterdam, Exerpta Medica, 1991, vol 2, pp 402-408.
- 6 Timsit-Berthier M: Contingent negative variation (CNV) in psychopharmacology, Electroencephalogr Clin Neurophysiol Suppl 1991;42: 142-152.

- 7 Pitchot W, Hansenne M, Gonzalez Moreno A, Ansseau M: Suicidal behavior and growth hormone response to apomorphine test. Biol Psychiatry 1992;31:1213-1219.
- 8 Roy A, Agren A, Pickar D, et al: Reduced CSF concentration of homovanillic acid and homovanillic acid to 5-hydroxyindolacetic acid ratio in depressed patients: Relationship to suicidal behavior and dexamethasone nonsuppression. Am J Psychiatry 1986;143:1539–1545.
- 9 Roy A, Karoum F, Pollack S: Marked reduction in indexes of dopamine metabolism among patients with depression who attempt suicide. Arch Gen Psychiatry 1992;49:447–450
- 10 Callaway E: Discussion: Towards a pharmacology of information processing. Electroencephalogr Clin Neurophysiol Suppl 1991;42:173–176.
- Plutchick R, van Praag HM, Conte HR, Pilard
 Correlates of suicide and violence risk. 1:
 The suicide risk measure. Compr Psychiatry
 1989;30:296–302.
- 12 Rockstroh B, Elbert T, Lutzenberger W, Birbaumer N: Slow Brain Potentials and Behavior. München, Urban & Schwarzenberg, 1982.

- 13 Seligman MEP: Depression and learned helplessness; in Friedman R, Katz M (eds): The Psychology of Depression: Contemporary Theory and Research. New York, Wiley, 1974, pp 83-113.
- 14 Beck AT: Hopelessness as a predictor of eventual suicide; in Mann JJ, Stanley M (eds): Psychobiology of Suicidal Behavior. Ann NY Acad Sci 1986;487:90-96.
- 15 Beck AT: Cognitive Therapy and Emotional Disorders. New York, International Universities Press, 1976.
- 16 Klorman R, Brumaghim JT: Stimulant drugs and ERPs. Electroencephalogr Clin Neurophysiol Suppl 1991;42:135–141.
- 17 Rösler F, Manzey D, Sojka B: Delineation of pharmacopsychological effects by means of endogenous event-related brain potentials: An exemplification with flupentixol. Neuropsychobiology 1985;13:81-92.
- 18 Tecce JJ: Dopamine and CNV: Studies of drugs, disease and nutrition. Electroencephalogr Clin Neurophysiol Suppl 1991;42:153– 164
- 19 Agren H: Symptoms patterns in unipolar and bipolar depression correlating with monoamine metabolites in the cerebrospinal fluid: II. Suicide. Psychiatry Res 1983;3:225-236.