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 compelling pleasure seeking behaviour.

Conversely, reports of partial self induced seizures are extremely rare. We have recently observed a patient with spontaneous and self induced simple partial seizures. He was 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. The patient described his 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 started with 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.

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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, 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. Cataplexy lasted 5 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 abductor hallucis nerve, 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 polygraphic 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 flexor hallucis brevis muscle by the percutan-
were markedly suppressed during attack. The attacks.

nerve per second. 20% electrical response was judged relative to shortest latency, was patient during the period to a and recorded to similar normal of base awake...of F-responses were observed during 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.

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

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

<table>
<thead>
<tr>
<th></th>
<th>Amplitude (2SD) (µV) (%)</th>
<th>Shortest latency (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before cataplexy</td>
<td>156 (54)</td>
<td>100 37</td>
</tr>
<tr>
<td>During cataplexy</td>
<td>26 (54)</td>
<td>50 37</td>
</tr>
<tr>
<td>After cataplexy</td>
<td>132 (40)</td>
<td>100 37</td>
</tr>
<tr>
<td>Before sleep attack</td>
<td>188 (62)</td>
<td>100 36</td>
</tr>
<tr>
<td>During sleep attack</td>
<td>28 (50)</td>
<td>33 36</td>
</tr>
<tr>
<td>After sleep attack</td>
<td>156 (72)</td>
<td>100 36</td>
</tr>
</tbody>
</table>

caracterised by sequences of hyperpolarising shifts of motor neuron membrane potential and occasional blocks of antidromically induced spikes. 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.

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