SHORT REPORT

Onset and offset of electromyographic (EMG) silence in asterixis

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Abstract

The onset and offset of electromyographic (EMG) silence were studied physiologically by silent period locked averaging method (SPLA) combined with a computer-assisted method for detecting EMG changes in 11 patients with asterixis of various aetiologies. The onset followed the EMG discharge which was closely associated with a sharp wave probably generated by the motor cortex in three patients. No EEG activity could be shown to be related to the offset of EMG silence in every patient. Jerky movement of asterixis was temporally related to the offset of EMG silence rather than the onset.

We have recently described a method which allows analysis of EEG events associated with sudden pauses in ongoing EMG activity (asterixis).¹ In this method, a trigger pulse is produced whenever the level of rectified EMG falls below a present level for a preset period of time. This pulse triggers a computer which can collect average data from before and after the trigger point. However, inspection of single trials shows that the precise onset of silence does not always have a constant temporal relation with the trigger point. A similar "jitter" was noted by Barrett *et al*² in their analysis of EEG events preceding onset of voluntary movement. In this paper we have adapted their method of computer assisted averaging to allow manual identification of the trigger point in each single record before averaging.

Subjects

Eleven patients with asterixis were studied and their clinical features are summarised in the table. Two patients had renal failure, five liver cirrhosis, one with metrizamide intoxication, two anticonvulsant intoxication and one shunt encephalopathy. The diagnoses were established on the basis of clinical signs and confirmed by liver biopsy, abdominal angiography, computed tomography and other laboratory studies. Patients 1-9 were the subject of our previous paper on silent period locked averaging (SPLA).¹ Their original data, stored on magnetic tape, were reanalysed using the new method described (see Methods).

Methods

The method involves two separate stages: 1)

data acquisition and 2) manual identification of the trigger points (the onset or offset of EMG silence or other points) before averaging.

Data acquisition

EEGs were recorded with scalp electrodes positioned in accordance with the international 10-20 system. EMGs were recorded from a pair of electrodes spaced 3-5 cm apart over the relevant muscle. The electrical activities from the EEG electrodes were recorded with a time constant of 0.3 s and a high frequency cut-off of 3 kHz by an EEG machine. The EMGs were recorded using a time constant of 0.003 s and a high frequency cut-off of 3 kHz. Accelerometric recordings of the hand or the foot also were made in some patients. All recordings were made while the patients voluntarily extended their wrists or dorsiflexed their ankles. The EMG was rectified and together with the EEG and accelerometric recording (Acc) was sampled by a computer (signal processor 7T18, NEC-San Ei). Whenever the rectified EMG fell below a certain level for a certain time, a special device triggered the computer to store a sample of raw data for a period from 1.024 s before to 1.024 s after the trigger. The computer could store a maximum of 500 samples.

Identification of EMG changes and averaging

The activities for each sample were analysed and averaged according to the method of Barrett *et al*.² When the operator presses a sense key, the computer displays all channels of a sample on the computer screen for an analysis time of 2.048 s. On this screen, a vertical line is drawn together with a window surrounding an analysis time of 0.512 s before the line and 0.512 s following. This vertical line can be moved together with the window using sense keys on the computer keyboard. When the vertical line coincides with the onset of EMG silence, one sense key is pressed to order the raw data in this window to be averaged in one memory box. The operator then moves the vertical line to coincide with the offset of EMG silence. At this time another sense key is pressed to average the raw data in this window in another memory box. The duration of the silent period in this movement was measured as the interval between the onset and offset of the silent period.

If we want to select other points for averaging, we again move the vertical line and press other keys for averaging in other memory boxes. At maximum we can select eight points for averaging in one movement, namely we

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Figure 1 SPLA of the left ECR muscle in patient 1. In SPLA relative to the trigger pulse produced by our device, (A) a negative EEG activity is associated with the onset of the silent period. In SPLA relative to the onset of the large EMG discharge just before the silent period, (B) the steeply increased EMG discharge before the silent period is closely related to a clearly distinguishable EEG activity. SPLA relative to the offset of the silent period (C) demonstrated no EEG activity. The wave associated with the onset of the silent period is not apparent in this record. Long vertical bar at the bottom shows the trigger point for averaging in each figures.

have eight memory boxes. After the analysis of one sample, the raw data of the next sample are displayed, analysed and averaged. This process is repeated for each sample obtained. The averaged data (maximum eight data) are stored in the different memory boxes according to the fiducial points for averaging, such as the onset of EMG silence, its offset, the trigger point transmitted by our device or other points. We can also use different memory boxes according to the different types of EMG silence, if there are two different types of silence in the same subject as described before.¹ Any displayed sample with large artefacts was rejected from analysis.

Results

The results are summarised in the table. Some EMG silences followed the usual background activities (Type I) and the others followed larger EMG discharges (Type II), as described previously.¹

Figure 1 compares the EEG activity associated with Type II asterixis in one patient when different trigger points were used to construct each average. In figure 1A, the trigger point was detected automatically on the criteria of EMG level and duration of the silence.¹ A wave was visible in the EEG, localised over the right central region, with an onset just before a large EMG discharge which

prefaced the silent period. This EEG wave persisted and was slightly larger when the same data were averaged to the onset of this EMG discharge (fig 1B). In this figure, the positive phase of the EEG discharge preceded the EMG discharge by 23 ms, lasted for 80 to 90 ms and had an amplitude of 18 μ V. The onset of the EMG discharge before the silence was seen more clearly in fig 1B than fig 1A. Figure 1C shows SPLA with the offset of the silent period. In contrast to the previous averages, no EEG event was associated with the offset of the silent period. The wave related to the onset of the large EMG discharge, before the silent period, was probably not apparent in this record because of the difference in duration of the EMG silence from one movement to another. Another consequence of this variability was the "smoothing out" of the EMG discharge before the silence.

SPLAs with respect to the onset and offset of Type I EMG silence in patient 9 are shown in fig 2A and 2B, respectively. Neither the onset nor the offset of the silent period were associated with any EEG activity. The amplitude of the averaged Acc in SPLA relative to the offset was about twice as large as that in SPLA relative to the onset. The EMG discharge terminating the silent period occurred about 15 ms after the onset of the acceleration (fig 2B). The duration of silent periods measured in the raw data were 35 to 140 ms with a mean of 61.8 ms (SD 20.8).

The onset of the Type II EMG silence was preceded by a sharp wave in three patients (patients 1–3). No EEG activity was associated with the onset of the Type I silent period in any patients. In each patient the offset of EMG

Figure 2 SPLA of the right ECR muscle in patient 9. Any EEG activity is associated with neither the onset (A) nor the offset (B) of the silent period. The amplitude of the accelerometric recording (Acc) is larger in SPLA relative to the offset of the silent period than that in SPLA relative to the onset. Acceleration precedes the EMG discharge terminating the silent period by about 15 ms.

Table Clinical features and physiological results

Case	Age	Sex	Diagnosis	Type of EMG silence	SPLA (onset) Duration of wave (ms)	SPLA (offset) Duration of wave (ms)	Acc (offset) Acc (onset)
1	84	M	Renal failure	I	—	—	ND
2	77	F	Renal failure	II	80–90	—	ND
3	70	M	Liver cirrhosis	I	70–80	—	ND
4	80	F	Liver cirrhosis	II	60–70	—	1.4
5	54	M	Liver cirrhosis	I	—	—	1.5
6	37	M	Liver cirrhosis	I	—	—	ND
7	64	F	Metrizamide intoxication	I	—	—	ND
8	56	F	Anticonvulsant intoxication	I	—	—	ND
9	52	M	Anticonvulsant intoxication	I	—	—	2.2
10	51	M	Shunt encephalopathy	I	—	—	1.3
11	37	F	Liver cirrhosis	I	—	—	1.3

M: male, F: female. SPLA (onset): silent period locked averaging (SPLA) with respect to the onset of the silent period. SPLA (offset): SPLA with respect to the offset of the silent period. Acc (offset), Acc (onset): the amplitude of accelerometric recording in SPLA (offset) and SPLA (onset), respectively. — no EEG activity, ND: not done.

silence was not associated with EEG activity, irrespective of the type of EMG silence. In all of the patients in whom accelerometric recording was made, the averaged amplitude of Acc recording was larger in SPLA relative to the offset of the EMG silence than that in SPLA relative to the onset. This implied that the jerky movement of asterixis recorded with the accelerometer was more closely related to the offset of EMG silence rather than its onset. The relative ratios of the amplitude of accelerometric recording in SPLA to the offset of EMG silence (Acc (offset)) to that in SPLA to the onset (Acc (onset)) are listed in the table. This ratio was larger if the duration of the silent period was more variable.

Discussion

Asterixis was studied physiologically with a method we have recently developed. In this method, SPLA¹ was performed to the point determined by a computer-assisted method for detecting the onset or offset of EMG silence in each movement.

In three patients, SPLA relative to the onset of EMG silence revealed a sharp EEG activity preceding the large EMG discharge just before EMG silence. This activity appeared to be localised to the area of motor cortex innervating the muscle under study. These results agreed with those already reported.¹ However, the onset of EMG silence was much clearer and steeper in the averaged rectified EMG using the present method for averaging than in the previous method¹ (see patient 1 (fig 1A, B)). Presumably, manual identification of the trigger point was superior to the electronic method.

This method also enabled us to study EEG activity associated with any point of EMG activity by a backward-averaging programme if we select that point as a fiducial point for averaging. Thus we could study the EEG activity related to the offset of EMG silence. SPLA relative to the offset of EMG silence showed no activity in every patient. This

suggested that the highly synchronous activation might not occur in the motor cortex in the generation of the EMG discharge terminating the silent period.

The onset of limb movement produced by EMG silence was more closely associated with the offset of the silence than the onset. This was true even though the movement itself always began before the end of the silent period. We are not certain of the reason for this. One possibility is that the amount and depth of EMG silence was variable from trial to trial. Because of this the interval between onset of silence and onset of movement was quite variable. The cause of resumption of EMG activity at the end of the silence is not known. However, if the offset of the silent period was triggered by some aspect of movement itself (for example, by a stretch reflex effect), then this would account for the close correlation between limb movement and time of offset of the silent period. The observation that the relative ratio of Acc (offset) to Acc (onset) was larger in patients with a silent period of more variable duration supports this view.

On the basis of these results, we concluded that this new method was superior to the method previously described¹ for the precise physiological analysis of asterixis. In this method, movements with artefact were also easily rejected for averaging.

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2 Barrett G, Shibasaki H, Neshige R. A computer-assisted method for averaging motor-related cortical potentials with respect to EMG onset. *Electroenceph Clin Neurophysiol* 1985;60:276–81.