according to our present knowledge of such requirements. Prolonged use of potent cod liver oil failed to induce healing. In each case, the blood serum calcium values were approximately normal, the phosphorus being markedly lowered. In this respect they differ from the findings with the rickets which may be associated with renal inadequacy, in which the calcium is low, the phosphorus normal or increased. Because of absence of any demonstrable exogenous etiology, these cases have been considered as of endogenous origin. While rachitic symptoms were sufficiently marked in each patient to demand medical attention, laboratory studies indicated the presence of other metabolic disturbances in each case. Among the diverse syndromes presented by these patients are included atypical diabetes mellitus, diabetes insipidus, and extrophy of the bladder with transplantation of the ureters to the rectum. Patients studied intensively showed disturbances of the acid-base balance, of varying type and degree. In one instance the rickets became inactive after sodium bicarbonate, 1 gm. 3 times daily, was added to the patient's previously ineffective antirachitic regime. The data indicate that probably an actual or relative base deficit was present in each case.

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Effect of Intravenous Injections of Alkali on Physiological Action of Curare.

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In an earlier paper[1] it was shown that a decrease in the pH of the blood, produced prior to the administration of lethal doses of strychnine, prevented violent tetanus and death. It was considered important to try the reciprocal experiment, that is, the effect of administering alkali to dogs that had previously received a lethal dose of curare.

Dogs used in this experiment received, intravenously, 5.7 mg. of curare per kilo of body weight. The drug used was the product of Burroughs, Wellcome & Co. Eight or 10 minutes after injection, when paralysis of the muscles, except the respiratory muscles, had appeared, a 5% solution of $NaHCO_3$ was injected intravenously. Complete recovery occurred within 15 minutes after approximately

[1] Wenner, W. F., and Blanchard, E. W., PROC. SOC. EXP. BIOL. AND MED., 1928, XXV, 726.

3.0 gm. of $NaHCO_3$ had been introduced. The "curare effect" did not reappear.

At the onset of muscle paralysis the pH and CO_2 capacity begin to fall. The acidosis produced evidently augments the effect of curare but is not, in itself, sufficiently pronounced to produce coma. Introduction of $NaHCO_3$ not only corrects anoxemia but also possibly neutralizes the curare effect at the myo-neural junction.

All controls died of respiratory paralysis within 20 minutes after receiving curare.

The following table gives the data from one of our experimental animals:

TABLE I. Dog No. 5, weight 7.5 kg.

Time	Amt. Curare given	Amt. $NaHCO_3$ given	Blood pH	CO_2 Capacity Vol. %	Remarks.
10:40			7.39	42.0	
10:42	40.5 mg.				Injected intravenously.
10:50			7.27	40.2	Muscle paralysis. Dog still breathing.
10:51 to 10:53		2.5 gm.			Injected intravenously.
10:55					Reflexes returned.
11:15			7.46	47.6	Complete recovery.

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Histolytic Influence of Atrophying Gills During Metamorphosis: Special Reference to Resistance of Fore-limb Integument.

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Helff[1] has shown that the perforations in the opercular integument are the end result of an orderly process of cellular histolysis, due to the histolytic action of the atrophying gills. The present investigation was designed to determine the stage of gill atrophy at which this histolytic influence is most effective.

Homoplastic transplants of gills in various stages of atrophy were made to regions beneath the back skin of metamorphosing *Rana pipiens* tadpoles. The following constitute the main stages

[1] Helff, O. M., *J. Exp. Zool.*, 1926, xlv, 1.