



Figure 1. Lead II rhythm strip showing paroxysms of wide QRS complex dysrhythmia. A ventricular fusion beat (second QRS complex) can be seen in the lower portion.

influence of this drug on the central and peripheral sympathetic nervous system. Extremely high doses can have a direct toxic action on heart muscle, thereby resulting in cardiac arrest.

We describe here a patient with accelerated ventricular rhythm arising as a consequence of cocaine abuse. To our knowledge this association has not been previously documented.

A 37-year-old man was admitted to Good Samaritan Hospital with palpitations. He had a 3-year history of drug abuse with methaqualone and hydromorphone. He had attended an outpatient methadone withdrawal program without success. Two days before admission, he began injecting cocaine intravenously, with at least five injections for each 24-h period. The estimated dosage of cocaine amounted to 140 mg. On the day of admission he experienced palpitations after an injection of the drug. He had no history of cardiovascular disease, chest pain, or dyspnea. At physical examination his blood pressure was 120/80 mm Hg and respirations 20/minute. His pulse was irregular, ranging between 90 and 100 per minute. He had puncture sites of the skin overlying antecubital veins.

Results of a complete blood count, urinalysis, SMA-12 (Technicon Instruments Corp., Tarrytown, New York), and serum Na, K, Cl, and CO₂ were within normal limits. Electrocardiogram showed a sinus rhythm with episodes of a wide QRS-complex dysrhythmia at a rate of 94 beats/minute. The ectopic rhythm manifested a left bundle-branch block configuration and was occasionally ushered in by fusion beats, thus suggesting its ventricular origin (Figure 1). A subjective sensation of palpitations correlated with the occurrence of this arrhythmia. The patient was placed on bedrest and observed. There was a progressive decline in the frequency of the paroxysmal dysrhythmia, and after 48 h the patient left the hospital against medical advice.

Cocaine is extracted from the leaves of *Erythroxylon coca*. When administered intravenously in amounts of 4 to 32 mg, there is a dose-related increase in blood pressure and heart rate with the duration of action dependent on the total quantity given (1). Three modes of action may account for the enhanced cardiac automaticity: a direct central sympathetic stimulation; alterations at the peripheral sympathetic nerve endings; and a direct action on the impulse-forming and myocardial tissue.

As a local anesthetic, cocaine is associated with a greater frequency of cardiac arrhythmias compared with other agents used during laryngoscopy (2). The potentiation of catecholamine effect by cocaine was suggested in 1910 and then in 1961 by Trendelenburg (3), who demonstrated that the pretreatment of cats with cocaine produced augmented blood levels of injected norepinephrine when compared with controls. It was postulated that the cocaine inhibited neuronal uptake of the amine, thus translocating norepinephrine from blocked nerve endings to other cell receptors. This action effectively increases the adrenergic response in susceptible organs giving rise to a state of catecholamine supersensitivity. Other inves-

Accelerated Ventricular Rhythm and Cocaine Abuse

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COCAINE has the peculiar quality of being both a local anesthetic and a sympathomimetic agent with powerful central nervous-system stimulant effects. In small doses cocaine can slow the heart rate, but larger amounts produce an increased heart rate and elevation of blood pressure. The latter effects are presumably mediated by the

tigations (4) indicate that cocaine plays a permissive role in accentuating the influence of epinephrine and norepinephrine on cardiac tissue. The positive inotropic effects of cocaine have also been likened to those of tyramine, which evokes norepinephrine release from sympathetic nerve endings.

The recognition of an accelerated ventricular (or fascicular) rhythm in our subject accords with other limited clinical observations on the influence of cocaine on ventricular automaticity. Propranolol has been used with success for the treatment of ventricular bigeminy and multifocal premature ventricular depolarizations arising as a consequence of laryngoscopy done with topical cocaine anesthesia (2). It is generally accepted that accelerated ventricular rhythms per se do not require any pharmacologic intervention (5). Beta blocking agents, however, might be considered as potentially useful drugs for the treatment of symptomatic or potentially lethal arrhythmias noted in association with cocaine abuse.

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REFERENCES

1. FISCHMAN MW, SCHUSTER CR, RESNEKOV L, SHICK JFE, KRASNEGOR NA, FENNELL W, FREEDMAN DX: Cardiovascular and subjective effects of intravenous cocaine administration in humans. *Arch Gen Psychiatry* 33:983-989, 1976
2. ORR D, JONES I: Anaesthesia for laryngoscopy. A comparison of the cardiovascular effects of cocaine and lignocaine. *Anaesthesia* 23:194-202, 1968
3. TRENDELENBURG U: Modification of the effect of tyramine by various agents and procedures. *J Pharmacol Exp Ther* 134:8-17, 1961
4. KALSNER S, NICKERSON M: Mechanism of cocaine potentiation of responses to amines. *Br J Pharmacol* 35:428-439, 1969
5. LICHSTEIN E, RIBAS-MENECLIER C, GUPTA PK, CHADDA KD: Incidence and description of accelerated ventricular rhythm complicating acute myocardial infarction. *Am J Med* 58:192-198, 1975

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