RAPID CYCLING AFFECTIVE DISORDER ASSOCIATED WITH E.E.G.
ABNORMALITY: A REPORT ON FOUR CASES

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Rapid cycling affective disorder (RCAD) as a distinctive group was first described by Dunner and Fieve (1974), although detailed clinical description of single patient had been published earlier by Bunney and Hartmann (1965). Rapid cyclers are defined as those patients who manifest at least four affective episodes, depression mania or hypomania per year (Dunner and Fieve, 1974). A number of abnormal factors have been described in association with RCAD. Electroencephalographic (EEG) abnormality is one among them.

Fifty patients who fulfilled the Research Diagnostic Criteria for Major Affective Disorder (Mania or depression) were selected to have EEG studies. Both wake and sleep EEG recordings were done. The neur­ologist who read the EEG was blind to the cases selected and the psychiatrist who selected the cases was blind to the EEG findings. Among the fifty patients, five patients showed EEG abnormalities. The abnormalities were confined to the temporal lobe. It was interesting to note that among the five patients who showed evidence of EEG abnormality, four of them showed rapid cycling, in the course of their illness.

Case I

Patient was a forty year old male, with history of depression in the past. He had his first attack in 1981, which remitted without any treatment. The second attack was in August 1983. After this episode, he had attacks of depression in January, March and August 1984 with complete remission in between these attacks. He was started on antidepressant medication in August 1984 and he remained well between September 1984 to December 1984. In January 1985, he developed depressive symptoms while on 75 mg of imipramine. At the time of admission in June 1985, he continued to have depressive symptoms.

A wake and sleep EEG was done. The EEG record showed a background activity of 10-11 Hz up to 50 micro volts over the posterior regions bilaterally which was reactive to eye opening and closure. Photic stimulation induced shift of background activity bilaterally. Hyperventilation did not induce any change. During the drowsy state, few sharp transients were noted from the left temporal. Sleep record showed vertex sharp waves, sleep spindles and diffuse slow waves bilaterally. Impression: deviant record due to the presence of sharp transient over the left temporal region. There is slowing of waves.

Neuropsychological tests were done in this patient. There was mild impairment of left temporal lobe function. He was treated with imipramine and became asymptomatic on 200 mg of imipramine. He was discharged and remained well for about three months after which he developed depressive symptoms again. He recovered after a course of ECT. He was followed up for one year and he remained well on antidepressants.

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Case II

Patient was a 30 year old male admitted in September 1985. His first episode of illness was in June 1978 and it lasted for four months. The symptoms were consistent with the attack of acute manic psychosis. Second attack was in 1983 with symptoms suggestive of depression. The episode lasted for a month and remitted itself. In August 1984, he had an attack of mania followed by an attack of depression in December 1984. There was a short period of normalcy between these attacks. He developed manic symptoms in May 1985, which subsided after a month, but he developed depressive symptoms in July 1985 and had another attack of mania in August 1985.

His EEG revealed paroxysmal burst of high amplitude slow waves in theta range noted bilaterally and synchronously. During spontaneous sleep, paroxysmal burst of slow waves in theta range was noted over both temporal regions. In addition to this, vertex sharp waves, sleep spindles and diffuse slow waves were noted bilaterally.

Impression: bitemporal dysrhythmia, paroxysmal bursts of slow waves in theta range bilaterally and synchronously lasting for 2-3 seconds.

There was evidence of temporal lobe dysfunction in the neuropsychological tests. He was started on lithium and he remained euthymic during the follow-up period.

Case III

Patient was 32 years old medical practitioner who was admitted for treatment in October 1985. He had no psychiatric symptoms until 1974. During the early part of 1974, he developed depressive symptoms. He was treated in the local psychiatric hospital and became euthymic after 6 weeks. He remained well for about 18 months. He had another attack in 1975 which was again suggestive of depression. He became symptom free after 2 months. He remained well for one month and then developed hypomanic symptoms; which lasted for one month. He was started on lithium in 1979. He took lithium for 6 months and then stopped. He remained well, but in 1983, he became symptomatic again. He showed a pattern of rapid cycling between 1983-1985. He had short periods of depressive symptoms followed by a period of normalcy and then hypomanic symptoms. At the time of admission in 1985 he was depressed.

The resting EEG record was characterized by a background activity of 9-10 Hz, 35 microvolts, alpha activity over the posterior regions bilaterally, which was reactive to eye opening and closure. Rare sharp transients were noted over the left temporal. Photic stimulation included minimal shift of background activity bilaterally. Hyperventilation did not induce any change. During sleep, vertex sharp waves, sleep spindles and diffuse slow waves were noted over both hemispheres.

Impression: deviant record due to the presence of rare left temporal sharp transients.

This patient did not show any evidence of dysfunction in the neuropsychological tests. He was restarted on lithium and was also given antidepressants until the depressive symptoms disappeared. He also had paranoid ideas and became very irritable. He was started on carbamazepine as an adjuvant to lithium therapy. He remained symptom free until 1987.

Case IV

This was a 40 year old man who had his first attack of depression in June 1978. Since 1982, these attacks have been more frequent. He also reported a few attacks of hypomania. Both attacks of depression and hypomania were of short duration. There was a period of normalcy in between these attacks, but these periods were very short.

EEG—The resting record was characterized by 9-10 Hz alpha rhythm up to 70 micro-
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volts over both the posterior regions which was reactive to eye opening and closing. Photic stimulation induced shift of background activity bilaterally. Hyperventilation did not induce any change. During sleep, spike discharges were noted from both the temporal regions. Normal sleep pattern was seen over both the hemispheres. 

Impression: bitemporal epileptiform abnormality.

Neuropsychological tests showed evidence of left temporal lobe dysfunction. Patient responded to carbamazepine and remained euthymic throughout the follow-up period.

DISCUSSION

Numerous reports in the past decade have identified various factors associated with RCAD. A major subgroup of RCAD patients are those whose cycling is drug induced. Mattson and Seltzer (1981) reported rapid cycling induced by MAO inhibitors. Kukopulos et al. (1980) suggested that lithium was an inducer of rapid cycling. Other drugs implicated are dibenzoxazepine, L-dopa, peribedal (Gerner et al., 1976) and propanalol (Potter et al., 1982). Another major factor associated with RCAD is clinical or subclinical hypothyroidism. The onset of rapid cycling in the post partum period in some patients further implicates hypothyroidism. Oppenheim (1982) has reported estrogen induced rapid cycling.

Another major factor associated with RCAD is neurological disease or insult, Kelner et al. (1983) described two cases of RCAD associated with multiple sclerosis. Forrest (1983) reported a case of a 45 year old male with a history of right hemispherectomy for intractable seizures developing recurrent depression 12 years later. Himmelhoch (1979) described two cases, one of clear rapid cycling and the other, a hybrid of rapid cycling and mixed state. Both patients had EEG abnormalities, the former with a specific temporal lobe focus. One of these patients was successfully treated with a combination of an anticonvulsant and MAO inhibitor. Himmelhoch (1979) suggests that the more labile mixed and rapid cycling forms of affective illness may be closely related to the affective states and syndromes reported to be associated with epilepsy, especially of the complex partial form.

Jones and Barney (1987) described four cases of bipolar affective disorder associated with mental retardation and rapid cycling. They have suggested that rapid cycling may be associated with cerebral dysfunction and therefore to be more frequent when there is a mental handicap.

In the four patients described, all had EEG abnormality in the temporal lobe. Two of them had bitemporal abnormality. One patient who showed definite bitemporal epileptiform abnormality was treated with carbamazepine and became asymptomatic. The other two patients were treated with conventional drugs and showed good response. Thus no subtyping could be made of these patients depending on their response to treatment. Dunner and Fieve (1974) reported the same finding in their study. EEG abnormalities were found in both temporal lobes. This is in contrast to the theory of non dominant hemispheric dysfunction reported by Flor Henry (1976) and Taylor et al. (1979).

Thomas and Wehr (1988) studied 51 patients with RCAD and the finding suggest that RCAD is phenotypically and genetically related to more typical form of affective disorder. As previously reported by Dunner and coworkers (1974), the age at onset of the affective illness in the rapid cycling patients was strikingly similar to those reported for non RCAD patients. In more than half the cases, patients who ultimately developed rapid cycling began their illness with a pattern of occasional isolated episodes, that resembled the typical course of non RCAD patients. In our data also patients had started having isolated episodes of mood distur-
Rapid cycling developed after a few years.

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