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Effect of Intracisternal Injection of Potassium Phosphate in Hemorrhagic Hypotension and Shock in the Dog.

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According to Stern[1] the injection of small amounts of potassium phosphates into the cisterna magna restores an animal from shock, even when due to loss of blood. The resulting elevation of arterial pressure is attributed to direct stimulation of the sympathetics by the potassium and to decrease in the tonus of the parasympathetics through the decrease in ionization of calcium by the phosphate ion. She also reports favorable results from the cisternal injection of 1-2 cc of the solution on the battlefield. We are reporting herewith the results we have obtained with intracisternal injection into dogs of the potassium phosphate solution employed by Stern.

Seven dogs under intravenous barbital anesthesia were subjected to varying degrees of hemorrhage from the femoral artery. Mean blood pressure was recorded with the mercury manometer from the other femoral artery, and the respirations by thoracic cage tambour. The drawn blood was reinjected immediately and followed by injection of 1/6 molar (isotonic) solution of potassium phosphate (mixture of KH_2PO_4 and K_2HPO_4 brought to pH 7.6).[2,3] by cisternal needle, and with the dog's head in a lateral dependent position. On the basis of 2 cc for a 70 to 75 kilo man, 0.4 to 0.5 cc was considered a therapeutic dose for the 15 to 18 kilo dog.

Injections of the solution at levels of arterial pressure greater than 60 mm of Hg. produced in one unbled and in 3 previously bled and transfused dogs a preliminary inspiratory pause followed by a rapid increase in rate and amplitude of respirations. There was a significant increase in blood pressure, as great as 90 mm, with initial increase in heart rate followed by slowing. The rise in blood pressure was maintained.

In dogs subjected to repeated hemorrhages varying from 10 to 60% of the estimated blood volume and with hypotension levels of less than 50 mm maintained for 1 hour and in others in which the arterial pressure was maintained at levels of 30 mm for 30 to 45 minutes, the responses were not uniform. In 3 instances respirations were actually depressed following the injection of the drawn blood and 0.4 cc of the solution, and the dogs died within 2 to 3 minutes with rapid fall in arterial pressure. In all cases respiration was arrested before any change in pulse or blood pressure was produced. When the blood and salt (0.4 cc) were injected immediately following cessation of respiration due to shock there was no recovery. This occurred even though artificial respiration was maintained and the heart beat was vigorous and the mean arterial pressure between 40 and 70 mm Hg. In one instance there was a slight rise in blood pressure which may have been due to asphyxia.

In 3 instances (one unbled dog and 2 bled and transfused dogs with arterial pressures of 90 mm or more), injections approximating 4 times the calculated therapeutic dose were followed by marked stimulation of respiration and rise of blood pressure which was maintained for 6 to 10 minutes, following which the arterial pressure fell abruptly, respiration became irregular and death ensued.

Summary. Although the intracisternal injection into normal dogs of therapeutic doses of potassium phosphate increases the rate and amplitude of respiration, the arterial pressure and heart rate, the response in hemorrhagic hypotension or shock was not uniform. Injection into normal dogs of larger doses resulted in death.

1 Stern, L. S., *Schweiz. Med. Wohnschr.*, 1941, **71**, 367.

2 Stern, L. S., *Lancet*, 1942, **243**, 572.

3 Stern, L. S., *Brit. Med. J.*, 1942, **2**, 538.