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# Serial changes in motor and somatosensory evoked potentials in putaminal haemorrhage

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**Abstract** Little is known about evoked potential changes in putaminal haemorrhage. In this study, somatosensory evoked potentials (SEPs) and motor evoked potentials (MEPs) have been serially evaluated and their role in the prognosis of putaminal haemorrhage is now reported. Nineteen patients with CT- or MRI-proven putaminal haemorrhage were examined after a mean duration of 13 days (range 2–30); there were 4 females and 9 males, ranging in age between 25 and 70 years. The haematomas were of medium size in 13 and large or small in 3 patients each. The changes in the clinical picture and the SEPs and MEPs were evaluated on admission, and after 30 and 90 days. Central motor conduction time (CMCT) could not be recorded in 13, but was prolonged in 2 and normal in 4 patients. Median SEPs revealed the absence of near field potentials in 11 and prolongation of N9–N20 conduction time in 1 patient. In the follow-up period MEP and SEP abnormalities only changed in 5 patients; MEPs changed in 4 and SEPs in 2. The period of normalisation of MEPs ranged between 1 and 6 months. CMCT correlated with motor and N9–N20 conduction time with sensory impairment. Eight patients had poor, 6 partial and 5 complete recovery. Power, sensation, CMCT, and size and location of haematoma made positive contributions to recovery.

**Key words** Somatosensory evoked potentials · Motor evoked potentials · Putaminal haemorrhage · Stroke

## Introduction

Putaminal haemorrhage is the most common type of intracerebral haemorrhage, accounting for 30–50% of cases [18]. The clinical picture of putaminal haemorrhage depends upon its size and extent. A number of clinical parameters, namely severity of hemiparesis, progressive neurological deficits, pupillary abnormalities and CT changes have been reported to be predictors of prognosis of putaminal haemorrhage [6, 16]. Evoked potential studies in these patients may provide an objective documentation of sensory or motor abnormalities which may have a prognostic significance. The pattern of evoked potential abnormalities in putaminal haemorrhage and their sequential changes have not previously been reported. In the present study of putaminal haemorrhage, serial changes in somatosensory (SEPs) and motor evoked potentials (MEPs) have been correlated with clinical and radiological features. The influence of these features on recovery is also emphasised.

## Patients and methods

Clinical and evoked potential studies were carried out in 19 patients with putaminal haemorrhage, whose diagnosis was confirmed by CT in 18 and MRI in 1. In all the patients haemorrhage had occurred at the most 30 days before the examination. All the patients were hospitalised and informed consent was given. Their age ranged between 25 and 70 years; 4 were females. The neuro-

logical examination included assessment of consciousness by the Glasgow Coma Scale; weakness was graded as complete (grade 0–1), partial (grade 2–4) and normal (grade 5) on the 0–5 MRC scale. The muscle tone, reflexes, pinprick, joint position and cortical sensations were also recorded. The severity of stroke was assessed on the Canadian Neurological Scale [2] and the activities of daily life by the Barthel index [11].

Putaminal haemorrhage was diagnosed if the bulk of the blood was in the putamen with or without extension to the other areas revealed by CT. Haematomas were classified as large if the maximum diameter exceeded 4 cm, medium (2–4 cm) and small (less than 2 cm) [1]. The haematomas were also classified according to their location as medial, lateral or subcortical [20].

Median SEPs and MEPs were recorded in all the patients on admission, as soon as their clinical condition permitted.

### Motor evoked potentials

For stimulating the motor cortex, a Digitimer D–180 stimulator, delivering a single shock up to 750 V with a time constant of 50–100 μs, was used. The stimulating electrodes were 1–cm–diameter saline–soaked felt pads mounted on a plastic handle. To activate the abductor digiti minimi (ADM), the cathode was placed at the vertex and anode 7 cm laterally and 1 cm anterior to a line drawn from vertex to tragus. For cervical spinal cord stimulation, the cathode was placed below the spinous process of the seventh cervical vertebra (C7) and the anode proximal. In order to obtain the maximum response, the patient was asked to contract the ADM slightly (10% of the maximum force) irrespective of the degree of weakness. Three responses were obtained at 10–s intervals. MEPs were recorded by surface electrode from the ADM in a belly tendon montage. The EMG signal was amplified and filtered through 20 Hz – 2 kHz at a gain of 0.5–2 mV/division. Stimulus intensity was 90–100% of the maximum output for cortical and 50–60% for spinal stimulation. On spinal stimulation, MEPs were recorded while the subject was relaxed. Minimum onset latency and amplitude of the negative phase of the MEPs were recorded. Central motor conduction time (CMCT) was calculated by subtracting the latency of MEPs on C7 stimulation from that on cortical stimulation [15]. MEPs were recorded bilaterally in all patients.

### Somatosensory evoked potentials

Median SEPs were obtained by stimulating the median nerve at the wrist. A 0.1–ms square wave pulse was delivered at 3 Hz with an intensity adequate to produce a painless visible twitch of the thumb. The active surface recording electrode was placed at Erb's point and at the contralateral parietal cortex, 3 cm behind and 7 cm lateral to the vertex, with a midfrontal reference. The maximum impedence of the electrodes was below 5 kΩ. The frequency band was 2–3000 Hz and analysis time 100 ms. Five hundred and twelve responses were averaged twice to ensure reproducibility. The latencies at Erb's point (N9) and that of cortical potentials N20, P25, N30, P45, interpeak latencies: N9–N20 and N20–P45 were also measured [10].

The absence of MEPs or any of the cortical wave forms of SEPs was considered abnormal. The normal values of CMCT, and interpeak latencies, i.e. N9–N20 and N20–P45, were compared with the normal values of our laboratory, which were obtained from 32 healthy volunteers. Their mean age was 28.9 (range 18–50) years. The upper limit of normal was defined as mean ± 2.5 SD of controls.

In all the patients clinical and neurophysiological studies were repeated at the end of 1 and 3 months and 8 were followed up for 6 months. Recovery was defined as complete, partial or poor [4] on the basis of the Barthel index score at the end of 3 months. A score of 20 indicated complete, 19–12 partial and below 12 poor recovery [9].

### Statistical methods

To study the effect of various clinical, radiological and evoked potential parameters on recovery, the adaptive least square method was used [17]. This method was employed because the dependent variable, recovery, was classified into three categories (1 = poor, 2 = partial, 3 = complete). The categorisation of independent variables included size (1 = large, 2 = medium, 3 = small), location (1 = subcortical, 2 = lateral, 3 = medial), weakness (1 = complete, 2 = partial, 3 = absent), tone ( 1 = reduced, 2 = increased, 3 = normal), pinprick and joint position sense (1 = absent, 2 = reduced, 3 = normal), CMCT and N9–N20 conduction time ( 1 = unrecordable, 2 = prolonged and 3 = normal) and raw scores on the Canadian Neurological Scale. MEPs, SEPs and corresponding motor and sensory findings at the time of admission, and after 1 month and 3 months were analysed separately using the same statistical method. In this analysis weakness, tone and Canadian Neurological Scale score were correlated with CMCT, pinprick and joint position sensations with N9–N20 conduction time.

## Results

Nineteen patients with putaminal haemorrhage were examined after a mean duration of 13 (range 2–30) days from ictus. The haematomas were of medium size in 13 and small or large in 3 patients each. The locations of the haematomas were medial in 3, lateral in 9 and subcortical in 7 patients. The mean duration of follow-up was 131 (range 30–187) days.

The evoked potential values in the control group are presented in Table 1. The amplitudes of evoked potentials had a wide normal range and therefore were not used for defining the abnormality. In the study group, the MEPs were not recordable in 13; CMCT was prolonged in 2 and normal in 4. On spinal stimulation the MEPs were normal

**Table 1** Normal values of motor (*MEPs*) and somatosensory evoked potentials (SEPs) (*Lat* latency, *Amp* amplitude)

| Evoked Potential | Mean, SD | Cut-off point |
|---|---|---|
| SEPs | | |
| N9 Lat ms | 11.9, 1.8 | 16.4 |
| Amp μV | 2.7, 1.8 | – |
| N20 Lat ms | 19.5, 1.6 | 23.5 |
| Amp μV | 2.8, 2.2 | – |
| P25 Lat ms | 25.2, 2.7 | 32.0 |
| Amp μV | 7.8, 6.5 | – |
| N30 Lat ms | 32.2, 6.2 | 47.7 |
| Amp μV | 4.9, 4.9 | – |
| P45 Lat ms | 41.1, 4.6 | 52.6 |
| Amp μV | 3.9, 2.8 | – |
| N9–N20 ms | 8.3, 1.2 | 11.3 |
| P45–N20 ms | 22.3, 4.0 | 32.3 |
| MEPs | | |
| Cortex Lat ms | 19.2, 1.2 | 22.2 |
| Amp mV | 3.5, 1.8 | – |
| C7 Lat ms | 13.9, 1.0 | 16.4 |
| Amp mV | 6.1, 1.9 | – |
| CMCT ms | 5.1, 1.2 | 8.1 |

**Table 2** Serial changes in MEPs and SEPs in putaminal haemorrhage *S* small, *M* moderate, *L* large, *CMCT* central motor conduction time, *R* reduced, *A* absent, *Jp* joint and position sense, *Pp* pinprick, *I* increased, *H* hypotonia, *N* normal, *C* complete, *P* partial, *Lat* lateral *Med* medial, *SC* subcoritcal, – not done

| Case | Age (years) /sex | Day of stroke | Location/ size | Canadian Neurological Scale | Tone | Weakness | CMCT (ms) | Sensation | | SEP (ms) | | | Improvement |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | Jp | Pp | N20 | N9–N20 | N20–P45 | |
| 1 | 50/M | 2 | SC/L | 4.5 | H | C | A | A | Red | A | A | A | |
| | | 27 | | 5 | I | P | A | A | Red | A | A | A | Poor |
| | | 93 | | 5 | I | P | A | A | Red | A | A | A | |
| 2 | 36/M | 9 | Lat/M | 4 | H | C | A | Red | Red | – | – | – | |
| | | 19 | | 9,5 | I | P | A | Red | Red | 20.0 | 10.0 | 24.0 | |
| | | 68 | | 9.5 | I | P | 4.6 | Red | Red | 20.0 | 10.0 | 23.4 | Complete |
| | | 157 | | 11.5 | I | N | 5.0 | N | N | – | – | – | |
| 3 | 52/M | 5 | Med/S | 11 | N | P | 6.6 | N | N | 21.4 | 9.2 | 20.6 | |
| | | 65 | | 11.5 | N | N | 6.4 | N | N | 21.2 | 9.0 | 20.8 | Complete |
| | | 173 | | 11.5 | N | N | 4.6 | N | N | 20.4 | 8.2 | 21.8 | |
| 4 | 25/F | 15 | SC/M | 3 | H | C | A | N | N | 19.2 | 8.4 | 20.8 | |
| | | 12 | | 8 | I | P | A | N | N | 20.0 | 10.0 | 21.2 | Partial |
| 5 | 30/M | 30 | Med/M | 1.5 | H | C | A | A | A | A | A | A | |
| | | 90 | | 7 | I | P | A | Red | Red | A | A | A | Partial |
| 6 | 52/M | 22 | SC/L | 1.5 | H | C | A | ? | ? | A | A | A | |
| | | 90 | | 4.5 | I | C | A | ? | ? | A | A | A | Partial |
| 7 | 55/M | 18 | SC/L | 1.5 | H | C | A | ? | ? | A | A | A | |
| | | 86 | | 3 | I | P | A | ? | ? | A | A | A | Poor |
| | | 187 | | 6 | I | P | A | ? | ? | A | A | A | |
| 8 | 58/M | 20 | SC/M | 1.5 | H | C | A | Red | Red | A | A | A | |
| | | 90 | | 7 | I | P | A | Red | Red | A | A | A | Poor |
| 9 | 30/M | 5 | Lat/M | 1.5 | N | C | A | ? | ? | A | A | A | |
| | | 60 | | 6.5 | N | C | A | Red | Red | A | A | A | Poor |
| | | 90 | | 9.5 | I | P | A | Red | Red | A | A | A | |
| 10 | 60/M | 15 | Lat/M | 5 | I | C | A | N | N | 17.6 | 9.0 | 25.4 | |
| | | 30 | | 8 | I | P | A | N | N | 17.2 | 9.2 | 26.8 | Poor |
| | | 180 | | 8 | I | P | A | N | N | 18.6 | 10.6 | 23.6 | |
| 11 | 53/M | 5 | Lat/M | 7 | H | C | A | N | N | 26.4 | 15.6 | A | |
| | | 32 | | 7.5 | I | P | 9.6 | N | N | 22.4 | 12.4 | 24.8 | Partial |
| | | 183 | | 9 | I | P | 5.2 | N | N | 21.7 | 11.3 | 28.3 | |
| 12 | 61/M | 16 | Lat/M | 5 | I | P | 5.6 | N | N | A | A | A | |
| | | 90 | | 9 | I | P | 6.6 | N | N | A | A | A | Partial |
| | | 183 | | 9.5 | I | P | 6.6 | N | N | 19.2 | 9.2 | 28.8 | |
| 13 | 49/F | 15 | SC/M | 5 | I | C | A | Red | Red | A | A | A | |
| | | 30 | | 7 | I | P | A | Red | Red | A | A | A | Poor |
| | | 180 | | 7 | I | P | A | Red | Red | A | A | A | |
| 14 | 70/M | 3 | Lat/M | 3.5 | H | C | A | A | A | A | A | A | |
| | | 30 | | 7.5 | H | C | A | A | A | A | A | A | Poor |
| | | 90 | | 7.5 | H | C | A | A | A | A | A | A | |
| 15 | 45/M | 15 | Med/S | 6 | N | C | 4.4 | A | A | A | A | A | |
| | | 90 | | 9.5 | N | P | 4.4 | A | A | A | A | A | Partial |
| 16 | 60/F | 15 | SC/M | 1.5 | H | C | A | Red | Red | A | A | A | |
| | | 90 | | 4 | I | C | A | Red | Red | A | A | A | Poor |
| | | 180 | | 5.5 | I | P | A | Red | Red | A | A | A | |
| 17 | 45/F | 10 | Lat/S | 9 | I | P | 6 | N | N | 18.8 | 9.2 | 22.4 | |
| | | 30 | | 9 | I | P | 4.8 | N | N | 18.8 | 9.2 | 22.4 | Complete |
| | | 180 | | 11.5 | I | N | – | N | N | 19.0 | 9.0 | 21.0 | |
| 18 | 42/M | 12 | Lat/M | 8.5 | I | P | 10.2 | N | N | 21.0 | 9.4 | 24.5 | |
| | | 30 | | 11.5 | I | N | 6.6 | N | N | – | – | – | Complete |
| | | 120 | | 11.5 | I | N | – | N | N | – | – | – | |
| 19 | 45/M | 6 | Lat/M | 8.5 | I | P | 10.4 | N | N | 20.8 | 10.4 | 23.4 | |
| | | 30 | | 11.5 | I | N | 6.2 | N | N | 21.8 | 11.0 | 23.4 | Complete |

in all the patients, thus excluding a peripheral abnormality. MEPs were abnormal in all the 7 patients with subcortical haemorrhage, in 7 of the 9 patients with lateral and in 1 of 3 patients with medial putaminal haemorrhage.

The patients with a small haematoma had normal MEPs, but in those with large haematomas MEPs were unrecordable. In 12 of 13 patients with medium-sized haematomas MEPs were abnormal (unrecordable in 10 and prolonged CMCT-ADM in 2). Cortical SEPs were unrecordable in 11 and N9–N20 conduction time was prolonged in 1 patient at the initial examination. SEPs were abnormal in 6 patients with subcortical, 4 with lateral and 2 with medial haematomas. SEPs were normal in 2 of the 3 patients with small haematomas. In contrast, in all the patients with large haematomas SEPs were unrecordable. The initial SEP studies were carried out in 12 patients with medium-sized haematoma; SEPs were unrecordable in 8 and N9–N20 conduction time was prolonged in 1 patient. The size of the haematoma had a positive and significant correlation with MEPs ($r = 0.50$); SEPs, however, had a positive but insignificant correlation ($r = 0.32$). Location of haematoma also had a positive correlation with MEPs ($r = 0.37$) and SEPs ($r = 0.16$) but these correlations were not significant.

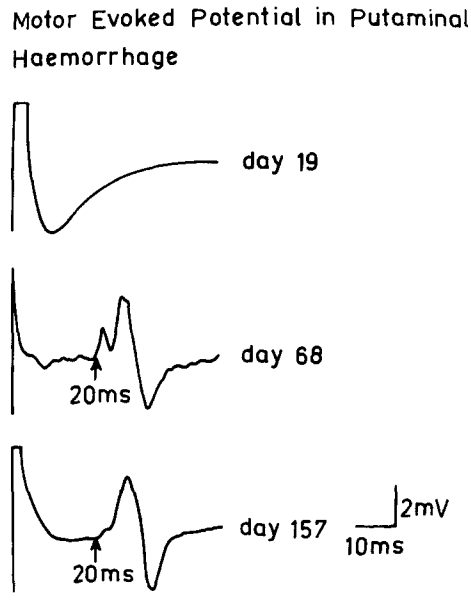


**Fig. 1** Unrecordable central motor conduction time improved to normal in a patient with a moderate-sized lateral putaminal haematoma (patient 2)

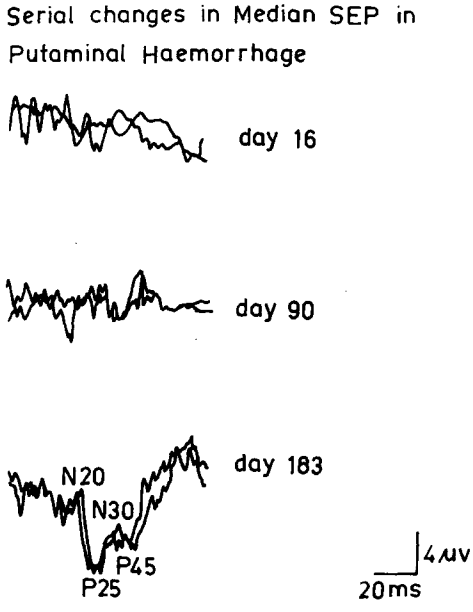


**Fig. 2** Unrecordable somatosensory evoked potential became recordable in a patient with a medium-sized lateral putaminal haematoma (patient 12)

## Follow-up

The details of clinical and evoked potential follow-up studies are presented in Table 2. MEPs and SEPs were stable in most patients. Four patients had changes in their MEPs and 2 in SEPs. All the patients whose MEPs improved (Fig. 1) had improvement in their muscle power as well. Two patients whose SEPs improved (Fig. 2) had no sensory impairment. In 9 patients with unrecordable SEPs sensation continued to be abnormal. The relationship of motor signs with MEPs and sensory impairment with SEPs at different stages of follow-up is shown in Table 3. On admission power and sensory impairment had a significant correlation with respective evoked potentials, which revealed an increasing trend with the duration of follow-up.

On the non-hemiplegic side, CMCT was prolonged in patient 1 (8.8 ms) and patient 6 (9.2 ms) but returned to normal on day 93 and day 90 respectively. Both these patients had large haematomas with evidence of midline shift and tentorial herniation on CT. Both of them had a poor outcome. N9–N20 conduction time on the non-hemiplegic side, however, was normal in all the patients.

The analysis of different clinical, radiological and evoked potential variables revealed a significant positive correlation of size ($r = 0.46$), location ($r = 0.50$), Canadian Neurological Scale abnormality ($r = 0.77$), power ($r = 0.72$), sensations ($r = 0.72$), CMCT ($r = 0.82$) and N9–N20 conduction time ($r = 0.70$) with recovery. Using recovery as the dependent variable, the regression coefficients of different variables are shown in Table 4. Size and location of haematoma, power, sensation and CMCT made positive contributions to recovery. The model thus derived was able to classify correctly all the patients but one. The high value of the Spearman rank correlation coefficient (0.999) indicates the high predictive value of this model.

**Table 3** Correlation coefficients of MEPs and SEPs with respective clinical variables at different stages of follow-up

| | MEP | | | SEP | |
|---|---|---|---|---|---|
| | Canadian Neurological Scale | Tone | Power | Joint position | Pin prick |
| Admission | 0.406 | 0.280 | 0.622* | 0.533* | 0.671** |
| 1 month | 0.756** | 0.337 | 0.632* | 0.671** | 0.661** |
| 3 month | 0.833** | 0.427 | 0.680* | 0.900** | 0.903** |

*$P < 0.05$, **$P < 0.01$

**Table 4** Regression coefficients of different variables on recovery

| Variables | Coefficients | Standard error |
|---|---|---|
| Constant | –4.45 | |
| Size | 0.23 | 0.70 |
| Location | 0.29 | 0.51 |
| Canadian Neurological Scale | –0.04 | 0.31 |
| Tone | –0.48 | 0.85 |
| Power | 1.05 | 0.83 |
| Central motor conduction time | 0.48 | 0.46 |
| Sensation | 2.48 | 1.28 |
| N9–N20 time | –1.11 | 0.76 |

## Discussion

In patients with putaminal haemorrhage unrecordable MEPs in 13 and SEPs in 11 patients were the most frequent abnormalities. These changes can be attributed to damage or pressure on the respective motor or sensory pathways at the level of the internal capsule and adjacent areas. Inexcitability of the central motor pathways has been reported in both coritcal and subcortical lesions, although the prolongation of CMCT has mainly been reported in subcortical lesions [10]. In our study CMCT was prolonged in 2 patients initially and in 1 patient in the follow-up period. Putaminal haemorrhage is less likely to affect the generation of cortical responses except in large haematomas extending to the motor area. Complete interruption of motor pathways may lead to unrecordable CMCT and partial damage to prolongation of CMCT. Partial damage of motor pathways may selectively involve the fast conducting fibres or may produce temporal dispersion of the descending volleys, thus resulting in prolongation of CMCT [19]. In our study CMCT was found to improve not only in the early stage but even 6 months after the ictus (patient 11). Early improvement may be attributed to resolution of the haematoma and clearing of oedema, which may occur in up to 8 weeks [7]. The factors responsible for later recovery of CMCT inlcude reorganisation of the motor pathways and neuronal plasticity [8, 13].

Cortical SEPs were unrecordable in 11 and N9–N20 conduction time was prolonged in 1 of our patients at the initial examination. Compared with MEPs, SEPs were more stable in the follow-up period as they imporved in only 2 patients. The positive correlation between evoked potentials and respective clinical signs highlights the important role of evoked potentials in documenting the respective clinical deficits.

For defining the evoked potential abnormalities, the values on the non-hemiplegic side have been used because of wide interpersonal variation in the SEP values [3]. We depended on the normal values obtained from a control population. In 2 of our patients, CMCT on the non-hemiplegic side was prolonged and it correlated with the midline shift and CT evidence of tentorial herniation. SEPs, however, did not reveal such a change. Prolonged CMCT on the non-hemiplegic side may be due to greater vulnerability of the motor fibres to compression [14]. In tentorial herniation, the cerebral peduncle may be compressed against the tentorium and result in CMCT prolongation on the non-hemiplegic side. In view of these observations, the use of MEPs on the non-hemiplegic side, especially in cases of acute stroke, may not be reliable for defining abnormality.

In the literature the outcome of putaminal haemorrhage is reported to depend on the size [6], location and extent of the haematoma [20]. In our study, both size and location were interrelated ($r = 0.68$) and had significant correlation with recovery. Most of the medial haematomas were small, most lateral haematomas were of medium size, and most subcortical haematomas large. In our study, the recovery correlated with MEPs but not with SEPs. Recordable MEPs predicted a good outcome, because 4 of 6 such patients had complete and 2 partial recovery. In recent studies similar results have been reported. In 118 patients with infarction, those with recordable MEPs had a better outcome than those with unrecordable MEPs [5]. In our own study of pure motor hemiplegia due to putaminal haemorrhage, the patients with recordable MEPs improved [12]. The poor contribution of SEPs to the recovery may be due to the stability of SEPs in the follow-up period.

For predicting the outcome of putaminal haemorrhage a number of clinical variables like the Glasgow Coma Scale, vomiting pupillary asymmetry, gaze palsy, hemianopia, severity of hemiplegia and urinary incontinence have been reported to be useful [6, 9]. A number of these features may be present in the first few days only. The patients in our study were recruited after a mean duration of

13 days (range 2–30). It is therefore not possible to compare the relative usefulness of evoked potentials with a number of these variables. In the acute stage, a number of variables such as sensation, muscle power or hemianopia cannot be reliably assessed because of the altered sensorium. In 3 of our patients, sensation could not be assessed because of altered sensorium or aphasia, and SEPs were unrecordable. MEPs and SEPs have the advantage of objectivity and can be carried out even in a comatose patient.

From this study we conclude that unrecordable MEPs and SEPs were the most common abnormalities in patients with putaminal haemorrhage. MEPs not only were more frequently abnormal but also revealed greater changes than SEPs in the follow-up period, which may explain their better correlation with the outcome.

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