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**Use of cognitive event-related potentials in the management of psychiatric disorders: Towards an individual follow-up and multi-component clinical approach**

Campanella S. Cognitive ERPs in psychiatry

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**Abstract**

Relapse prevention remains a major challenge in psychiatry, thus indicating that the established treatment methods combining psychotherapy with neuropharmacological interventions are not entirely effective. In recent years, several intervention strategies have been devised that are aimed at improving psychiatric treatment by providing a complementary set of add-on tools that can be used by clinicians to improve current patient assessment. Among these, cognitive event-related potentials (ERPs) have been indexed as valuable biomarkers of the pathophysiological mechanisms of various mental illnesses. However, despite decades of research, their clinical utility is still controversial and a matter of debate. In this opinion review, I present the main arguments supporting the use of cognitive ERPs in the management of psychiatric disorders, stressing why it is currently still not the case despite the vast number of ERP studies to date. I also propose a clinically-oriented suitable way in which this technique could — in my opinion — be effectively incorporated into individual patient care by promotion of the use of individual ERP test-retest sessions and the use of a multi-component approach.

**Key Words:** Event-related potentials; Psychiatry; Cognitive disorders; Follow-up; Multi-component approach; Personalized medicine

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**Core Tip:** Despite decades of intense research and many promising results, cognitive event-related potentials (ERPs) have yet to be implemented in daily psychiatric care units as an add-on tool to psychotherapy and medication. I present here the main arguments supporting the notion that ERPs represent a highly suitable tool for performing individual “neuro-cognitive” assessments in psychiatric patients. Such ERP data could help clinicians to specify individual cognitive interventions that will target each patient’s specific needs, thus promoting an “individualized” or “personalized” medicine.

**INTRODUCTION**

The 1990s have been referred to as the “Decade of the Brain”, with developments such as brain imaging tools allowing patterns of distributed neural activity associated with both normal and pathological behaviors to be identified[1]. On this basis, major mental illnesses, such as schizophrenia, autism, major depression, anxiety disorders, and addictions, were redefined as brain diseases[2], with a deep impact of the environment at both the social and physical levels[1]. Nowadays, the separation of neurology and psychiatry appears arbitrary, and in the framework of modern neuroscience, psychiatrists and neurologists could be called “clinical neuroscientists” who apply neuroscientific discoveries to the care of patients with brain disorders[3]. Clearly, the management of a mentally ill patient necessarily requires consideration (mainly through psychotherapy) of a single individual embedded in a specific social-cultural context in order to encompass the social and psychogenic aspects of individual clinical symptoms[4]. However, mapping the live brain activity of a patient, by—for instance—the use of positron emission tomography (PET), magnetic resonance imaging (MRI), or electroencephalography (EEG), has demonstrated that all normal or dysfunctional mental processes are ultimately biological[2]. It is, therefore, also important to consider these cognitive, emotional, and social processes, subtended by specific choreographed patterns of brain activity that — when dysfunctional — can mediate the onset and persistence of specific clinical symptoms[5]. For instance, an alteration of mental state attribution, the ability to infer mental states of others in order to guide social interactions, is classically observed in schizophrenic patients[6]. This deficit, mainly subtended by neural alterations in the prefrontal cortex and the superior temporal sulcus[7], is associated with a poor outcome, social functioning, and social competence in schizophrenia[8]. As antipsychotic medication has been shown to have a limited impact on recovery and social cognition[9], the challenge is to develop new therapeutic strategies to specifically improve social cognition in schizophrenia. Promising results, revealing improvement of social functioning and reduction of psychotic symptoms, have been achieved through social cognition training programs and psychosocial interventions[10] as well as by brain stimulation through transcranial direct-current stimulation (tDCS)[11] or transcranial magnetic stimulation[12]. Similarly, in major depressive disorder, deficits in cognitive inhibition, subtended by a hyperactivated amygdala insufficiently controlled by a hypoactivated prefrontal region[13] appear to be a main causal factor for ruminations[14]. Sessions of training to inhibit negative thoughts[15] as well as a combined tDCS-mindfulness program resulted in a decrease in ruminations and lower depressive scores[16], while medication only has been reported to provide modest improvements[17]. In alcohol dependence, an increased salience of alcohol-related cues grasping drinkers’ attention (hyperactivated mesolimbic activity[18]) combined with a lack of inhibitory resources (anterior cingulate and frontal hypoactivity[19]) defines the main neurocognitive mechanisms triggering relapse[20]. In light of the modest effect of anti-craving medications[21], cognitive bias modification training alone[22] or combined with tDCS[23] has shown promising trend on treatment outcomes by reducing craving and by improving early abstinence. Overall, convergent empirical data illustrating alterations in brain networks that underlie cognitive impairments have provided foundational information about transdiagnostic circuits and promising targets for intervention[24]. Indeed, numerous studies have provided consistent evidence that mental illness involves significant cognitive impairments that represent valid therapeutic targets, as enhancing cognitive functioning leads to a reduction of clinical symptoms and a better quality of life. One of the main challenges at present consists of developing new ways to use neurocognitive mechanisms as an add-on tool in the clinical and conventional management of psychiatric patients.

Due to their high anatomical resolution, PET and functional MRI (fMRI) clearly constitute the most suitable tools to assess the distributed brain networks involved in diverse cognitive functions[25]. However, their coarse temporal resolution (1-2 s) does not allow definition of the temporal activation sequence, thus preventing isolation of the series of individual sensory, cognitive, affective, and motor processes that occur between a stimulus and a response[26,27]. Electrophysiological tools, by recording spontaneous electrical brain activity from multiple electrodes placed over the scalp[28], are more suitable for this purpose due to their optimal temporal resolution on the order of milliseconds[29]. EEG is an inexpensive and non-invasive tool defining a valuable clinical first-line method to exclude a diagnosis of epilepsy, drug intoxication, or sleep disorders in psychiatric patients[30]. A derivative of the EEG technique refers to event-related potentials (ERPs), *i.e.*, averaged EEG responses that are time-locked to the cognitive processing of stimuli. The past several decades have witnessed a vast number of task-dependent ERP components being described and studied among healthy people. While studies on healthy participants have helped to define the various cognitive steps needed throughout the entire information processing stream to achieve a cognitive function, such data also have great relevance in pathology. Indeed, by accessing the various cognitive steps needed to achieve a cognitive function, cognitive ERPs may then also allow definition of where a dysfunctional behavior originates at the cognitive level. This has great clinical relevance, as a similar altered behavior may be subtended by various cognitive disorders[31]. Therefore, by indexing the specific neurocognitive functions that are dysfunctional in a patient, ERPs pinpoint cognitive functions that should be rehabilitated in each patient through specific and individualized cognitive remediation procedures[26]. However, despite a solid theoretical basis[29] and decades of research showing alterations of these components in various psychiatric diseases[32], their relevance in clinical settings is still a matter of debate[33]. The scope of this paper is not to provide an exhaustive review of the literature regarding ERPs in various psychiatric diseases. Rather, my aim is to present relevant arguments supporting the notion that it is important to incorporate the use of cognitive ERPs in the management of psychiatric disorders, by also stressing why it is still not the case nowadays despite thousands of ERP studies to date. I then propose a clinically-oriented suitable way in which this technique could—in my opinion—be effectively incorporated into individual patient care.

**Decades of ERP studies in psychiatry: why so many hopes and promising results for such a minor clinical impact to date?**

Depending on the cognitive task one is confronted with and the cognitive processes one is focusing on, several ERP components have been described in recent decades in the literature. The P50, the contingent negative variation (CNV), the mismatch negativity (MMN), the P300 with its P3a and P3b subcomponents, the No-go N2 and No-go P3, the error-related negativity (ERN), and the N400 are some of the most studied ERPs. When elicited through a specific task in healthy subjects, such ERPs are the neural correlates that assess the efficiency of diverse cognitive processes, such as sensory gating[34], arousal and motor preparation[35], auditory discrimination[36], novelty processing *vs* decision making[37], cognitive and motor inhibition[38], insight[39], and semantic congruency[40]. As ERP amplitudes reflect differences in the intensity of responses whereas measurements of latency inform regarding the processing time duration[40,41], several anomalies in amplitude and/or latency of these components have been reported in various psychiatric disorders[32,42]. In such a view, ERPs could characterize biological markers of pathophysiological mechanisms[43]. Such biological markers can be state (only present during the acutely ill state but stabilized after remission) or trait (always present, during and after the disease) markers[44,45]. On the one hand, by reflecting pathophysiological processes that are active during the disease, state markers could provide clinicians important input to assist with choosing the most appropriate treatment. For instance, a decreased amplitude of the P3b component is considered to be a state marker of depression[46]: The P3b amplitude has been shown to be increased after four weeks of antidepressant treatment[47] as well as following recovery from electroconvulsive therapy[48]. Similarly, chronic schizophrenic patients exhibit reduced MMN amplitudes compared to healthy controls[49], and antipsychotics such as aripiprazole[50] or drugs acting on the NMDA (N-Methyl-D-aspartate) receptor[51] appear to induce its recovery, while the CNV is reduced in amplitude in children[52] and adults[53] with ADHD but has been shown to exhibit an amplitude recovery with even just a single dose of stimulant medication[54]. Such instances clearly highlight how ERP state markers could be particularly useful in monitoring the efficiency of a treatment. On the other hand, trait markers can also be particularly useful, mainly as indicators of vulnerability[45]. Indeed, the amplitude reduction of the P3b as well as the absence of P50 suppression in schizophrenia[55], an enhanced ERN in child and adult anxiety disorders[56], and an altered P3 component in cocaine users[57] are examples of trait markers, indexing during and after the disease, of impaired cognitive functions that play a pivotal role in the onset and persistence of these mental diseases. But importantly, such alterations can also define a risk marker for healthy people with, for instance, a positive family history to further develop it[58-60]. Therefore, such markers, if present, can improve early detection of illness, and, as such, facilitate more effective and targeted interventions[44].

Overall, state and trait ERP markers can serve to aid diagnosis (as prognostic elements[61]), assist in choosing the most appropriate treatment for psychiatric disorders[62], and help with detecting illness at an early phase[63]. However, although such empirical data, even in meta-analyses[64-68], may appear convincing and useful, the reality is quite different as, up to now, the utility of cognitive ERPs in daily clinical care settings remains (very) modest[30]. Several explanations may account for this situation. First, at a technical level, the worldwide current ERP screening procedure favours the huge number of ERP studies, despite the heterogeneity of the data. For instance, an amplitude reduction and/or a delayed latency of the P3b and the MMN is usually considered to be a state marker of depression[69] and early psychosis[70], respectively. However, contradictory data have also been reported, suggesting no P3b and MMN differences between depressive[48] and psychotic[71] patients, respectively, with controls. Such heterogeneity has understandably led to a degree of scepticism among clinicians as it raises questions regarding reliability. The main factors accounting for these discrepancies are the clinical subtypes of patients included in these studies, with comorbidities inducing particular responses[72], higher artifact contamination in clinical patients than in typical subjects[27], a potential influence of medication[27], and differences in ERP recording protocols leading to data misinterpretations[73]. In this respect, a very interesting initiative, called “ERP CORE” (Compendium of Open Resources and Experiments)[74], has recently been launched in order to provide standardized ERP paradigms for seven widely used ERP components (N170, MMN, N2pc, N400, P3, lateralized readiness potential, and ERN). By providing researchers with a useful tool and guidelines for selected tasks to record specific ERP components, this will notably promote the possibility of comparing ERP data sets from different laboratories. At a clinical level, such guidelines already exist[75]; however, their use in studies around the world is still by no means ensured, and this could notably lead to a degree of misinterpretation of the data[73]. It is, therefore, urgent to again underscore that using such guidelines accepted by the field would clearly help with clinical implementation[76] by providing access to normative data gathered on large samples in order to follow the progression of patients as a function of the treatment, but also to control for potential confounding factors, such as gender, age, medication, and comorbid symptoms. Secondly, at a conceptual level, the potential role of ERP components in the management of mental disorders has also suffered from the predominance in psychiatry of the official nosological systems, such as the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Diseases (ICD)[77]. In such a categorical view, patients either do or do not meet the criteria to be diagnosed with a mental illness, thus suggesting that the presence of a specific cluster of symptoms will necessarily correspond to a specific mental disease (*e.g.*, borderline personality disorder). On this basis, the psychiatrist will select what they consider the best option of treatment among the range of those appropriate for that specific diagnosis (*e.g.*, selective serotonin reuptake inhibitors; SSRIs). If this treatment proves to be ineffective, they will then have to choose another option (*e.g.*, combination of SSRIs and quetiapine[78]) or reconsider the former diagnosis[79]. A main and crucial point that makes this categorical approach still dominant in psychiatry is that it greatly facilitates clinical communication among mental health practitioners, as all textbooks and practice guidelines have been developed based on these categories[80]. In such a categorical view, mental illnesses are discontinuous entities with distinct symptoms, etiologies, and biomarkers[81]. However, inter-individual variability in the severity of symptoms among patients receiving a similar diagnosis, common symptoms across different disorders, and an extremely usual comorbidity factor are well-documented facts in clinical experience[77]. In other words, common liabilities across these “categories”[81,82] are more suggestive of a dimensional underlying cross-cutting transdiagnostic structure for mental disorders[77]. This still ongoing debate regarding the transdiagnostic *vs* the categorical frameworks to guide psychopathology assessments[83] is not the scope of this paper. However, it clearly appears that, due to the dominance of the nosological approach in psychiatry, the imprecision of categorical psychiatric diagnoses[84] has been a limiting factor in understanding the pathophysiological mechanisms of human behavioral abnormalities. Indeed, although decades of research have provided evidence of the relevance of various ERP components as biological markers of mental illnesses[32,43], their clinical sensitivity has been hampered by the fact that their parameters (*e.g.*, amplitude and latency) are diagnostically unspecific[33]. In other words, ERP deficits are a common feature of several psychiatric afflictions, but they will not assist clinicians in deciding whether a given patient is depressed, paranoid, or an alcoholic (high sensitivity but low specificity). Moreover, cognition is not considered as a primary treatment target, being still envisaged as a particular category of symptoms (among others), and not as a core phenomenon triggering the onset and/or the persistence of the disease. Many psychiatrists still then focused on finding the best suited drugs combination in order to contain symptoms et minimize side effects. Therefore, the approach using ERPs to classify patients according to DSM categories was entirely inappropriate, and, in the next section, I will specify how I think ERPs may be genuinely useful in clinical settings, mainly as predictive biomarkers, *i.e.*, as measured indices that may be used to predict clinical responses to treatment[85]. Finally, at a clinical level, a major practical issue is that the majority of ERP-based studies compare their results with matched controls using grand-averaged data. While such “group results” have ample merit at a fundamental “research” level, there is now a need for more “individualized”, “personalized” medicine[86], *i.e.*, individual data that helps with devising interventions that are specifically targeted based on each patient’s needs[26]. In the next section, I will try to provide some insights on how ERPs may be used effectively in clinical settings as an individual monitoring tool to reveal (or not) expected changes in brain function in response to a treatment[87].

**Using ERPs in psychiatry: what perspectives?**

In an influential paper published in 1991 arguing for the use of PET in clinical care[88], Wagner[88] stated that: “…Today's medical practice is yesterday's research. The bridge linking the two is technology assessment, which makes possible the acceptance or rejection of new technologies in the practice of medicine. Experience is the key determinant of effectiveness. If the information provided is not useful to the physician caring for the patient, the procedure will eventually fall by the wayside...”. The notion that ERPs are useful for managing psychiatric diseases, such as, for instance, depression[89], alcohol disorders[90], or schizophrenia[91], is not novel at all. However, despite decades of research, ERPs have yet to be implemented in the clinical management of mental illnesses[27]. It clearly appears at present that the clinical value of ERP components as a diagnostic index is low[33], merely reflecting a common measure of brain dysfunction[92]. With this in mind, it is, therefore, now urgent to precisely define what constitutes be the best use of ERPs in the management of psychiatric disorders. In order words, there is a need to find out which properties of ERPs as a tool could be the best-suited ones to help with managing a currently still unsolved clinical question.

A prominent issue in the treatment of mental illnesses relates to the relapse rate[93], which is approximately 50% at 1 year and 70% at 5 years for manic episodes in bipolar disorders[94]; approximately 35% at 18 mo and 74% at 5 years following a first episode of schizophrenia[95]; and approximately 50% at 3 mo and 85% at 1 year for recently detoxified alcoholic patients[96]. Despite the beneficial impact of psychotherapy and neuropharmacological interventions, as well as the positive effect of more recent intervention strategies such as multisystemic[97], cognitive behavioral[98], or mindfulness[99] therapies, the relapse rate is still extraordinarily high. Clearly, the idea is not at all to discredit the existing treatment methods, but providing a complementary set of add-on tools to be used by clinicians to improve current patient assessments is still a major challenge. Starting with the fact that mental diseases are also brain disorders, a neurocognitive approach has emerged[100]. This can be summarized as follows: (1) Mental illness involves significant cognitive impairment[101]; (2) These cognitive alterations, subtended by perturbed brain networks, may trigger the onset and/or persistence of clinical symptoms, thereby defining valid therapeutic targets[24]; and (3) Retraining these cognitive functions (through the use of cognitive retraining programs and/or neuromodulation tools) has been reported to reduce clinical symptoms and to enhance patient quality of life[11,16,22,23]. Overall, once admitted to a psychiatric care unit, there is still a huge difficulty with assessing “partial recovery”, which allows patients to leave the hospital to return home, and achievement of long-term “complete clinical recovery”, defined as the reduction of psychiatric symptoms and functional disabilities[102].

In my opinion, it is in this regard that ERPs may have an important clinical role in the management of psychiatric patients, as a monitoring and a predictive biomarker tool. Indeed, psychiatric evaluations are made almost entirely on the basis of clinical symptoms, and the longitudinal course is determined by a clinician speaking with the patient and informants (as well as sometimes by the use of clinical scales), but diagnostic frameworks do not usually incorporate biomarkers[103]. However, once the notion that mental illnesses are subtended by impaired neural functioning is acknowledged, a main assumption is to consider that this dysfunctional brain should undergo significant and enduring neural changes in order to be reflected in medium-to-long-term real-world behavioral modifications[104]. Indeed, test-retest ERP studies on healthy participants have, for instance, shown that subjects exhibiting improvements in inhibitory performance in a Go/No-go task had a similar residual gain in inhibition one week after post-training, albeit only when this effect was neurophysiologically indexed by faster Nogo-N2 latencies[105]. Accordingly, in neuropsychiatric conditions, several studies found that specific cognitive gains induced specific brain changes that were positively associated with decreased symptoms and better quality of life, even 6 mo later[106-108]. Taking these neural modifications into account would, therefore, be of the greatest clinical relevance, as their absence would suggest a high vulnerability to relapse. In this view, state ERP markers provide the possibility (1) to monitor the change, triggered spontaneously or by a specific treatment, in neurocognitive mechanisms that are involved in the onset and maintenance of clinical symptoms in an individual patient; and (2) to verify whether these neural changes induced by the treatment are predictive of the clinical trajectory. By fostering a longitudinal follow-up and intra-individual ERP approach, recent works have tried to verify the clinical utility of such designs, in which ERP measurements are included just as one would include measurements of symptoms. In a study by our laboratory of alcohol-dependent patients undergoing a four-week detoxification program[109], we showed that monitoring the changes in dual-processes that are well-known to trigger addictions (No-go P3 for inhibition[110] and P3 for cue reactivity[111]), at the start and at the end of the program, can provide clues about the mechanisms involved in abstinence or relapse. Indeed, specific changes in cognitive ERP markers during detoxification (a preserved oddball P3 and an enhanced No-go P3) indexed complete abstinence (over a 3-mo period) in alcoholic patients. The main clinical relevance of such test-retest ERP data is the possibility of pinpointing the change in specific neurocognitive functions (cue reactivity, inhibition) during the detoxification program that can predict further abstinence. Such a procedure necessarily implied: (1) Specification of the various cognitive mechanisms that should be considered as the primary targets subtending the clinical symptoms of interest; and (2) Selection of the appropriate cognitive tasks that will generate specific and reliable ERPs related to these specific processes at an individual level.

A crucial step, therefore, concerns the identification of the various cognitive processes of interest. As such, transdiagnostic (as opposed to disorder-specific) factors appear uniquely suited to bridge psychiatric phenomena and biological substrates of behavior[77]. Transdiagnostic impairment of cognitive control[82], self-referential processes[112], working memory[113], decision making[114], and attention[115] largely contribute to the real-world socio-occupational impairment common across disorders. Decades of research have validated reliable ERP markers for such processes (No-go P3, ERN, P300, MMN, and P50, respectively). Indeed, an altered No-go P3 response inhibition, as well as a deficit in P300 (in tasks tagging updating in working memory or decision making), are neurophysiological disorders present in psychotic[116,117], bipolar[118,119], unipolar[48,120] depressive disorders as well as in anxiety[121,122] and substance use disorders[123,124]. Studies also commonly reported an altered ERN in anxiety disorders and in obsessive-compulsive disorders[125]; an altered MMN (indexing preattentive auditory memory processing) in major depression, schizophrenia, psychosis, and substance use disorders[36]; and a deficit in P50 sensory gating in stable schizophrenic patients and euthymic bipolar patients[58]. In such a dimensional transdiagnostic view, the lack of specificity reported for these various ERP components tagging specific cognitive functions will vanish and allow monitoring of the change in these processes during a treatment, independently of a categorical disorder. Such a proposition relies on two main “technical” recommendations for future ERP screening methods: (1) The use of individual ERP test-retest sessions; and (2) The use of a multi-component approach[126].

ERP serial recordings may be used in clinical contexts for the assessment of changes in cortical function during follow-up programs[33]. Indeed, although slight differences in the wave shape, size, and timing of ERPs between individuals are usually observed, these tend to be highly stable within an individual across recordings, as high internal consistency and high test-retest reliability have been reported[127-130]. A high degree of reliability is a key factor that makes state ERP markers highly suitable for examining changes in brain activity resulting from treatment intervention or disease progression in a single patient[12]. This could provide ERPs a novel and particularly useful role in the management of psychiatric disorders. Indeed, assessing that neural changes have been induced spontaneously and/or by a treatment is a necessary outcome to envisage medium to long-term positive behavioral changes[104]. For instance, recovering a normalized MMN amplitude or P50 suppression appears to be a good indicator of abstinence in alcohol and cocaine users, respectively[131]. In such a view, it is also particularly important to always merge ERP data obtained through “active” tasks with behavioral results. To illustrate this crucial point, we can focus on contradictory data showing that excessive alcohol users compared to controls exhibited decreased No-go P3 amplitudes[38,123], probably reflecting poor neural resources indexing a poor performance; while other studies have reported increased No-go P3 amplitudes[132], probably reflecting compensatory neuro-functional mechanisms that allow drinkers to achieve a similar performance level as controls. In other words, an increased amplitude should not necessarily be interpreted as a recovered activity, and a decreased amplitude should not necessarily be interpreted as a disrupted function. Therefore, merging test-retest ERP data with behavioral performances would allow identification of patients who need to recover more neural resources to achieve better performance (*e.g.*, an increase of inhibitory No-go P3 resources to maintain abstinence in alcoholic patients[109]), while patients exhibiting compensatory or exacerbated mechanisms may need to reduce the activity allocated to the task (*e.g.*, decreased ERN in anxiety disorders to assess better emotional regulation[56]). These types of individual assessments would help clinicians specify individual interventions that will target each patient’s needs, thus providing “individualized” or “personalized” medicine[26]. For instance, a recently detoxified alcoholic patient who does not exhibit improvements on the No-go P3 component could be redirected to post-cure specific inhibitory boosting programs[109], while a patient with a severe anxiety disorder who does not exhibit a reduced ERN due to a drug treatment could be directed to therapies that address emotional regulation such as mindfulness[133]. Naturally, it is important at this point to outline that, on the basis of DSM or ICD psychiatric categories, some ERP trait markers have also been described. For instance, obsessive-compulsive disorder has been characterized by an increased ERN both before and after therapy[68] as well as for the altered P3 component in cocaine users[57]: Such trait markers, *i.e.*, neurophysiological mechanisms persisting during and after the disease, suggest that such ERP markers could be related to the risk for the disorder but not the expression of its phenotype. Even in a dimensional perspective, such trait markers, if present, can improve early detection of illness (*e.g.*, in healthy people with a positive family history), and, as such, facilitate more effective and targeted interventions44. Moreover, this also highlights a main challenge of future “ERP research applied to the clinic” to develop novel interventions/manipulations that could modify an ERP of interest. As such, it was found for instance that performing a secondary dual-task resulted in a reduced ERN, and this reduction was larger in patients with obsessive-compulsive disorder than in the group of healthy participants[134], as well as a single session of attention bias modification[135] or expressive writing[136], thus suggesting that increased ERNs in clinical anxiety disorders can be normalized, at least temporarily[137]. Increased sensitivity of the P3a and the P3b amplitudes to depression severity are also now observable thanks to the development of adapted new ERP protocols, such as the three-stimulus oddball design[138,139] and bimodal oddball protocols[140,141], respectively. Research assessing the efficiency of a procedure or a treatment to impact an ERP of interest, even in healthy participants, will, therefore, remain of fundamental relevance in the ERP research area.

Many ERP studies have focused on “a single ERP component” (*i.e.*, P50, MMN, P3a, P3b, *etc.*), comparing it in a pathological population *vs* healthy matched control. However, at the clinical level, it has been suggested that although ERPs clearly exhibit high sensitivity and predictive power, they suffer from poor specificity[26,33]. The idea that a “multivariate endophenotype”, based on a weighted combination of diverse electrophysiological features, may provide more information than any single endophenotype, is not novel[142]. Price and colleagues compared and contrasted four electrophysiological endophenotypes — MMN, P50, P300, and antisaccades — and showed that this combination of features decreased the impact of group heterogeneity. In the same vein, at an individual level, a prominent idea is that a psychiatric patient will exhibit various cognitive disorders, of varying severities, that will subtend their own clinical symptoms. In a dimensional view, such disturbances may evolve differentially, so that some ERP measurements that index a specific cognitive function may recover during a disorder, while others will exhibit long-lasting damage. As an example, we recently showed that a post-cure 3-mo abstinence period in alcoholic patients can be neurophysiologically indexed by an increased No-go P3 yet similar oddball P3 components between the start and the end of a detoxification program[109]. Future studies should hence adopt a multi-component approach in order to potentially increase the sensitivity of ERP recordings, as the change in the patterns of ERPs could be specific (*e.g.*, which components recover, and which remain disturbed) from one psychiatric patient to another. In my opinion, such patterns indexing the changes of various ERP components through test-retest sessions appear to be the best way for ERPs to provide clinicians with relevant information regarding change in the disease (due to a treatment) in a single patient and the residual cognitive impairments that still need to be addressed. Indeed, once cognitive disturbances have been characterized through ERP screening of individual patients, psychiatrists will be able to orient the “cognitive” treatment (individually or in groups that present homogeneous patterns of cognitive deficits). More precisely, specific cognitive retraining procedures could be used to target deficits and to increase cognitive efficiency, as both cognitive training and neuromodulation boosting methods have already been shown to reduce clinical symptoms[143,144].

**CONCLUSION**

The main aim of this opinion review was to present the main arguments in favour of the clinical utility of ERP components to help in the management of psychiatric disorders. ERPs may be of great value to psychiatrists for the identification and monitoring of cognitive processes that should be rehabilitated on a patient-by-patient basis[126]. Such a proposition is limited per se, as, naturally, the complexity of dealing with a mental illness encompasses a large variety of stakeholders, such as psychologists, psychiatrists, nurses, and social workers, as well as neuropsychologists and neurophysiologists. I am, therefore, fully aware that the ERP contribution would be minimal, as moreover, many other EEG tools (*e.g.*, event-related oscillations[145] or microstates[146]) could also be of interest. Also, combining ERPs data with more structural and functional information (through for instance fMRI studies[147,148]) would be of the greatest relevance in order to better capture the pathophysiological mechanisms underlying specific clinical symptoms and orient treatment. I am also fully aware that there is still a long way to go before such a proposition could be widely implemented in clinical care units: If such a procedure could be quite easy to install at a technical level for inpatients in psychiatric clinics in highly developed countries, the situation could be more problematic for lower-income countries, and even more for outpatients visiting on a punctual daily basis a psychiatric office. If such a procedure would reveal high efficiency in the future, economical discussions will have to be undertaken to furnish full access of such a material to all countries in order to (1) manage, monitor and orient treatment for inpatients; and (2) allow straight collaborations between research centers and outpatients’ psychiatric office in order to deliver information to clinicians that could be of help in orienting treatment. In such a view, lack of normative data, technical artefacts linked to the recordings with patients, adoption of clear multisite guidelines, as well as a constructive dialogue between researchers and clinicians in the assessment of a suitable cognitive-ERP battery are still some of the main issues that warrant our full attention. A major issue with such a proposal indeed relates to the fact that clinicians and researchers have to agree on a cognitive ERP-battery that could be used across centres (in terms of its content, but also, naturally, its multisite technical guidelines) and across disorders. Such a battery should be as complete as possible, but not too time-consuming in order for it to be adapted to all types of psychiatric patients (probably approximately a maximum of 45 min for a session recording?). It is nowadays well-accepted that transdiagnostic impairment of cognitive control[83], self-referential processes[112], working memory[113], decision making[114], and attention[115] largely contribute to the real-world socio-occupational impairment common across disorders. Therefore, I am inclined to suggest that such a cognitive ERP-battery should at least include two active and two passive tasks: (1) A Go/No-go task, which appears to be the best-suited task to assess cognitive control[143], and to record the No-go N2, the No-go P3, and the ERN as the main ERPs of interest; (2) A bimodal (visual-auditory) three-stimulus oddball task, in order to probe for updating memory and decision-making processes through the recording of P3a and P3b components[87,140]; (3) A passive auditory paired-stimulus paradigm, classically used to record sensory gating through the P50[149]; and (4) A passive auditory oddball design in order to access the MMN component[150]. Monitoring the changes in these components in a single patient during treatment would be of the greatest clinical interest for identifying neural changes that are positive predictors of the clinical trajectory as well as cognitive functions that still warrant being trained. Clearly, much work is still needed to achieve this aim, such as reaching an agreement regarding the battery content as well as establishing multicenter large sample recordings to obtain normative data and to test the efficiency of the procedure at a clinical level. Nevertheless, as EEG is a cheap method that can be readily implemented in any type of psychiatric care unit, and because ERPs can provide invaluable information regarding the neurocognitive status of a patient as a monitoring and a predictive biomarker tool, I very much think this method deserves attention and should be given more consideration for further development. The challenge for future studies will be to establish whether this procedure, driven by serial follow-up recordings of various ERP components in a singular patient, is efficient enough to be incorporated into novel psychiatric treatment methods.

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