Spontaneous seizure occurring only with subjective symptoms (somatognosic illusion affecting left upper arm), accompanied by rhythmic, low voltage discharge, most prominent on right frontal or fronto-central areas.

various sources of artificial light (TV, video games). In these cases, patients generally explain self induction with vague motivations and most authors consider auto-induction as a compulsive pleasure seeking behaviour.

Conversely, reports of partial self induced seizures are extremely rare.¹⁻⁴ We have recently observed a patient with spontaneous and self induced simple partial seizures. He was a 43 year old, right handed man without familial neurological disorders and with unremarkable personal history apart from the sporadic occurrence of depressive episodes. At the age of 36 he was involved in a car accident during which he experienced a minor head injury with a brief loss of consciousness. After a few months he began to suffer from ictal episodes, generally diurnal and often occurring many times a day. These attacks began with a subjective sensation which the patient described as an altered perception of the left arm ("as if my arm changed its size, or moved, or were crossed by waves"); this sensation was often followed by a violent adduction of the legs, a rapid versive movement of the head and trunk towards the left side, and, successively, rhythmic clonic jerks of the left eyelid. The whole episode occurred with preserved consciousness and lasted about 30 seconds. These episodes were interpreted as psychogenic and treated with benzodiazepines with poor results. The patient discovered accidentally that he was able to induce these attacks by rubbing his left eye. As physicians were generally puzzled by the description of the episodes, he began to provoke his seizures during medical examinations, to prove the truth of his ailments.

Neurological and funduscopic examination and brain CT scan with contrast enhancement were normal. Basal EEG showed diffuse, low voltage fast activity, without interictal paroxysmal activity. During the recording the patient experienced a spontaneous ictal episode, limited to subjective abnormal sensation in his left arm. The last part of the attack was accompanied by a rhythmic, low voltage discharge, progressively decreasing in

frequency (from 9–10 to 3 Hz) and increasing in amplitude, most prominent on the right frontal or fronto-central areas (figure). During a further polygraphic recording, as no spontaneous episodes were observed, the patient was asked to provoke an attack. He rubbed his left eye and, after about 10 seconds, experienced the abnormal sensation, followed by a sudden diffuse muscle contraction with adduction of the legs and by clonic movements of the left eyelid. In the last part of the episode a focal rhythmic discharge, having the same frequency of the eyelid jerks, was seen on the right frontal region. He was treated with carbamazepine (CBZ) at a dose of 10 mg/kg/day. Both spontaneous and self induced episodes disappeared in a short time. One year later the patient was still free of seizures.

The site of origin of the seizures was difficult to localise, as neuroradiological evaluation was normal and examination with special electrodes was not available. The early ictal semeiology (somatognosic illusion), however, suggested a right parietal or temporo-parietal origin,⁵ while the unilateral eyelid jerks implied a successive spreading of the discharge to a limited area of pre-rolandic cortex. In all documented cases of self induced partial seizures patients induced their seizures because of the resulting pleasant experience ("hedonistic motivation"). In the case reported by Van Reeth¹ the seizures were accompanied by an extremely enjoyable sensation; the patient was able to induce the attacks by means of a casually discovered mechanism (inhaling rapidly and deeply while smoking a cigarette) and considered them as a pleasant diversion. The patient reported by Spadetta and Giachheddu² provoked his seizures by means of apnoea or Valsalva manoeuvre; the reason given for inducing the attacks was the intense feeling of pleasure which accompanied them ("as if I were in heaven"). Jacome *et al*³ reported a patient with gelastic seizures who was able to precipitate his seizures by rapidly hyperextending his trunk and neck, and

provoked them because of the intense sexual pleasure which he experienced before losing consciousness. The patient described by Klass⁴ self induced her pleasant trance-like states, followed by the stereotypical vision of a dancing couple by playing records of music and concentrating intensely. In our case, conversely, the seizure was not experienced as a pleasant episode, and the reason for self induction indicates a peculiar utilitarian motivation: the patient, who casually discovered the possibility of provoking his seizures, used the triggering manoeuvre to persuade the sceptical physicians of the truthfulness of his disturbances.

SALVATORE STRIANO
ROBERTA MEO
LEONILDA BILO
PATRIZIA RUOSI
Department of Neurology,
2nd School of Medicine,
University of Naples,
Naples,
Italy

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F-response during cataplexy

Cataplexy is one of the cardinal symptoms of the narcolepsy-cataplexy syndrome and is characterised by brief, sudden episodes of muscle weakness without loss of consciousness. Although the H-reflex is known to be abolished or clearly diminished during cataplexy,¹ the pathophysiology of the atonia is not well studied. We report the results of the continuously recorded F-responses before, during, and after cataplexy and sleep attack.

The patient was a 77 year old woman who had been suffering from daytime sleep attack, cataplexy, sleep paralysis, and sleep hallucination since the age of 15. The cataplexy lasted seconds or minutes and was often triggered by a sudden emotional surge, such as excitement and surprise. During cataplexy, all limbs were flaccidly paralysed and the deep tendon jerks could not be elicited. Standard polysomnographic recordings showed sleep onset REM sleep, increased REM sleep, and frequent sleep apnoea. The results of nerve conduction study of the right tibial and sural nerves, brainstem auditory evoked potential, and brain computed tomography were normal. The H-reflex was not obtained from the soleus muscle by the electrical stimulation of the tibial nerve. The symptoms were clearly improved by oral administration of imipramine 90 mg per day.

For the recording of polygraphy during cataplexy and sleep attack, the relaxed patient lay on a couch in a semidark, warm and quiet room. In addition to electroencephalography (EEG), electro-oculography (EOG), and submental electromyography, F-response was continuously recorded from the right flexor hallucis brevis muscle by the percutan-

Figure F-responses (A) before, (B) during, and (C) after cataplexy. Amplitude and frequency were markedly suppressed during attack.

eous electrical stimulation on the right tibial nerve at the ankle. The stimuli were adjusted 20% above the threshold of maximum M-response and were delivered at a rate of 0.5 per second. F-response was analysed for one minute periods before, during and after the attacks. The F-response variables noted were shortest latency, mean amplitude, and frequency (the number of F-responses relative to the number of stimuli). The patient was asked to continue speaking, we then judged the onset of cataplexy or sleep attack when she stopped speaking and did not respond to our order of raising her hands. We recorded a cataplexy of 1.5 minutes' duration and a sleep attack of 5 minutes' duration. During the cataplexy the EEG remained similar to the normal base line awake EEG recorded before the attack. The amplitude of

F-response was clearly suppressed and the frequency was reduced during the attack. On the other hand, the shortest latency of F-response did not change (figure). Sleep attack was identified by the EEG finding of sudden onset REM sleep. Decrease in amplitude and frequency of the F-response was also observed during the sleep attack (table). The M-response did not alter during these attacks despite the clear change of the F-response.

Although the physiology of F-response is still unknown, its presence in deafferented limbs² and after myelotomy³ indicates that it depends in part on back firing of motor neurons, and amplitude and frequency of F-response are considered to reflect excitability of the motor neuron.^{2,4,5} During REM sleep motor neuron excitability is depressed with a clear postsynaptic inhibition, which is

Table Amplitude, frequency and shortest latency of F-response before, during and after cataplexy and sleep attack

	Amplitude (SD) (μ V)	Fre- quency (%)	Shortest latency (ms)
Before cataplexy	156 (54)	100	37
During cataplexy	26 (34)	50	37
After cataplexy	132 (40)	100	37
Before sleep attack	188 (62)	100	36
During sleep attack	28 (50)	33	36
After sleep attack	156 (72)	100	36

characterised by sequences of hyperpolarising shifts of motor neuron membrane potential and occasional blocks of antidromically induced spikes.^{6,7} Our results clearly showed marked reduction of amplitude and frequency of the F-response during cataplexy and sleep attack. The observation that F-response was similarly suppressed in cataplexy as in sleep attack suggests a similar mechanism of a depressed excitability of motor neurons in cataplexy as in REM sleep.

TAKANORI YOKOTA
TOSHIO SHIMIZU
HIDEAKI HAYASHI
KAZUHIKO HIROSE
HITOSHI TANABE

Department of Neurology,
Tokyo Metropolitan Neurological Hospital

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