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IMAGES AND VIGNETTES IN ELECTROPHYSIOLOGY

Cocaine-Induced Na+ Channel Blocking Effect

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A 37-year-old man was brought into our emergency department for seizures and treated by lorazepam. He reported having snorted 4 grams of cocaine 3 hours before. He complained of palpitation with no chest pain. The first electrocardiographic evaluation was as shown in **Figure 1A**.

Six hours after intoxication, he became less responsive, partial tonic-clonic seizures relapsed, and the electrocardiogram changed (**Figure 1B**), while blood pressure remained stable. Bedside echocardiography revealed normal ventricular function. His high-sensitivity troponin T level remained normal.

After treatment, the seizures stopped, and the electrocardiogram again evolved (**Figure 1C**). The following hours were uneventful, and he left against medical advice 20 hours following intoxication with a normalized electrocardiogram (**Figure 1D**).

Known as a major cause of ischemic heart events and death in young adults, cocaine can produce an Na+ channel blocking effect in cardiomyocytes, which can impair conduction and lead to severe cardiotoxicity. After the initial management of the airway and hemodynamics, treatment consists of hypertonic sodium bicarbonate infusion to increase the electrochemical gradient across cell membranes, which helps to offload sodium channels.[1] Meanwhile, sympathomimetic effects of cocaine (increased contractile force, tachycardia, and possible coronary vasospasm) and cocaine-induced seizures will increase the myocardial oxygen demand and be a risk of further cardiovascular deterioration.

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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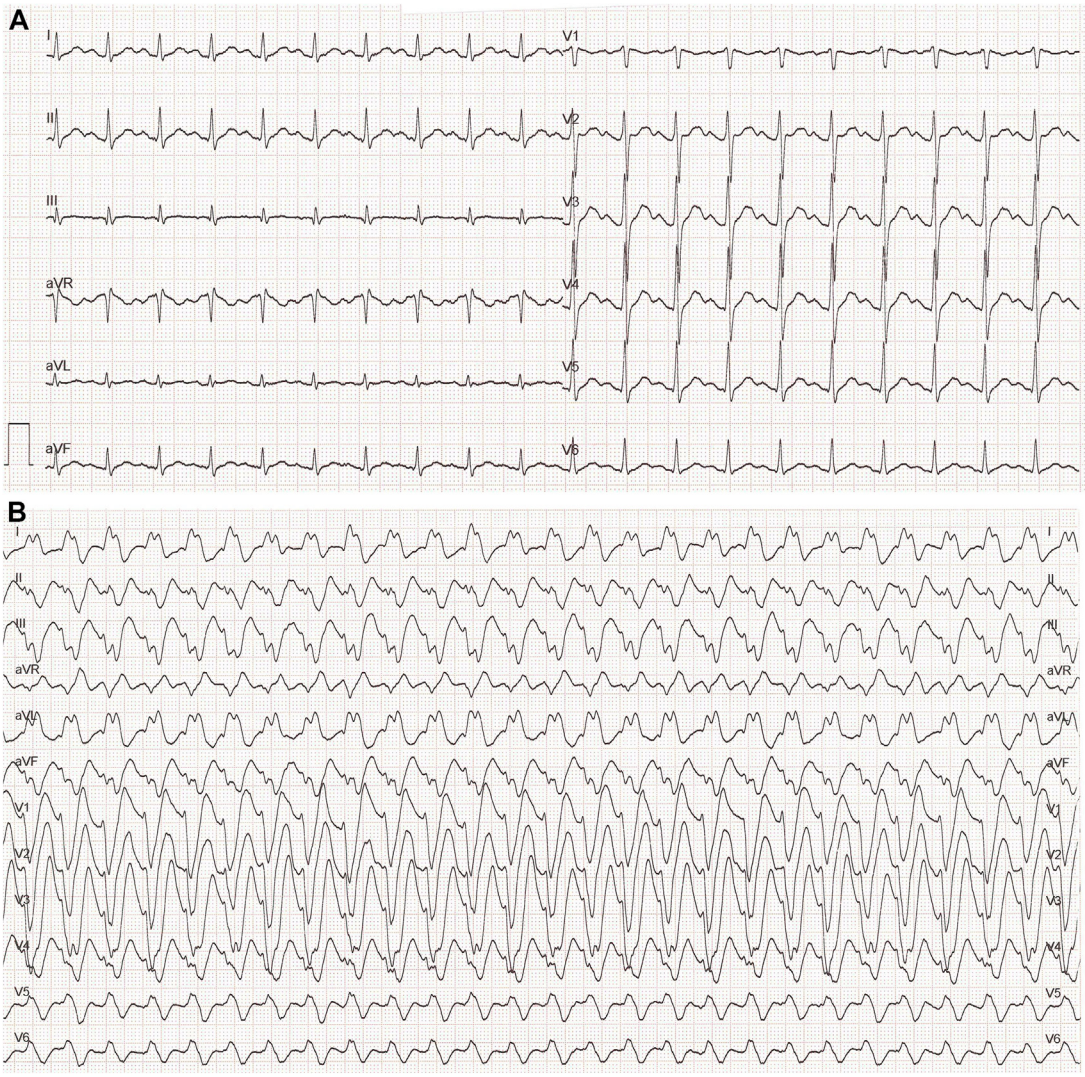
FIGURE 1 Electrophysiological Evolution of the Na+ Channel Blocking Effect Induced by Cocaine Intoxication



(A) Evolution to a wide QRS complex (>200 ms) tachycardia. **(B)** QT interval is also prolonged (>500 ms). **(C and D)** should be suspected in every QT interval (>500ms) and/or QRS complex enlargement (>120 ms). **(E)** A pseudo-Brugada pattern developed when QRS duration narrowed after treatment. All electrocardiograms are scaled as 25 mm/s, 10 mm/mV.

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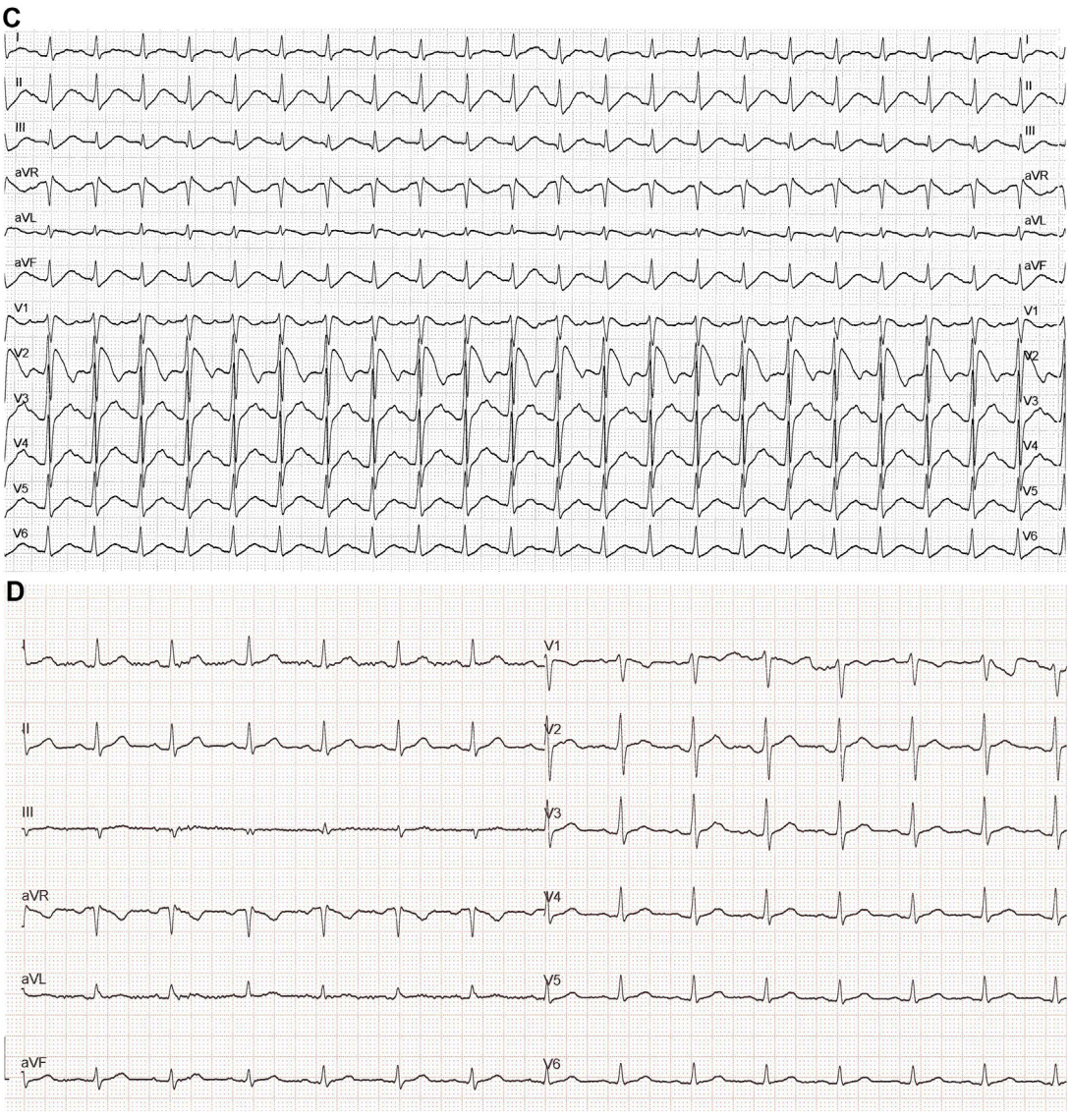
FIGURE 1 Continued



REFERENCE

1. Mauro AD, Pirrone G, Tabita V, Alongi B. Management of sodium-channel blocker poisoning: the role of hypertonic sodium salts. *Eur Rev Med Pharmacol Sci*. 2010;14(1):25–30.

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