

# Physiological analysis of asterixis: silent period locked averaging

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**SUMMARY** Asterixis was studied in nine patients, using a new electrophysiological technique: silent period locked averaging (SPLA). There were two types of electromyographic (EMG) silence in the movements clinically judged as asterixis. The jerky movement in one of the two types might be caused by the silent period after the subclinical cortical myoclonus. SPLA would be useful for studying asterixis as well as other EMG silences.

Since asterixis was first described by Adams and Foley in patients with hepatic encephalopathy,<sup>1</sup> it has been reported to be commonly found in a wide variety of disorders.<sup>2-5</sup> There has not, however, been any procedure for studying the physiological mechanisms underlying asterixis, other than the electroencephalogram (EEG)—electromyogram (EMG) polygraph.<sup>5,6</sup>

We have described a new technique for the physiological analysis of asterixis: silent period locked averaging (SPLA).

## Subjects

Nine subjects with asterixis were studied, four patients with liver cirrhosis, two with renal failure, two with anticonvulsant intoxication and one with metrizamide intoxication.

## Methods

Both the electroencephalogram (EEG)—electromyogram (EMG) polygraph and silent period locked averaging (SPLA) methods were used.

### EEG-EMG polygraph

EEGs were recorded with silver-silver chloride cap electrodes positioned in accordance with the international 10-20 system. The filter setting of the amplifier was 0.5 to 3000 Hz (-3db).

The EMGs of several muscles were also recorded, including those afflicted by asterixis. Recordings were made with

pairs of saddle type surface electrodes. Filters were set 3db down at 8 Hz (high pass) and 3000 Hz (low pass). EEGs and EMGs were recorded simultaneously, with patients extending their wrists or dorsiflexing their ankles. Accelerometric records of hand or foot also were made in some cases.

### Silent period locked averaging (SPLA)

We constructed a special device (Nihon-Kohden Co. Ltd.) that generates a trigger pulse when the amplitude of the rectified EMG remains lower than a preset level for a preset duration (fig 1). Whenever this device produced a trigger

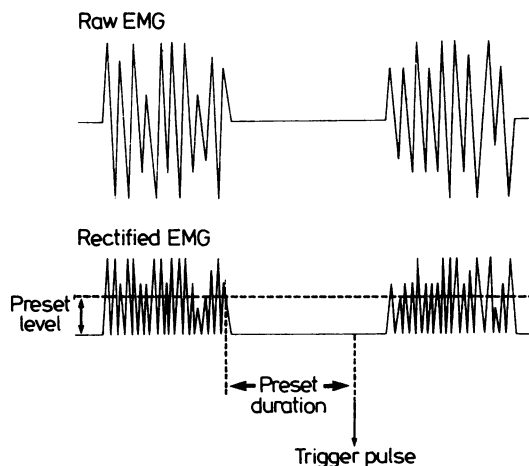


Fig 1 Schematic diagram of the silent period locked averaging [SPLA] method. Asterixis displays electrical silence in surface EMG recording [upper trace]. A trigger pulse is generated when amplitude of rectified EMG activity remains less than a preset level for a preset duration [lower trace]. Backward-averaging is performed using this pulse as a trigger.

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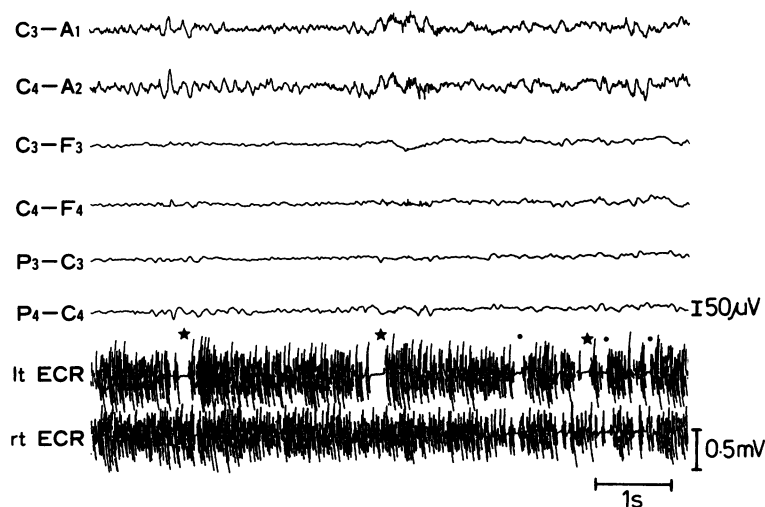


Fig 2 EEG-EMG polygraph in patient 7. EMG silences are irregularly seen in both ECR muscles. Some of them (Type I) followed background EMG activities (·), and the others (Type II) followed a EMG discharge preceded by a short silent period (\*). No EEG activities are demonstrated to be related to the EMG silences.

pulse, a computer (Signal Processor 7T18, NEC San-Ei Co. Ltd.) stored a sample of raw data of EEGs, EMGs and accelerometric recordings. At maximum, the computer could store 500 samples. From the samples stored, we made two sets of averages of EEGs, rectified EMGs and accelerometric recordings according to the types of the silence in the raw EMG described below, using a backward averaging program triggered by the pulse produced by our device.

## Results

### EEG-EMG polygraph

The EEGs of all patients showed poorly organised background activity. The electrically silent periods typical of asterixis appeared irregularly, when patients voluntarily extended their wrists or dorsiflexed their ankles, in the surface EMG of the extensor carpi radialis (ECR) or the tibialis anterior (TA) muscles. Two types of EMG silence were shown. In one type (Type I), the silent period followed usual EMG background activities (fig 2). In the other type (Type II), the silent period followed a bit large EMG discharge preceded by a short EMG silence (figs 2, 3). Although the EMG discharge in the latter type seemed to be an action myoclonus, we clinically judged the movement as asterixis because accelerometric recording showed that a large movement occurred after the silent period, not at the EMG discharge just before the silent period (fig 3). The EMG patterns similar to the latter type have been described as asterixis judging from the clinical features and the results of EEG-EMG polygraph.<sup>3,57</sup> Only type I EMG silence was shown in six patients (Patient 1–6). In the other three patients (Patients 7–9), EMG silences of both types were

demonstrated. The conventional EEG-EMG polygraph failed to reveal any EEG discharge having a consistent correlation with the EMG silent periods. The durations of the silent periods ranged from 50 to 120 ms. Neither action myoclonus nor resting myoclonus were apparent clinically.

### Silent period locked averaging (SPLA)

SPLA revealed no EEG activity closely associated with the EMG silent period in the six patients in which only type I silent period appeared (fig 4).

SPLA of type I silent period also demonstrated no EEG activity associated with the EMG silence in patients who had both types of EMG silence. However, an EEG activity was demonstrated to be related to type II silent period as described below.

*Patient 7 (renal failure):* SPLA revealed that the type II silent period in the left ECR muscle was closely associated with a negative EEG discharge of about 20  $\mu$ V in amplitude in the right central region (C4) (fig 5). The duration of this sharp wave was 100 to 110 ms. The onset of the sharp wave preceded the EMG discharge just before the silent period by 18 to 20 ms (fig 5). The wave displayed phase reversal in the right central region, indicating it is limited to, or at least dominant over, the central region contralateral to the muscle demonstrating asterixis.

*Patient 8 (renal failure):* SPLA revealed a sharp EEG activity preceding the EMG discharge in the left TA muscle just before the type II silent period by 28–32 ms. This sharp wave displayed phase reversal in the mid-central region.

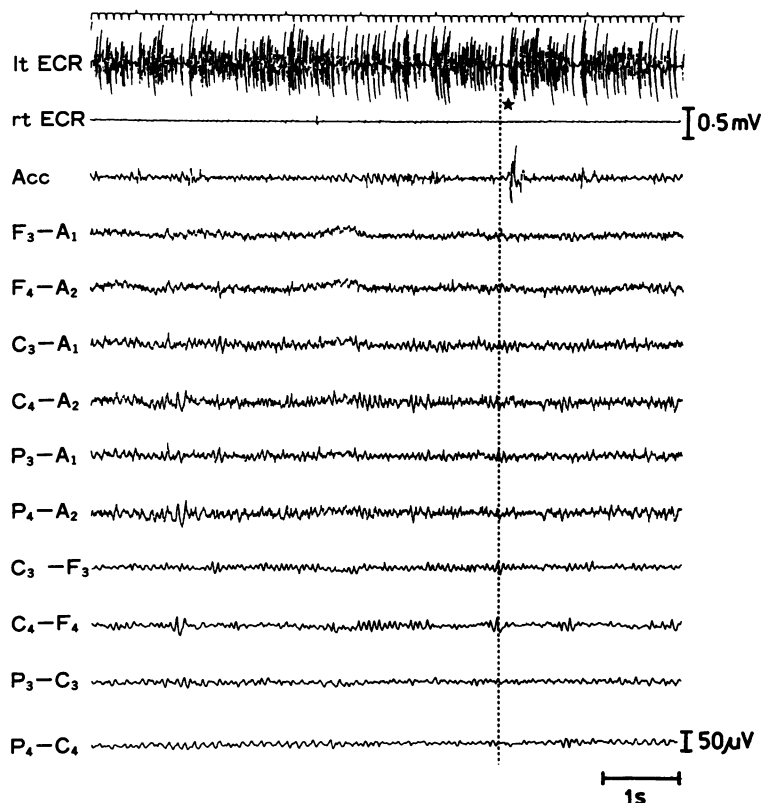


Fig 3 EEG-EMG polygraph in patient 9. A Type II EMG silence is shown in the left ECR muscle (\*). A large movement detected by accelerometer occurs after the silent period, not at the EMG discharge (indicated by a dashed line) prior to the EMG silence.

*Patient 9 (liver cirrhosis):* Onset of type II EMG silent period in left ECR muscle preceded that of acceleration by about 90 ms. Averaged EEG demonstrated a negative sharp wave in the right central region, the onset of which preceded the EMG discharge just before the silent period by about 16 ms.

The temporal relationship between EMG silent period and acceleration did not differ between the two types of the silent period.

### Discussion

The electromyographic characteristic of asterixis is electrical silence in the contracting muscle. Both the mechanism of the electrical silence and the nature of the central defect are still unclear. We devised the new method we refer to as SPLA in order to study asterixis electrophysiologically.

All of our subjects suffered from metabolic disorders previously reported as causing asterixis.<sup>1-7</sup> The EEG-EMG polygraphy, including accelerometric recording, displayed the EMG silent periods charac-

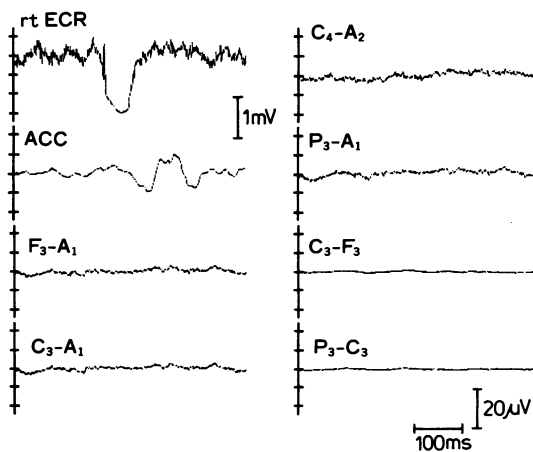


Fig 4 Onset of EMG silence in right ECR muscle preceded acceleration by about 60 ms. SPLA did not demonstrate any EEG activity closely associated with silent period.

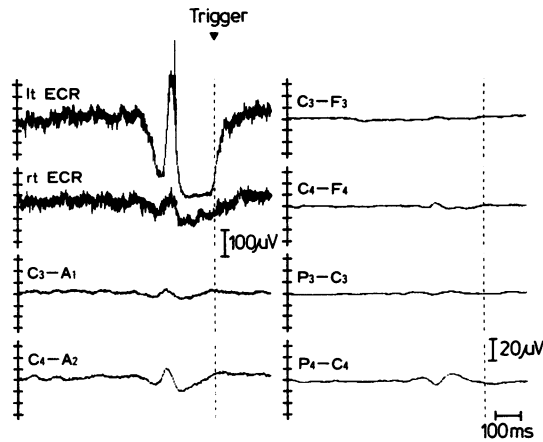


Fig 5 SPLA of type II silent period in patient 7. Averaged EEGs triggered by type II silent period in EMG of left ECR muscle shows a sharp wave over right central region [C4], preceding onset of large EMG discharge just prior to silent period by about 20 ms.

teristic of asterixis. The durations of these silent periods ranged from 50 to 120 ms, their onsets preceding acceleration by about 60 to 100 ms. EEG-EMG polygraphy did not show any waves in EEG consistently associated with asterixis. These results were all compatible with those of previous reports.<sup>5,6</sup>

Two types of EMG silence were demonstrated in the EEG-EMG polygraph. Type I was typical of asterixis, the silent period being preceded by no change in the background EMG activity. Type II followed a brief EMG discharge which might be called an action myoclonus; however, it was clinically judged as a kind of asterixis because the main movement was caused by the EMG silence and similar EMG activities have been previously described as asterixis.<sup>3,5,7</sup> Leavitt *et al.*<sup>6</sup> described the triple pattern of silence, discharge and silence in patients with asterixis. The discharge of this triple pattern appeared just after the acceleration. In contrast, the EMG discharge appeared just before the silent period in type II EMG silence, at the end of which acceleration took place. This large discharge is therefore different from the discharge of the triple pattern of Leavitt *et al.* In the rectified EMG, silent period durations ranged from 50 to 120 ms, and intervals between silent period onset and acceleration from 60 to 100 ms. These values are compatible with those of asterixis reported to date.<sup>5,6</sup>

No EEG correlations were demonstrated for type I EMG silence, by our technique of SPLA. It could be that the motor cortex does not participate in the generation of this type of EMG silence of asterixis, or that the cortical activities are too meagre to be

detected by this technique. Percutaneous electrical stimulation of the human motor cortex has been shown to produce the silent periods, which did not follow large EMG activity in the voluntarily contracting muscles.<sup>8</sup> Some electrical stimulation within the human internal capsule also produced the 50 to 200 ms periods of silence in muscles under voluntary contraction.<sup>9</sup> It would not be surprising, therefore, if the motor cortex participates in the generation of the EMG silence of asterixis. In the present study, however, no EEG activity could be shown to be associated with the silent period not following large EMG discharge (Type I).

In contrast, SPLA revealed a sharp wave in EEG closely associated with the type II silent period. The sharp waves preceding the silent periods of the ECR or TA muscles were seen to undergo phase reversal in the contralateral central or Cz regions, respectively. These sharp waves were thought to be localised in the motor cortices innervating the muscles displaying asterixis. The latencies between the onsets of these sharp waves and the EMG discharges just prior to the silent periods were identical with the intervals from cortical cell discharges to onset of EMG discharges of the respective muscles, as determined by the percutaneous electrical stimulation of the brain (16–20 ms for ECR, 27–34 ms for TA).<sup>10,11</sup> The EMG discharges preceding the type II silent periods were probably generated by the motor cortex. These results were similar to those of the cortical myoclonus<sup>12,13</sup> and suggested that some jerky movements clinically judged as asterixis might be generated by the silent period after the subclinical cortical action myoclonus.

It has not previously been possible, using backward averaging, to assess electrocerebral activity preceding asterixis.<sup>7</sup> Our new SPLA method enables us to use the backward averaging technique for the analysis of asterixis. This method will be useful for studying the origin of asterixis and could possibly lead to an improved classification thereof. It would also enable us to study various kinds of EMG silences as well as those found in asterixis.

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