

Toxic cannabis psychosis is a valid entity

K. SOLOMONS, V. M. NEPPE, J. M. KUYL

Summary

One hundred black men admitted to hospital with acute psychiatric symptoms were investigated for the presence of urinary cannabis metabolites in order to delineate the psychiatric role played by 'dagga', the potent South African cannabinol, in the study population and to determine the diagnostic value of the entity 'toxic psychosis (dagga)'. Cannabinoids were present in 29% of patients, and 31% were discharged with a diagnosis of toxic psychosis (dagga). Clinical and demographic material was gathered for all patients and no consistent differences were found between dagga-positive and dagga-negative patients or toxic dagga psychotic patients and 'functional' psychotics other than a history of recent dagga use and the dagga screening test result. The latter measure was found to be both more sensitive and more specific than the history of dagga use alone. The findings support the routine use of a simple screening test for dagga in the sample population studied. The study demonstrated the heterogeneous nature of the toxic dagga psychosis syndrome by documenting a variety of different clinical presentations, which included schizophrenia (42%), paranoia (26%), maniform psychosis (16%) and organic psychosis (16%).

S Afr Med J 1990; 78: 476-481.

Epidemiological studies on dagga, the potent southern African cannabinoid, are very scanty: There are no reliable estimates of the incidence of dagga use, intoxication or other associated syndromes for southern African populations.¹⁻¹³ A 1983 study by Nel¹ found measurable amounts of dagga in 83 of 192 violent deaths (43%) in the Durban area. The three youngest people, aged 7 years, 12 years and 13 years, were pedestrians with cannabinoids in their bodies. During the same period, cannabinoids were detected in 15 of 23 cases (65%) where people died suddenly from natural causes.

The ratio of dagga offences per head of population rose from 81/100 000 in 1945 to 198/100 000 in 1970 and 135/100 000 in 1980. These figures compare favourably with figures from Europe.¹⁴ The poor state of knowledge regarding dagga use in South Africa is particularly noticeable with regard to the indigenous black population — the 12 reports published in South Africa have not included the black population.¹³

Department of Psychiatry, University of Witwatersrand, Johannesburg and Sterkfontein Hospital, Krugersdorp, Tvl
K. SOLOMONS, M.B. CH.B., D.O.H., D.T.M. & H., M.MED. (PSYCH.)

V. M. NEPPE, M.B. B.CH., D.P.M., B.A., F.F. PSYCH. (S.A.), M.MED. (PSYCH.), PH.D. (MED.), F.R.C.P.C., DIP. A.B.P.N. (Present address: Division of Neuropsychiatry, Department of Psychiatry and Behavioral Sciences, RP-10, University of Washington, Seattle, Washington, USA)

Department of Chemical Pathology, South African Institute for Medical Research, Johannesburg

J. M. KUYL, M.B. CH.B., F.F. PATH. (S.A.), B.S.C. (Present address: Department of Chemical Pathology, University of the Orange Free State, Bloemfontein)

This study was designed to answer three questions:

1. Is there a value to routine screening for urinary cannabinoids in the indigenous southern African male psychotic population?
2. Does the patient whose urine is positive for cannabinol differ clinically from the patient whose urine is negative?
3. Is there a recognisable psychiatric entity of toxic dagga psychosis, and do subgroups of this entity exist?

Research design

The sample included 110 consecutive black men admitted to a mental hospital with acute psychiatric symptoms. The 110 patients were evaluated to ensure that, after exclusions, at least 100 patients would be available for full study. Patients were excluded if they failed to remain in the ward for the first 7 days after admission for any reason, medical or otherwise.

Patient details were obtained on admission by an unstructured open-ended interview, which evaluated demographic background, current psychiatric status, past medical and psychiatric history, family medical and psychiatric history, and current physical and mental status. A differential diagnosis was formulated and a plan of management determined. The differential diagnosis and management plan was reviewed at each subsequent interview. Each patient was seen by two physicians — a registrar and a psychiatric consultant.

Urine specimens were collected on admission and after 7 days. These were screened for cannabinoids by the EMIT semiquantitative enzyme immunoassay.¹⁵

Patients were rated on the Brief Psychiatric Rating Scale (BPRS) of Overall and Gorham,¹⁶ at the initial interview and on the 7th day. The BPRS has been used extensively in research in many countries. No rating scale of symptoms has been validated in the indigenous South African population and the use of the BPRS was felt to be justified as a way of documenting the overall symptom severity and the change in this over time. The 18 items of the BPRS were scored from 0 (no pathology) to 6 (extreme severity). One physician rated all patients.

Diagnostic criteria

Diagnoses were made according to the diagnostic criteria laid down in the *Diagnostic and Statistical Manual of Mental Disorders*⁶ of the American Psychiatric Association (DSM III).¹⁷ For conditions diagnosed as toxic psychoses, which did not adequately fit the clinical picture, the diagnosis was made using *ad hoc* clinical criteria.

The diagnosis of toxic dagga psychosis was made when all three of the following criteria were present:

1. The patient had either a history of recent dagga use or the patient's urine tested positive for cannabinoid or both.
2. The patient had to be psychotic or have been psychotic shortly before admission to hospital. Psychotic features included: formal thought disorder; hallucinations; delusions; behaviour disturbances; and lack of insight.
3. During hospitalisation the patient had to respond to treatment reasonably rapidly, with clear evidence of return to pre-episode personality functioning. An 8-week period was used as an arbitrary cut-off point. Beyond this period, no-one was diagnosed primarily as having toxic dagga psychosis.

The course which these symptoms ran was also noted, particularly whether they returned to normal on recovery from the acute episode.

The diagnosis of toxic dagga psychosis was further sub-classified into: (i) maniform; (ii) paranoid; (iii) schizophreniform; or (iv) organic; according to the predominant mode of presentation.

In the maniform mode, the patient was agitated and restless, his speech pressured, his thoughts grandiose and he had either circumstantiality, tangentiality or flight of ideas. Sleep was diminished and the patient was overactive in a purposeless way.

The paranoid presentation was noted in patients whose most prominent symptom was paranoid ideation with an appropriate and congruent mood.

The schizophreniform presentation was characterised by blunting of affect; withdrawal; poverty of ideation, volition and movement; bizarre somatic or other delusions and formal thought disorders.

The features characterising the organic presentation were clouding or fluctuation in consciousness, disorientation and cognitive or intellectual deficit.

Data analysis

The data were analysed by the BMDP statistical software package¹⁸ using parametric and non-parametric techniques to ascertain whether any statistically significant differences in any of the parameters could be detected between the patients whose urine tested positive for cannabinoids and those who were negative, and between the toxic dagga psychosis group, and the remaining non-toxic dagga psychosis group. Discriminant analysis was performed on all factors found to differ significantly between both the urine-positive and -negative groups and the toxic dagga psychosis and non-toxic dagga psychosis groups. This was done to determine whether any of the factors, either singly or in combination, could predict the group into which each patient would be classified on the basis of each factor and whether this predictive power was significantly greater than the 50% prediction by simple guesswork.

Results

Demographic findings

The population studied were mostly young men (mean age 28,2 years), single (81%) and childless (83%), who lived in townships in and around Johannesburg (83%). In the main they were poorly educated with a mean educational level of standard 4, and a mean of 6,38 years of schooling. Only 39% had progressed beyond junior school and only 5% had matriculated. Some 7% had received no education at all and 9% had not advanced beyond grade II. The majority of the sample were unemployed (69%) and most of the rest (25%) were employed as unskilled labourers. The study population spoke 8 different languages with Sotho, Zulu, Tswana and Xhosa being the most common. Most of the patients had been committed by the Courts (72%) and a significant minority were voluntary patients (28%). Readmissions accounted for 62% of all admissions with 5% having 5 or more previous admissions. Just over one-third of the study population (38%) had never been psychiatric inpatients before.

The study group tended to be physically small with a mean height of 165 cm and a mean weight of 57,14 kg; as many as 60% weighed less than 50 kg. In spite of all this, the men tended to be physically healthy with avitaminoses and poor nutrition apparent in only 7% (Table I).

TABLE I. DEMOGRAPHIC AND CLINICAL RESULTS: COMPARISON BETWEEN TOTAL POPULATION, URINE-POSITIVE PATIENTS AND TOXIC DAGGA PSYCHOSIS PATIENTS (MEAN \pm SD)

Factor	Total population	Urine positive	Urine negative
Age (yrs)	28,2 \pm 8,7	24,9 \pm 6,2	25,0 \pm 5,7
Height (m)	1,65 \pm 0,09	1,67 \pm 0,10	1,68 \pm 0,10
Weight (kg)	57,1 \pm 10,4	58,9 \pm 10,3	60,2 \pm 9,4
Duration of stay (d)	27,9 \pm 23,7	23,9 \pm 23,0	16,2 \pm 10,8
Dagga last taken (days before admission)	8,3 \pm 13,2	4,4 \pm 6,2	6,2 \pm 8,2
BPRS initial total	18,8 \pm 12,1	16,6 \pm 10,7	17,3 \pm 11,9
	%	%	%
Unmarried	83,0	93,0	86,7
No children	83,0	96,6	90,0
Lifestyle*			
Urban	83,0	82,0	76,7
Rural	9,0	10,3	13,3
Semi-rural	8,0	6,9	10,0
Years at school			
< 6	48,0	58,6	56,7
> 6	52,0	41,4	43,3
Employment			
Nil	69,0	69,9	66,7
Labourer	25,0	25,0	23,3
Language group			
Sotho	30,0	—	—
Tswana	22,0	—	—
Zulu	21,0	—	—
Xhosa	14,0	—	—
Article 4†	28,0	31,0	26,7
Article 9	44,0	27,6	36,7
Article 12	28,0	41,4	36,7
Physical state			
normal	77,0	82,8	80,0
Number of admissions			
1	38,0	33,8	50,0
> 1	62,0	66,2	50,0
History of dagga use	45,0	79,3	93,7
History of alcohol use	60,0	44,8	56,7
BPRS initial score			
0 - 3	53,0	47,9	66,7
4 - 6	47,0	52,1	33,3

* Lifestyle was defined as follows: urban patients living in the major cities; rural patients living on farms; semi-rural patients living in small farming towns.

† Article refers to the Section of the Mental Health Act under which patients are admitted to hospital. Article 4 provides for patients who are admitted voluntarily to hospital by a family member. Article 9 provides for patients who are certified by two doctors via the Courts as involuntary patients. Article 12 provides for patients who are urgently certified by one doctor in a manner which initially bypasses the Courts — those patients are admitted against their will, as with Section 9 patients.
BPRS = Brief Psychiatric Rating Scale

Clinical findings

The primary diagnoses made at the time of discharge or at the end of the study are shown in Table II. Toxic dagga psychosis was diagnosed in 31% of cases. Other diagnoses

TABLE II. DIAGNOSES AT DISCHARGE AND AT END OF STUDY

Primary discharge diagnosis	Frequency (%)
No axis-I psychiatric disorder	7
Toxic dagga psychosis	31
Toxic alcohol psychosis	11
Schizophreniform disorder	8
Schizophrenia	9
Affective disorder	
Major depressive episode	3
Manic episode	4
Organic brain syndromes	
Post-ictal confusional state	4
Neurosyphilis	2
Acute confusional state	1
Phenytoin toxicity	1
Organic amnesic syndrome	1
Other	
Paranoid disorder	3
Atypical psychosis	6
Brief reactive psychosis	1
Schizo-affective disorder	1
Dysthymic disorder	1
Anxiety disorder	1
Temporal lobe epilepsy	2
Chronic alcohol abuse	3
Total	100
Secondary discharge diagnosis	
Chronic substance abuse	20
Toxic precipitation of functional disorder by dagga	3
Toxic precipitation of functional disorder by alcohol	11
Mental retardation	8
Personality disorder	4
Other	
Epilepsy	5
Avitaminosis	2
Trauma	2
Total	55

commonly found included toxic alcohol psychosis (11%), schizophrenia (9%), schizophreniform episodes (8%), affective disorders (7%) and organic brain syndromes (9%). A secondary discharge diagnosis was made in 55% of cases and included chronic substance abuse (20%), toxic precipitation of functional disorders by dagga (3%) and alcohol (11%), mental retardation (8%), personality disorders (4%) and 'other' in the remaining 9%.

The total BPRS score on admission varied between 0 and 52 with a mean of 18,8. This had dropped to 9,1 when rated 7 days later with 62% of the study population scoring less than 10. Whereas 25% of the population was initially assessed as being extremely severely disturbed, only 7% were still found to be as severely disturbed 7 days later.

A history of dagga use was obtained from 45% of the patients. Dagga was reported to have been last taken within the month preceding admission in 27% of cases, between 1 and 6 months preceding admission in 10% and more than 6 months before admission in 8% of cases. No history of dagga use was obtained in the remaining 55% of the study group.

A history of previous alcohol use was elicited from 60% of the study group with 40% having used alcohol within the month preceding admission. No one who was clinically intoxi-

cated with alcohol was present in the study population. When the study ended, 90 days after the first patient entered the study, 76% of the population had been discharged and were still out of hospital whereas 9% had been discharged but readmitted, 12% were still in hospital and the remaining 3% had absconded from hospital without being discharged and were lost to follow-up.

The mean duration of hospital stay was 27 days and most of the population (72%) were discharged within 1 month of admission.

The role of dagga

The test for cannabinoids in the urine was positive in 29 cases (29%). Of these 29, 26 were diagnosed as having toxic dagga psychosis (89,6%) while of the remaining 3 patients, 2 were diagnosed as having schizophreniform episodes with toxic precipitation by dagga and 1 had active neurosyphilis as well as cannabinoids in his urine. In 16 cases (55%) the urine reverted to negative when tested 7 days later. The remaining 13 cases (45%) remained positive.

A history of dagga use within the month preceding admission was elicited in 19 cases (65,5%); 4 patients admitted to dagga use, but not within the preceding month (13,8%), and 6 patients denied any previous dagga use (20,7%).

Of the 71 patients whose urine were negative for cannabinoid metabolites, 5 were diagnosed as having a toxic dagga psychosis (7%) and 8 volunteered a history of dagga use within the month before admission and urine testing (11,2%). Sixteen volunteered a history of previous dagga use but not within the month preceding admission (22,5%) and in 47 cases, no history of previous dagga use was obtained.

Patients with cannabinoid metabolites in their urine differed significantly from those without such signs in that they were 4 years younger (P 0,0053), had fewer children (P 0,02), had fewer side-effects from medication (P 0,03) and were more conceptually disorganised on the BPRS rating (P 0,0034). They also gave a history of previous dagga use more frequently (P < 0,0001) and had been at home more recently before entry into this study than the patients whose urine was negative (P 0,01). They also tended more often to be single (P 0,05), were less likely to consume alcohol (P 0,07) and were more likely to be admitted as urgent certifications than patients with negative urine tests (P 0,07). The first four and latter three factors are not useful in clinically differentiating individual patients despite being statistically significant.

However, on discriminant analysis of these nine parameters, the three variables of previous dagga use, previous alcohol use and conceptual disorganisation taken together raise the possibility of correctly classifying each patient into the urine positive or urine negative group from 50% by guessing to 76,3%. These three variables therefore were useful predictors of whether the patient's urine would test positive or negative.

Toxic dagga psychosis

The diagnosis of toxic dagga psychosis was made in 31 cases (31%) according to the criteria described above. A history of recent dagga use in the month preceding admission was obtained in 18 of the cases (58,1%), a further 8 patients volunteered using dagga in the past but not in the month preceding admission (25,8%) and the remaining 5 (16,1%) denied any previous dagga use. In 26 of these 31 patients, the urinary cannabinoid levels were positive (83,9%). Of the remaining 5 urine-negative patients, 4 had spent 4 or more weeks in a general hospital immediately before admission to this hospital. The 5th person had spent 2 weeks in a general hospital before admission to this hospital.

In 8 cases, the discharge diagnosis was made before the urine results were known, and the diagnoses were subsequently changed to include toxic dagga psychosis after the urine test results were found to be positive. In 2 of these cases, the initial discharge diagnosis was atypical psychosis in mild mental retardates, in another 2 cases, toxic dagga psychosis was added to the diagnosis of post-traumatic confusional episode, atypical psychosis (of dissociative-hysterical kind), alcohol-related seizure (with a normal EEG) and alcoholic hallucinosis was altered to include toxic dagga psychosis. Thus urinary cannabinol results influenced diagnosis.

A history of recent coexisting alcohol use within the month preceding admission to hospital was obtained in 10 cases of toxic dagga psychoses (32,3%). No history of alcohol use at all was obtained in 14 cases (45,2%) and 7 patients claimed to use alcohol but not within the month preceding admission (22,6%). It is possible that the 10 patients who used alcohol together with dagga before the onset of their psychiatric symptoms and admission to hospital were in fact suffering from a mixed dagga and alcohol toxic psychosis. The study design was not able, however, to differentiate the influence of these two factors from each other.

According to the criteria outlined above, the 31 toxic dagga psychosis patients were subdivided into four subcategories as follows: schizophreniform presentation (13 cases; 41,9%); mani-form presentation (8 cases; 25,8%); paranoid presentation (5 cases; 16,1%); and organic presentation (5 cases; 16,1%).

This spread of different presentations in the toxic dagga psychosis group confirms the view that toxic dagga psychosis is probably not a homogenous condition with a single mode of presentation.

Statistically significant differences between the toxic dagga psychosis group and the control group were found with the following variables reflected in Table III — age, height, weight, number of neuroleptic drugs and side-effects *ipso facto* correlate highly with the toxic group. Clinically relevant findings, however, are less neuroleptic agent, shorter duration (half the length) of hospital stay, and less psychomotor retardation.

Differences which tended towards significance included more hostility (*P* 0,09), less overall severity (*P* 0,08) on the initial BPRS rating, less emotional withdrawal (*P* 0,05) and less conceptual disorganisation (*P* 0,07) on day 7 in the toxic group (Table III).

Discriminant analysis performed on these variables produced a classification equation whereby history of dagga use and presence of urinary cannabinoids together significantly increase the possibility of correctly classifying a patient into the toxic or non-toxic groups. History of dagga use alone had a predictive power of 80% and the urine test result had a predictive power of 89%. The two variables together had a combined predictive power of 89,7%. These results indicate that if a person had a history of previous dagga use, there was an 80% chance that he would be diagnosed as having toxic dagga psychosis. If his previous dagga history was not known, there existed a 50% chance that he would be correctly classified into either the toxic or non-toxic group. If the patient had measurable cannabinoids in his urine, then there was an 89% chance that he would be correctly classified as having toxic dagga psychosis. Whereas the addition of the variable urine cannabinoid result increased the chance by 9,7% over previous dagga history alone, the addition of previous dagga history to urinary cannabinoid result only raised the possibility of correct classification by 0,7%. It follows that urinary cannabinoid results were more valuable in diagnosing toxic dagga psychosis than the history of previous dagga use.

The discriminant analysis results also confirmed the finding that in spite of demonstrable statistical significance of the remaining factors, their clinical value for classifying a person into the toxic or non-toxic group, and thus their utility, was negligible.

In this study, a history of dagga use (less than 1 month before admission to hospital), had a sensitivity of 74,0% for the diagnosis of toxic dagga psychosis. A history of any previous dagga use yielded a sensitivity of 57,8%. The sensitivity of the urinary cannabinoid assay for toxic dagga psychosis was 89,6%. The specificity of these two parameters for correctly making the diagnosis of toxic dagga psychosis was 58,1% for the dagga history and 83,9% for the urine cannabinoid assay technique over a history of dagga use alone for the diagnosis of toxic dagga psychosis (Table IV).

TABLE III. DIFFERENCES BETWEEN THE TOXIC DAGGA PSYCHOSIS AND CONTROL GROUPS

Variable	<i>P</i> value
Age	0,0040**
Height	0,0427**
Weight	0,0443**
History of dagga use	0,001***
Positive urinary cannabinoid test	0,0001***
Duration of hospitalisation	0,0001**
No. of neuroleptics in treatment	0,0185**
Side-effects	0,0301**
Motor retardation (initial)	0,0366
Motor retardation (d 7)	0,0341**
BPRS score (initial)	0,0841*
Hostility (initial)	0,0941*
Emotional withdrawal (d 7)	0,0526*
Conceptual disorganisation (d 7)	0,0678

* Of no substance value because differences between groups were too small; statistically significant at *P* < 0,05 level.
 ** *P* < 0,001; substantive differences of clinical value.
 *** Tending to significance; 0,01 < *P* < 0,05.

TABLE IV. SENSITIVITY AND SPECIFICITY OF DAGGA HISTORY AND URINARY CANNABINOID ASSAY FOR TOXIC DAGGA PSYCHOSIS

Measure	True-positive	False-positive	Total	Sensitivity (%)
Sensitivity				
Dagga history	20	7	27	74,0
Urinary assay	26	3	29	89,6
Specificity				
Dagga history	42	13	55	76,3
Urinary assay	66	5	71	92,9

The profile of the typical dagga psychotic in this population was a childless, single, urban, poorly educated, unskilled or unemployed young man of normal weight and height who was physically healthy. He had usually been committed (certified) but might be a first or a readmission. He provided a history of dagga use and might or might not be a drinker. He was psychiatrically ill with a lowish BPRS score and required neuroleptic treatment but was unlikely to develop side-effects. He recovered rapidly and was discharged after 16 days. He was unlikely to present with anxiety, tension, mannerisms, and posturing, guilt or depressed mood and was more likely to present with somatic complaints, emotional withdrawal, motor retardation, excitement and pressured speech. He was likely to be conceptually disorganised, unco-operative and hostile, suspicious, grandiose, hallucinated and disoriented with emotional

blunting and unusual thought content. He might present with either a maniform, paranoid, schizophreniform or organic picture which resolved rapidly.

Discussion

The study was limited by a number of factors. Corroborative information about the patient's current and past psychiatric history from family members, employers and other professionals was unavailable or scanty in most cases. This was due to the socio-economic circumstances of the patients as well as insufficient staffing to permit satisfactory eliciting of details. The reliability of clinical assessments was constrained by factors such as the understaffing and overcrowding of ward where the study was carried out and that it had a very high turnover of patients. An average monthly admission and discharge rate for the 80-bed ward of 100 - 120 was not uncommon. This shortcoming was exacerbated by the lack of other evaluation sources, since no social workers, psychologists or occupational therapists worked on the black admission ward. The nursing staff were as overworked as the psychiatric medical staff and their clinical input was similarly sub-optimal.

Cannabis was the only toxic substance measured in body fluids. Neither alcohol nor other toxins, e.g. methaqualone (Mandrax) was tested for strategic reasons, despite histories of recent alcohol and Mandrax use in 40 and 2 cases, respectively.

The important variable of set and setting, which is important in psychological responses to dagga^{19,20} was not and could not be evaluated and controlled for in the study design. The groups of patients with urine positive and negative for cannabinoids, and toxic dagga psychosis and control patients were not prospectively matched for demographic variables but this was compensated for by the finding that the groups did not differ substantially.

The results were analysed as though no false-positives occurred with the EMIT assay technique, whereas false-positives and false-negatives do occur. No more accurate method of confirming the presence of cannabinoid metabolites, e.g. thin-layer chromatography, gas chromatography/mass spectrometry or radio-immunoassay, was used,²¹⁻²⁴ thereby limiting an accurate assessment. Previous studies comparing the EMIT with more sensitive techniques have found false-negatives to be more of a problem than false-positives;²⁴⁻²⁶ a 2,9% occurrence of false-positives and 18% of false-negatives was found in one study.²⁶ The bias introduced by false-positive results was unlikely to have influenced the results in this study, and an underestimate of cannabis use was more likely than an overestimate.

In spite of these drawbacks, this study sheds light on a number of previously undocumented concerns. Demographic similarity between the toxic dagga psychosis and positive urinary cannabinoid groups was evident, as was similarity between urine-positive and urine-negative groups. Where differences did occur, they were of little clinical value. For example, the age difference was only 4 years and the mean age of all groups was below 30 years. These demographic features and profiles accord with those found in other communities where the toxic dagga psychosis is encountered frequently.²⁷

Dagga was demonstrated to be a factor in a significant proportion of the study population. This substantial frequency correlates well with the frequency with which the diagnosis was made over the same 4-week period the year before and for the entire year during which the study was performed. In the 4-week period of the previous year, 21 out of 68 patients (30%) were discharged with the diagnosis of toxic dagga psychosis and 237 of 720 patients discharged (33%) for the full year were diagnosed as having toxic dagga psychosis. The principal difference between the diagnoses made previously and those

made in this study was that the previous diagnoses were made without any objective evidence that dagga had, in fact, been used.

The clinical presentation of psychotic reactions in association with dagga use in this study population was similar to the variety of psychotic reactions to dagga that have been extensively reported.²⁸⁻²³ The heterogenous nature of the toxic dagga psychosis was ably demonstrated in this study where maniform, schizophreniform, paranoid and organic presentations were all encountered. This study demonstrated the validity and applicability of using the postulated diagnostic criteria for toxic dagga psychosis.

This finding is well supported by the large number of physicians who have, anecdotally, clinically described this entity in areas such as southern Africa where the potency of cannabinol is 20-30-fold greater than American marijuana.^{9,27} In general, marijuana has been perceived as a non-psychotogenic substance in the USA and the clinical demonstration of a real cannabinol psychosis is therefore important.^{34,35}

This study also confirmed the value of routine screening of urine for dagga metabolites in this population. This is for four reasons: (i) dagga metabolites are present in a high percentage of cases (29%); (ii) these findings affect the final discharge diagnosis in a number of patients (35%); (iii) no reliable criteria distinguish demographically or clinically between dagga-using patients and the rest of the population; and (iv) the diagnosis of toxic dagga psychosis is made almost exclusively in urine-positive patients. Theoretically this is almost a tautology but even theoretical tautologies require scientific validation in practice. Thus, the value of a simple, reliable screening procedure that is able to differentiate toxic dagga psychotic patients from the rest is an invaluable aid to patient management, particularly in this clinical setting where so little information is readily available.

REFERENCES

1. Nel JP. The prevalence of cannabinoids and alcohol in body fluids of persons who died violently in the Durban area. *South African Conference on Dagga*. Pretoria: Department of Health and Welfare, 1983: 41-44.
2. Simon AM. A study of drug abuse in a group of South African university students. *S Afr Med J* 1982; 61: 666-668.
3. Herr P, Morley JE. Drug use patterns among South African undergraduates. *S Afr Med J* 1972; 46: 1404-1407.
4. Levin SM, Berman C, Cobb H, McLraith J. Dagga (cannabis) usage among medical students in Johannesburg. *S Afr Med J* 1983; 63: 507-609.
5. Du Toit BM. *Drug Use and South African Students*. (Papers in International Studies, African Series No. 35.) Athens, Oh.: Ohio University Center for International Studies, 1978.
6. Levin A. The pattern of drug taking among drug-dependent South African national servicemen. *S Afr Med J* 1972; 46: 1690-1694.
7. Levin A. 'n Ontleiding van die gebruik van divelsmasmiddels en sekere gevolge daarvan, met klem op *Cannabis sativa*, by 'n monster jongmans opgeroep vir militêre diensplig. Thesis for the degree M.D., University of Pretoria, 1974.
8. Le Roux PHduP, Botha EM. Dagga use in the Cape Peninsula. *South African Conference on Dagga*. Pretoria: Department of Health and Welfare, 1983: 16-26.
9. Van der Burgh C. Some epidemiological aspects of dagga use. *South African Conference on Dagga*. Pretoria: Department of Health and Welfare, 1983: 13-15.
10. Botha EM, Le Roux PJ, Du Pre PJ. *Daggagebruik in die Kaapse Skiereiland*. Bellville: Institute for Social Development, University of the Western Cape, 1981.
11. Logie P, Morley JE, Bensusan AP. The dagga smoker: a survey. *S Afr Med J* 1972; 46: 1400-1403.
12. Rottanburg D, Robins AH, Ben-Arie O, Tegin A, Elk R. Cannabis-associated psychosis with hypomanic features. *Lancet* 1982; 2: 1364-1366.
13. Neethling LP. The extent of the dagga problem in the Republic of South Africa. *South African Conference on Dagga*. Pretoria: Department of Health and Welfare, 1983: 8-11.
14. Solomons K, Neppe VM. Cannabis: its clinical effects. *S Afr Med J* 1989; 76: 102-104.
15. Package insert, EMIT d.a.u. Cannabinoid 20 Assay. Palo Alto, Calif.: Syva Company, 1984.
16. Overall JE, Gorham DR. The Brief Psychiatric Rating Scale. *Psychol Rep* 1962; 10: 799-812.
17. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: APA, 1980: 59-89.
18. Dixon WJ, Brown MB, Engelman C et al. *BMDP Statistical Software*. Berkeley, Calif.: University of California Press, 1961.

19. Smith DE. The acute and chronic toxicity of marijuana. *Psychodelic Drugs* 1968; **2**: 37-47.
20. Jones RT. Marijuana-induced 'high': influence of expectation, setting and previous drug experience. *Pharmacol Rev* 1971; **23**: 359-369.
21. Whiting JD, Manders WW. Confirmation of a tetrahydrocannabinol metabolite in urine by gas chromatography. *J Anal Toxicol* 1982; **6**: 49-52.
22. Whiting JD, Manders WW. The confirmation of 9-carboxy-THC in urine by gas chromatography/mass spectrometry. *Aviat Space Environ Med* 1983; **54**: 1031-1033.
23. Schwartz RH, Hawks RI. Laboratory detection of marijuana use. *JAMA* 1985; **254**: 788-792.
24. Sutheimer CA, Yarborough R, Hepler BR *et al*. Detection and confirmation of urinary cannabinoids. *J Anal Toxicol* 1985; **9**: 156-160.
25. Silber TJ, Getson P, Ridley S, Iosefschn M, Hicks JM. Adolescent marijuana use — concordance between questionnaire and immunoassay for cannabinoid metabolites. *J Pediatr* 1987; **111**: 299-302.
26. Schwartz RH, Willette RE, Hayden GF, Bogema S, Thorne MM, Hicks J. Urinary cannabinoids in monitoring abstinence in a drug abuse treatment program. *Arch Pathol Lab Med* 1987; **111**: 708-711.
27. Nahas GG. Cannabis: toxicological properties and epidemiological aspects. *Med J Aust* 1986; **145**: 82-87.
28. Bensusan AD. Drug pollution — the problem of abuse. *S Afr Med J* 1971; **45**: 834-838.
29. Chopra JS, Smith JW. Psychotic reactions following cannabis use in East Indians. *Arch Gen Psychiatry* 1974; **30**: 24-27.
30. Thacore VR, Shukla DRP. Cannabis psychosis and paranoid schizophrenia. *Arch Gen Psychiatry* 1976; **33**: 383-386.
31. Knight F. Role of cannabis in psychiatric disturbance. *Ann NY Acad Sci* 1976; **282**: 64-71.
32. Rottanburg O, Robins AH, Ben-Arie O, Teggin A, Elk R. Cannabis-associated psychosis with hypomanic features. *Lancet* 1983; **2**: 1364-1366.
33. Harding T, Knight F. Marijuana-modified mania. *Arch Gen Psychiatry* 1973; **29**: 635-637.
34. Negrete JC. What's happened to the cannabis debate? *Br J Addict* 1988; **83**: 359-372.
35. Taschner KL. Psychopathology and differential diagnosis of so-called cannabis psychoses. *Fortschr Neurol Psychiatr* 1983; **51**: 235-248.