

OLSEN, C. W.; MARINACCI, A. A.; RAY, J. W., AND AMYES, E. W.: *Intravenous Procaine Hydrochloride in the Treatment of Asphyxia due to Carbon Monoxide. Report of Three Treated Cases and Two Untreated Cases with Electroencephalographic Correlation.* Bull. Los Angeles Neurol. Soc. 14: 23-31 (March) 1949.

"Accidental death is more commonly due to carbon monoxide than to any other form of poisoning. . . . Carbon monoxide has no specific toxic action, aside from its affinity for hemoglobin, which is from 200 to 400 times that of oxygen. . . . In the early stages, symptoms of acute or chronic poisoning are not very specific. Headache, dullness, fatigue, nausea, abdominal cramps, and even vomiting and diarrhea do not in themselves suffice to rouse suspicion of poisoning by carbon monoxide. In acute poisoning an extraordinary muscle weakness immediately precedes coma. At this critical stage sudden realization of the danger comes too late, for the victim finds it quite impossible to open a door or raise a window. He may manage to get out of bed, only to collapse on the floor. . . . Beyond the fundamentals of resuscitation, up to now no special method of treatment has been found effective for the relief of acute, and the prevention of chronic, residuals in the nervous system. For this reason the authors considered it worth-while to determine whether the reported beneficial effects of procaine hydrochloride injected intravenously in cases of carbon monoxide poisoning could be reproduced. [Five] cases were selected in which recovery was incomplete, and particularly those in which the residual symptoms could be correlated with cerebral dysrhythmia as shown in electroencephalograms. . . .

"The rationale for this use is based on an analogy that can be drawn between cerebral asphyxia and local

asphyxia elsewhere, as seen in embolism, toxic or reflex anuria, angina pectoris, decubitus and stasis ulcers, Raynaud's disease, frostbite, and pre-gangrenous conditions. In all of these conditions there have been sufficiently impressive results from administration of procaine intravenously to justify further trial of the technique, and even to give rise to definite assertions of its value. There are two 'standard' methods of intravenous use. The French authorities recommend 5 cc. of 1 percent solution in normal saline, injected quite rapidly within one or two minutes. The method advocated by Graubard, in New York, is a unit dose of 4 mg. per Kg. of body weight, diluted to 1:1000 or 0.1 percent in normal saline, to be given intravenously in 20 minutes. A protective dose of barbiturate, such as 0.1 Gm. pentobarbital orally or intravenously, is a prerequisite, especially in the first injection for a patient whose tolerance is uncertain. The recommended antidotes are ephedrine sulphate, 50 mg. by intravenous injection, in case of collapse, and sodium amylal, 0.25 Gm. by intravenous injection in 2 to 5 cc. of distilled water, in case of convulsions. We believe that in the state of dehydration accompanying carbon monoxide poisoning there is a good deal to be gained by intravenous administration of physiologic saline solution, and procaine hydrochloride can be administered simultaneously in the 0.1 percent concentration. This method of administration as well as the more convenient method of administration by syringe injection, of as much as 10 cc. of 1 percent solution, seems to have contributed to the early recovery of the patients who received it. The consistency of correlation between the clinical symptoms and the electroencephalographic findings, even in patients who made rapid recovery, is a remarkable feature of this study."

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