Introduction

This paper compares the EEG abnormalities of thirteen patients whose use of cannabis was associated with psychosis, with:

— a group of cannabis users who claim they have no adverse reactions to this substance;

— a group of schizophrenic patients; and

— a group of patients who had required neurological consultation.

All these patients were in the same age category.

EEG Abnormalities and Psychiatric Diagnosis

There have been reports of excessive theta activity in schizophrenics, patients with psychopathic personalities, behaviour disorders, arrested homosexuals and people suffering from various types of organic psychoses, but so far nothing has been conclusive as far as psychiatric diagnoses and EEG abnormalities are concerned (4, 2, 3, 7, 5, 6).

The recent study by Ritvo et al demonstrated no significant correlations between the clinical diagnosis and EEG findings. However, they did note that 14 per cent of patients with non-organic psychotic reactions had abnormal EEGs (eight out of fifteen-seven). Their results indicated that the presence of organic brain dysfunction was the chief factor which determined the incidence of EEG abnormalities, which they found to be 35 per cent of this category (12).

In his study of ten patients Ames has shown that there can be EEG changes three hours after
the ingestion of 4 to 7 grains of cannabis resin (1).

Professor Miras of Greece has demonstrated EEG changes immediately after hashish smoking (11).

Method

In the past two years thirteen patients, between the ages of sixteen and twenty-two, who had used cannabis (but no other drugs such as LSD, mescaline, STP or MDA) were seen in psychiatric consultation. A breakdown into the descriptive diagnostic categories of these patients is as follows:

1) Schizophrenic-like reaction (prolonged psychosis, two males and one female).

2) Psychotic depressive reaction (prolonged psychosis, four females).

3) Paranoid phenomenon (transient psychosis) associated with:
   a) echo reaction, one male
   b) homosexual panic, one male
   c) phobic state, one male

4) Antisocial personality disorder (transient psychosis) associated with epilepsy, one female. This patient had a previous psychiatric admission and had a history of epilepsy.

5) Amotivational syndrome, two males.

In addition one male developed a series of echo reactions lasting longer than one year. He had taken LSD before and had a previous psychiatric admission, and now developed a psychotic reaction only after using cannabis. Also, two females developed prolonged schizophrenic reactions after using cannabis; however, one had a previous psychiatric admission and both had a previous history of taking amphetamines.
Of those who had a cannabis effect without a history of other drugs, eleven EEG recordings were obtained, seven female and four male. The EEG apparatus used was a Grass Electroencephalogram, Model VI. Only two of the patients had a previous history of EEG abnormality before taking cannabis. One had been investigated for migraine headache, and was discovered to have sharp and theta wave activity, felt to be compatible with migraine. She took cannabis one year after the EEG examination and then developed a psychotic depressive reaction with similar electroencephalogram. The other female had had an epileptic EEG and had been on anti-epileptic medication. On a previous psychiatric admission she was diagnosed as having an antisocial personality disorder. She developed a transient psychosis, followed by a prolonged depressive reaction after taking cannabis.

The EEGs were not done at any specific time after the cannabis was taken. The time ranged from two days to two months after taking either marijuana or hashish. The amount of cannabis reported to have been taken varied, and ranged from one nineteen-year-old male who had taken marijuana continuously for three weeks and presented himself in Emergency in a semi-comatose state, to a twenty-two-year-old female who had 'done up' marijuana on three occasions two months previously, or a nineteen-year-old male who had 'done up hash' on one occasion two months before his EEG recording.

In this study those who developed psychotic reactions to cannabis and required psychiatric help were compared to three other groups. Firstly a small community was used as the source from which to obtain eleven controls who were between the ages of sixteen and twenty-two, who had apparently been using cannabis in varying degrees without obvious psychiatric implications. The method used was that two contacts were made, and from these the eleven people were found who did not appear to have psychiatric problems at that time. Of the eleven, five had used cannabis only, while five had used cannabis and LSD and one had taken cannabis, LSD and amphetamines.

The second group for comparison was a random sample of ten people who had neurological consultations and who also were between the ages of sixteen and twenty-two.

The reasons for neurological investigation ranged from possible convulsive disorders to headaches.

A third group used for comparison were patients between the ages of sixteen and twenty-two.
who were admitted during the past three years to the University of Alberta Hospital with a diagnosis of schizophrenia, and who in addition had electroencephalogram. The total number reviewed here was twenty-nine.

A. Description of EEG Abnormalities. The EEGs were first interpreted by a neurologist who knew only that they were done at the request of the psychiatrist. They were then re-read by a different interpreter, who had no knowledge of the groupings, to check the degree of abnormality. There was a difference of opinion on only two of the sixty-one EEGs. Each of the EEG abnormalities included at least one of the following:

1) Recurrent excess of sharp and theta wave activity (4-7 cps) — this activity was amplified by hyperventilation and was particularly noted in the temporal and frontal regions.

2) Excessive theta wave activity — this again was amplified by hyperventilation and was distinguished from occasional theta wave activity, which was excluded from the abnormal EEGs.

3) Moderately severe slow dysrhythmia, including a diffuse dysrhythmia there were moderate irregular theta waves in the temporal and frontal areas, with some tendency to synchronize.

4) Abnormalities compatible with convulsive disorder — localized theta and sharp activity were predominantly more severe in the right or left temporal lobe region.

Figure 1 is an example of excessive theta wave activity which was noted mostly in the frontal and temporal regions.

B. Comparison of EEG abnormalities. The incidence of EEG abnormalities in the groups under study is depicted in Figure 2. In the group who had psychotic reactions to cannabis and who required psychiatric intervention, ten of the eleven EEGs were abnormal. In the group using cannabis without apparent psychiatric implications, eight of the eleven EEGs were abnormal. In the latter group it should be noted that of the five who took only cannabis, four had electroencephalograms compatible with epileptic disorders (all females). Of the other six, there were three normal and three abnormal records, one of the latter having a pattern suggestive of epilepsy. It appears that in both of these groups there is a higher incidence of electroencephalogram abnormalities than one might expect.

In the
schizophrenic control group eleven of the twenty-nine EEGs were abnormal.

This incidence is comparable to studies reported in the literature.

In the neurological control group, four of the ten EEGs were abnormal.

This small sample shows an increased incidence of EEG abnormalities associated with cannabis usage, and the abnormalities may have a relationship to the effect of cannabis on the individual.

**Discussion**

It appears that there is an increase in the number of abnormal EEGs associated with psychotic reactions linked with cannabis compared to the normal, schizophrenic and neurological controls. It is further noted that cannabis usage without apparent psychiatric implications also has a large number of EEG abnormalities as compared to the normal, schizophrenic and neurological control groups.

Without discussing the topic exhaustively one can offer two plausible explanations: Firstly, a patient with a vulnerable personality who also has an electroencephalogramic 'abnormality after taking cannabis may show both an increased tendency toward psychotic reaction and an EEG abnormality which may or may not be a variant of the previous EEG abnormality. Two patients are mentioned who had EEG abnormalities before they took cannabis and who developed psychotic reactions. This might represent the action of cannabis, that is, the active ingredient, tetrahydrocannabinol, on brain centres with pre-existing abnormalities.
Secondly, a person with a vulnerable type of personality but a normal EEG, after taking cannabis, might have a psychotic reaction plus an abnormal EEG. This process may nor may not be reversible. Marijuana has been noted to produce transient EEG changes and has also precipitated epileptic attacks. It has been thought by some that there is a similarity between the repetitiveness of an epileptic attack and the echo phenomenon of this and other drugs (11, 9).

The echo phenomenon usually occurs at times of psychological stress and after an initial bad trip which itself seems to be associated with emotional conflicts and confused motives. The cannabis-associated form of dementia has been mentioned in Eastern reports (1, 13), which also have noted that there may be a lasting and cumulative effect of marijuana on the brain cells, which might explain the experience of some users that it takes less and less of this drug to feel high — the effect of reversed tolerance (13). This might be an alternative explanation of the echo phenomenon. This organic hypothesis suggests that cannabis in conjunction with any neurological disturbance may be a factor in causing EEG abnormalities, as well as the psychotic reactions. According to this view cannabis might induce a psychosis and also be responsible for EEG abnormalities, both of which might then, in turn, contribute to the altering of the patient's personality. It could be that the active ingredient, tetrahydrocannabinol, acts strongly on patients with these immature brain wave patterns, or even that people who have persistent theta waves have an inherent predisposition to psychosis.

In any case, in this small series, it would appear that cannabis usage does contribute to an increased incidence of EEG abnormalities but further research is urgently needed.

Summary

Comparison was made among the electroencephalograms of eleven patients with psychotic reactions after the use of cannabis, twenty-nine patients admitted with a diagnosis of schizophrenia, ten for neurological consultations and eleven cannabis users with no apparent psychiatric implications. It was found that there was a greater increase in the EEG abnormalities of cannabis users than in the rest. It is therefore suggested that the drug may be a factor in creating the EEG abnormalities as well as contributing to the psychotic reactions observed, especially when the other factors which could create similar EEG abnormalities have been ruled out.
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References


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