autonomic imbalance. Sympathetic blocks may be a useful adjunct to stimulation when such a problem arises. Further study of the effects of transcutaneous electrical stimulation on autonomic function seems warranted.

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Acute Medicine

SODIUM BICARBONATE AND CARDIAC ARREST Administration of alkali during cardiopulmonary resuscitation has become almost axiomatic. The authors question whether there are dangers in such a practice. Among possible problems are hyperosmolality, myocardial depression secondary to increased carbon dioxide tension, and cerebrospinal fluid acidosis. They therefore evaluated the effects of sodium bicarbonate treatment on blood gases and osmolality in mongrel dogs after ventricular fibrillation had been induced electrically. Following cardiac arrest apnea was allowed to supervene for three minutes, after which cardiopulmonary resuscitation was instituted. Nine dogs were treated (1 mEq/kg of 7.5 per cent sodium bicarbonate administered into the superior vena cava over a 15-second period following one minute of resuscitation), and seven served as controls. In untreated animals, *p*H decreased to 7.22 ± 0.04 (SEM) after 30 minutes of resuscitation. The *p*H remained above 7.35 during the first 15 minutes of resuscitation. Pa_{CO_2} was always below control, while osmolality of arterial blood rose from 310 ± 2 mOsm/kg prior to arrest to 334 ± 5 mOsm/kg after 30 minutes. In dogs treated with alkali, *p*H was 7.38 ± 0.05 prior to bicarbonate administration. A peak level of 7.56 ± 0.06 was observed two minutes following injection of bicarbonate. Pa_{CO_2} increased from 27 ± 2.7 torr prior to bicarbonate treatment to 49 ± 10 torr 15 seconds after therapy. Osmolality rose from 309 ± 5 to 349 ± 11 mOsm/kg two minutes following bicarbonate injection. In six human patients treated with bicarbonate during resuscitation similar data were obtained. The authors conclude that the routine use of sodium bicarbonate should be questioned in a number of circumstances: 1) when carbon dioxide elimination is inadequate; 2) in repeated doses when acidosis has not been confirmed objectively; 3) in the face of only a brief period of cardiac arrest when acidosis may be unlikely. (*Bishop RL, Weisfeldt ML: Sodium bicarbonate administration during cardiac arrest. Effect on arterial pH, P_{CO_2} and osmolality. JAMA 235: 506–509, 1976.*) ABSTRACTER'S COMMENT: The authors do well to call attention to possible dangers of any "routine" therapy. However, since cardiac arrest alone produced an increase in arterial osmolality, bicarbonate therapy only contributed partially to this phenomenon. In addition, only laboratory values were furnished. The complete story will not be known until detrimental functional changes resulting from alkalinization have been documented—*PJC*.