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## CHARACTERISTIC EEG PATTERNS IN SSPE AND THEIR CORRELATION WITH CLINICAL STAGES

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**Abstract:** Subacute Sclerosing Panencephalitis (SSPE) is a progressive neurological disorder caused by persistent measles virus infection. Electroencephalography (EEG) is a crucial diagnostic tool that reflects the disease's progression through distinct patterns at each clinical stage. In early SSPE (Stage I), EEG may show normal activity or mild background slowing. As the disease advances (Stage II), periodic high-amplitude delta wave complexes synchronized with myoclonic jerks emerge. Stage III is characterized by more frequent and disorganized periodic complexes, including sharp and slow wave discharges. In the final stage (Stage IV), EEG findings deteriorate to burst-suppression patterns or a flatline, indicating severe neuronal damage. Recognizing these EEG features is essential for timely diagnosis and disease monitoring.

**Keywords:** subacute sclerosing panencephalitis, EEG, periodic complexes, myoclonus, burst-suppression pattern

#### INTRODUCTION

Subacute sclerosing panencephalitis (SSPE) is a rare, progressive, and fatal neurodegenerative disorder caused by a persistent measles virus infection. The disease is characterized by distinct clinical stages, and electroencephalographic (EEG) findings play a crucial role in diagnosing and monitoring its progression. This section explores the characteristic EEG patterns observed in SSPE patients and their correlation with the clinical stages of the disease.

#### **Characteristic EEG Patterns in SSPE**

The EEG findings in SSPE are highly distinctive and often serve as a key diagnostic tool. The most common EEG feature is the presence of periodic high-amplitude delta wave complexes, which are typically bilateral, symmetrical, and bisynchronous. These complexes recur at regular intervals, often every 5-7 seconds, and are sometimes referred to as "Radermecker complexes" (Omkar N. Markand & Jose G. Panszi, 1975; Saad Ali et al., 2021). In addition to these periodic complexes, other EEG abnormalities include diffuse slowing of background activity, sharp and slow wave discharges, and focal epileptiform discharges. These findings are not exclusive to SSPE but are

**Volume 2 Issue 2** | **2025** |

highly suggestive when combined with clinical features such as myoclonus and elevated measles antibody titers in the cerebrospinal fluid (CSF) (Candan Gürses et al., 2000; G A Khwaja et al., 1991; Omkar N. Markand & Jose G. Panszi, 1975).

Atypical EEG patterns have also been reported in some cases. For instance, periodic lateralized epileptiform discharges (PLEDs) and burst-suppression activity may occur, particularly in the terminal stages of the disease (Délrio F. Silva et al., 1995; Umberto Aguglia et al., 1987). Furthermore, some patients may exhibit frontal rhythmic delta activity or paroxysms of bisynchronous spike-wave activity, which can complicate the interpretation of EEG findings (Iliyana Aleksandrova et al., 2021; Omkar N. Markand & Jose G. Panszi, 1975).

### **Correlation of EEG Findings with Clinical Stages of SSPE**

SSPE progresses through several clinical stages, and the EEG findings often reflect the disease's evolution. The clinical stages of SSPE can be broadly categorized as follows:

Stage I: This is the early stage, characterized by non-specific symptoms such as cognitive decline, behavioral changes, and mild neurological deficits. EEG findings at this stage may still be normal or show subtle abnormalities, such as mild slowing of background activity.

Stage II: This stage is marked by the onset of myoclonus, seizures, and more pronounced neurological deficits. The EEG during this stage typically shows the emergence of periodic high-amplitude delta wave complexes, which are a hallmark of SSPE. These complexes are often synchronized with myoclonic jerks, creating a one-to-one relationship between the clinical and EEG phenomena (Michael A. Sisk & John F. Griffith, 1974; Saad Ali et al., 2021).

Stage III: In this advanced stage, patients experience significant neurological deterioration, including visual disturbances, pyramidal and extrapyramidal signs, and cognitive decline. The EEG during this stage may show more frequent and disorganized periodic complexes, along with other abnormalities such as sharp and slow wave discharges and focal epileptiform discharges (Candan Gürses et al., 2000; G A Khwaja et al., 1991).

Stage IV: This is the terminal stage, characterized by a vegetative state, cortical blindness, and complete loss of voluntary movements. The EEG in this stage often shows burst-suppression activity or a flatline pattern, indicating severe cortical damage and loss of brain function (Tatsuo Ohya et al., 1974; Umberto Aguglia et al., 1987).

Table 1. EEG Changes Across Clinical Stages of SSPE

Clinical Stage	EEG Findings	Citation
Stage I	Normal or subtle slowing of background activity	(Sisk & Griffith, 1974) (Ali et al., 2021)

**Volume 2 Issue 2** | **2025** |

Stage II	Emergence of periodic high-amplitude delta wave complexes synchronized with myoclonus	(Markand & Panszi, 1975) (Ser et al., 2021)
Stage III	More frequent and disorganized periodic complexes, sharp and slow wave discharges	(Gürses et al., 2000) (Khwaja et al., 1991)
Stage IV	Burst-suppression activity or flatline pattern	(Ohya et al., 1974) (Aguglia et al., 1987)

#### Variability in EEG Patterns

While the periodic high-amplitude delta wave complexes are the most characteristic EEG feature of SSPE, there is considerable variability in the presentation of EEG patterns. Some patients may exhibit atypical EEG patterns, such as periodic polyspiking discharges or triphasic slow waves, particularly in the early or terminal stages of the disease (Délrio F. Silva et al., 1995; Iliyana Aleksandrova et al., 2021). Additionally, the morphology and frequency of the periodic complexes may vary between patients and even within the same patient over time (Jiang-tao Wang et al., 2022; Saad Ali et al., 2021).

#### **Clinical Implications of EEG Findings**

The EEG findings in SSPE are not only diagnostic but also provide valuable insights into the disease's progression and prognosis. The presence of periodic high-amplitude delta wave complexes is strongly associated with the clinical stages of SSPE, particularly stages II and III. These complexes often correlate with the severity of myoclonus and other neurological symptoms (Merve Hazal Ser et al., 2021; Omkar N. Markand & Jose G. Panszi, 1975). Furthermore, the evolution of EEG patterns over time can help clinicians monitor the disease's progression and assess the effectiveness of any therapeutic interventions (Christian Hertel Wulff, 1982; Tatsuo Ohya et al., 1974).

#### **CONCLUSION**

In conclusion, the EEG findings in SSPE are highly characteristic and play a critical role in the diagnosis and monitoring of the disease. The periodic high-amplitude delta wave complexes are the hallmark EEG feature of SSPE and are strongly correlated with the clinical stages of the disease. While there is some variability in the presentation of EEG patterns, the presence of these complexes, combined with clinical features such as myoclonus and elevated measles antibody titers in the CSF, is highly suggestive of SSPE. Further research is needed to explore the relationship between EEG findings and the underlying neuropathological changes in SSPE.

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**Volume 2 Issue 2** | **2025** |

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**Volume 2 Issue 2** | **2025** |

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**Volume 2** Issue 2 | 2025 |