currently recommended might improve resuscitation rates after cardiac arrest.[6–13]

The present study and another published in the same issue of the *New England Journal of Medicine*[14] concluded that high-dose epinephrine did not improve the return of spontaneous circulation and survival rates in adult patients who had cardiac arrest compared to the use of standard-dose epinephrine. However, this conclusion is not tenable because in 60% of patients the cardiac arrest was not witnessed and hence the time between the onset of arrest and the receipt of epinephrine might have been prolonged. Even in patients in whom the cardiac arrest was witnessed, the average time prior to administration of epinephrine was 17 minutes. The high-dose regimen was not repeated if there was a failure with the initial high-dose injection. It has been recommended that epinephrine should be repeated at least every five minutes in advanced cardiac life support.[1] A subgroup analysis showed that when epinephrine was administered within 10 minutes of the cardiac arrest, there was a trend towards improved rates of survival to hospital discharge in the high-dose group.

Another possible reason for the authors not finding an apparent difference between the two groups is the concomitant increase in myocardial oxygen consumption resulting from the beta-adrenergic effects of high dose epinephrine particularly in patients with fixed atherosclerotic coronary vessels.[1] This might have offset any beneficial effects of high-dose epinephrine on survival rates. In fact, subgroup analysis showed a higher rate of return of circulation in patients with electromechanical dissociation (who probably have a lower incidence of coronary artery disease) when they were treated with high-dose epinephrine than when they received standard-dose epinephrine. A recent animal study has shown that the use of beta-blockers along with high-dose epinephrine resulted in a trend towards a lower number of deaths in the first 24 hours after resuscitation.[15]

An important finding of the present study is that there were no major adverse reactions to high doses of epinephrine. Of importance, too, was the absence of ventricular dysarrhythmias related to the use of high-dose epinephrine. This will dispel any fear about the use of high-dose epinephrine in future trials.

Most of the human studies reported have shown that the hospital discharge rates have not been significantly different for the standard-dose and high-dose epinephrine groups. This probably reflects the prolonged global cerebral ischaemia present prior to starting CPR which seems to determine the ultimate outcome.

Despite the negative results of the present study, there should be further clinical trials comparing the use of high-dose and standard-dose epinephrine especially in the very early phase of CPR.

REFERENCES

1 Standards and guidelines for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). *JAMA* 1986;**255:**2841–3044.
2 Chamberlain DA. Advanced life support. Revised recommendations of the Resuscitation Council (UK). *BMJ* 1989;**299:**446–8.
3 Redding JS, Pearson JW. Evaluation of drugs for cardiac resuscitation. *Anesthesiology* 1963;**24:**203–7.
4 Brown CG, Werman HA, Davis EA, Hamlin RL, Hobson J, Ashton JA. Comparative effect of graded doses of epinephrine on regional brain blood flow during CPR in a swine model. *Ann Emerg Med* 1986;**15:**1138–44.
5 Brown CG, Werman HA, Davis EA, Hobson J, Hamlin RL. The effects of graded doses of epinephrine on regional myocardial blood flow during cardiopulmonary resuscitation in swine. *Circulation* 1987;**75:**491–7.
6 Niemann JT, Cairns CB, Sharma J, Lewis RJ. Treatment of prolonged ventricular fibrillation. Immediate countershock versus high-dose epinephrine and CPR preceding countershock. *Circulation* 1992;**85:**281–7.
7 Holliman CJ, Bates MA. Review of all studies of cardiopulmonary resuscitation in animal models reported in the emergency medicine literature for the past 10 years. *Am J Emerg Med* 1992;**10:**347–53.
8 Koscove EM, Paradis NA. Successful resuscitation from cardiac arrest using high-dose epinephrine therapy. Report of two cases. *JAMA* 1988;**259:**3031–4.
9 Goetting M, Paradis NA. High dose epinephrine in refractory pediatric cardiac arrest. *Crit Care Med* 1989;**17:**1258–62.
10 Sherman BW, Rutherford WF. Resuscitation from cardiac arrest using high-dose epinephrine. *JAMA* 1989;**261:**990.
11 Martin D, Werman HA, Brown CG. Four case studies: High-dose epinephrine in cardiac arrest. *Ann Emerg Med* 1990;**19:**322–6.
12 Linder KH, Ahnefeld FW, Prengel AW. Comparison of standard and high-dose adrenaline in the resuscitation of asystole and electromechanical dissociation. *Acta Anaesthesiol Scand* 1991;**35:**253–6.
13 Cipolotti G, Paccagnella A, Simini G. Successful cardiopulmonary resuscitation using high doses of epinephrine. *Int J Cardiol* 1991;**33:**430–1.
14 Stiell IG, Hebert PC, Weitzman BN, *et al.* High-dose epinephrine in adult cardiac arrest. *N Engl J Med* 1992;**327:**1045–50.
15 Otto C, Berg R, Milander M, Kern K, Sanders A, Ewy G. Beta blockade attenuates tachycardia and early death after resuscitation from prolonged cardiac arrest using high-dose epinephrine. *Crit Care Med* 1992;**20** (suppl 4):S74.

PRAVEEN AGGARWAL
ANIL GUPTA
Departments of Emergency Medicine and Hospital Administration
All India Institute of Medical Sciences
New Delhi

'Cannabis-induced psychosis' may obscure paranoid schizophrenia

Mathers DC, Ghodse AH. (Department of Psychiatry, St. George's Hospital Medical School, London, UK.) Cannabis and psychotic illness. *Br J Psychiatry* 1992;**161:**648–53.

SUMMARY

The authors describe a controlled prospective study comparing psychotic illnesses with and without current cannabis use. They attempted to delineate specific psychotic symptoms and syndromes associated with cannabis use. The study was conducted among patients with psychotic symptoms admitted to the acute psychiatric wards of two London hospitals, over a period of 18 months. It comprised of 61 patients between 16 and 60 years who had psychiatric symptoms, were not alcoholic or using other illicit drugs in the month before admission and whose urine analysis was positive for cannabis and negative for all other illicit drugs. Forty-three controls met all the other criteria, with cannabis-free urine. Both groups were interviewed within a week, using present state examination (PSE) which is a structured psychiatric interview schedule for increasing the reliability and validity of information obtained from patients.

Follow up interviews and urine examinations were done at one and six months, the interviewer being blind to the urine reports. Drop-out rates were 18% and 23% for subjects and 14% and 21% for controls at one- and six-months follow up respectively.

Subjects were on an average younger than controls, more often males, non-Caucasians, with a past history of psychotic illness and a forensic record. At the initial assessment PSE showed a significant difference between the subjects and controls on five items (changed perception, non-verbal auditory hallucinations, thought insertion, delusions of control and delusions of grandiose ability). Only one item (delayed sleep) differed at one month and no significant differences were found at six months. PSE ratings were used to generate a computer diagnosis with the help of a software package, 'CATEGO'. Comparison of PSE-based computer diagnoses, using CATEGO, did not show any significant difference initially or at follow up visits. The consultants' clinical diagnoses were schizophrenia in 30 subjects and 20 controls, manic depressive psychoses in 15 subjects and 16 controls and other psychoses in 2 subjects and no controls. Of the 10 patients diagnosed as having cannabis-induced psychosis by consultants, 9 had CATEGO diagnoses of paranoid schizophrenia and 1 had manic depressive psychosis. Caucasian patients compared to Afro-Caribbeans showed significantly more sleep problems, loss of libido, flight of ideas and obsessional checking and significantly less culturally influenced delusions.

Urine examination for cannabis was positive for all 61 subjects initially and in 22 on the two follow up visits. Only 5 subjects had their urine positive at all three examinations.

The results of this study suggest that a short-lived psychotic episode, with specific symptoms does occur in clear consciousness after cannabis intoxication. But the most common diagnosis in these patients is schizophrenia. The use of the label 'cannabis-induced psychosis' may obscure a diagnosis of paranoid schizophrenia.

COMMENT

Cannabis is the most commonly used illicit drug all over the world.[1] Prevalence rates for cannabis use among the general population in European countries vary between 7% and 12%.[2,3] India is one of the main cannabis producing countries and here it is ingested as 'bhang' and smoked as 'charas' and 'ganja'. General population and hospital-based studies have demonstrated that in India, cannabis is the second most frequently abused drug next to alcohol.[4]

The relationship between cannabis use and psychosis has been debated for a long time. One of the early investigations carried out by the Indian Hemp Drugs Commission (1893–4), instituted by the British government in India concluded that the moderate use of cannabis produced no injurious effect on the mind, but this was not true with excessive use.[5] Subsequent reports from several countries, including India have suggested that a cannabis-induced psychosis does occur.[6–10]

The association between cannabis use and psychosis has been demonstrated by a number of epidemiological studies[11,12] but it may not be a cause-and-effect relationship. Psychotic persons may be more likely to use cannabis before or during the early stages of their illness. Alternatively, it may precipitate psychosis only in persons who are genetically or psychologically predisposed to it. Most studies have not been able to rule out these possibilities.

The present study attempts to answer these controversial questions using a clinical rather than an epidemiological approach. The presence of specific symptoms during the initial presentation of psychosis in cannabis users strengthens the evidence in favour of cannabis-associated psychosis being different from other acute psychoses. The symptoms showing significant differences in prevalence were an extension of acute effects of cannabis. However, an objective diagnostic process using specified criteria and a computer diagnostic programme assigned the majority of these patients to the category of schizophrenia. Over a six-month follow up, patients with and without cannabis use could not be differentiated by their symptoms. Hence initial cannabis use does not seem to affect the course of psychotic illness. This finding goes against the concept of a chronic cannabis-induced psychosis. The authors suggest that the ill-defined clinical entity of cannabis-induced psychosis is most often a schizophrenic illness. As major advancements have taken place in the treatment of schizophrenia by pharmacological and psychosocial methods, it is important that a diagnosis of schizophrenia is considered in all cases presenting with psychosis associated with cannabis use, so that the correct treatment is undertaken.

REFERENCES

1 Hughes PH, Canavan KP, Jarvis G, Arif A. Extent of drug abuse: An international review with implications for health planners. *World Health Stat Q* 1983;**36**:394–497.

2 Mott J. Self-reported cannabis use in Great Britain in 1981. *Br J Addict* 1985;**80**:37–43.

3 Sybling G, Persoon JMG. Cannabis use amongst youth in the Netherlands. *WHO Bull Narcotics* 1985;**37**:57–61.

4 Sethi BB, Dube S. Recent trends in cannabis research in India. In: Mohan D, Sethi HS, Tongue E (eds). *Drug abuse in India. Series II.* New Delhi:Jaypee Brothers, 1984.

5 Indian Hemp Drugs Commission. Report of the Commission. Simla: Government Printing Office, 1893–4.

6 Negrete JC. What's happened to the cannabis debate? *Br J Addict* 1988; **83**:359–72.

7 Verma LP. Cannabis psychosis. *Indian J Psychiatry* 1972;**14**:241–55.

8 Goyal DS, D'Netto TB. Cannabis: The habit and psychosis. *Indian J Psychiatry* 1974;**17**:238–43.

9 Chopra GS, Smith JW. Psychotic reactions following cannabis use in East Indians. *Arch Gen Psychiatry* 1974;**30**:24–7.

10 Thacore VR, Shukla SR. Cannabis psychosis and paranoid schizophrenia. *Arch Gen Psychiatry* 1976;**33**:383–6.

11 Negrete JC. Cannabis and schizophrenia. *Br J Addict* 1989;**84**:349–51.

12 Thornicroft G. Cannabis and psychosis. Is there epidemiological evidence for association? *Br J Psychiatry* 1990;**157**:25–33.

SHEKHAR SAXENA
Department of Psychiatry
All India Institute of Medical Sciences
New Delhi