posed to cocaine may be explained by increased levels of plasma norepinephrine. The normalization of these values on day 2 is probably due to the gradual disappearance of cocaine from the infant’s body.

Cardiac output and stroke volume are determined by preload, heart rate, afterload, and myocardial contractility and may be affected by various conditions, such as congenital cardiac malformations, patent ductus arteriosus, septicemia, and inotropic drugs. All infants in our study were in a stable clinical condition and did not require any special care and/or medication. Patent ductus arteriosus and congenital cardiac malformations were ruled out by echocardiography. This practically eliminated confounding of our findings by these factors. The relatively small differences in mean gestational age and birth weight between the infants who were exposed to cocaine and the control infants have a negligible effect on cardiac output and/or mean arterial blood pressure values.

We conclude that intrauterine cocaine exposure decreases cardiac output and stroke volume and increases mean arterial blood pressure in the newborn infant. This may have clinical consequences for the infant who was exposed to cocaine with transient myocardial dysfunction due to severe perinatal stress.

REFERENCES


ERRATUM

In the article “Medication Dosages During Pediatric Emergencies: A Simple and Comprehensive Guide” by Tendler et al (Pediatrics 1989;84:731-735), there is an error in Fig 1 on page 733 in the 50-kg column of the Narcan row. The volume listed for 5 mg should be 12 mL (not 1.2 mL).
ERRATUM

Pediatrics 1990:85:32

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