a small number of cases 2 doses of antigen or even 3 failed to produce a negative test, or at least one which persisted over periods varying from 3 weeks to 3 months. An increase in reaction was noted frequently after the giving of antigen, which as a rule tended later to decrease, but in individuals who were originally slightly sensitive might be marked and persistent.

Infants very rarely gave a positive reaction and were frequently negative to as much as 20 skin test doses. One infant, 2 weeks old, whose skin reaction was negative to 50 S. T. D., developed temporarily a positive Dick test, 16 days after the last portion of 900 S. T. D. of scarlet fever toxin (so-called) had been given.

The diversity of skin reaction, as indicated by the Dick test, following antigen administration, seems to find no parallel in the phenomena of true toxin reactions and antitoxin production. Nor does the development of a positive test in an infant previously negative, in response to treatment with scarlet fever streptococcus filtrate find its explanation on any but an allergic basis. Our observations support the view that susceptibility to scarlet fever is concerned with an allergic factor; that infants, born non-sensitive, develop a sensitivity through frequent contact and close association with the streptococcus, and that the Dick test gives some indication of the degree of the sensitivity. Under sufficient stimulation the body cells may react to such an extent as to mask the allergic state. A negative Dick test may signify that an individual has not yet been sensitized, or has been sufficiently stimulated to have developed circulating antibodies, which mask the allergy.

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Effect of Acidosis in Strychnine Poisoning.

W. F. WENNER AND E. W. BLANCHARD.

From the Zoological Laboratory, State University of Iowa.

It has been demonstrated by a number of investigators that changes in the reaction of the blood toward the acid side prevent the clonic contraction and spasticity of the skeletal muscles in parathyroid tetany. The experiments reported here show that a decrease in the pH of the blood produced prior to the administration of lethal doses of strychnine, prevents violent tetanus, convulsions and death.

Acidosis was produced in 7 dogs by subcutaneous injections of

nephro-toxic substances such as uranium nitrate and sodium tartrate. When the pH of the blood dropped to about 7.2 and the CO_2 capacity to around 22 volumes %, the dogs received subcutaneously 1/160 gr. per kilo of strychnine sulphate. Ten minutes later slight muscle tremors and spasms developed. In all cases recovery occurred within 45 minutes. One dog died 1 hour following injection of the lethal dose of strychnine. In this case death was apparently due to uranium nitrate poisoning.

In other series of dogs acidosis was produced by means other than those involving impairment of renal function. This was brought about by administering NH_4Cl to one series of animals and CO_2 to another. Muscle spasms from which the dogs soon recovered, followed the injections of a lethal dose of strychnine.

Control dogs receiving the same lethal dose of strychnine died in convulsions within 1 hour.

From the results we obtained it is evident that it is the initial drop in pH occurring before injection of the strychnine and not the fall produced by convulsions and the resulting anoxemia that is effective in preventing convulsions and death from strychnine poisoning. The data from 1 of our experimental and 1 of our control dogs are presented in Table I.

TABLE I.

Animal	Date	Time	Strychnine given in gr.	pH	CO_2 capacity vol. %	Remarks
Dog 12 14 kilo	Feb. 28	10:30 A.M.		7.39	46.8	Given 100 cc. 5% solution NH_4Cl by stomach tube.
		1:30 P.M.		7.19	21.1	
		2:45 P.M.	7/80			
		2:55—				
		3:20 P.M.				Spasms and muscle twitching.
		3:23 P.M.		7.04	17.5	
		3:35 P.M.				Recovered. Remained normal.
Dog 3C 8.75 kilo	Feb. 27	3:00 P.M.		7.38	43.2	
		4:10 P.M.	1/20			
		4:20 P.M.				Violent convulsions.
		4:40 P.M.		6.92	28.3	Violent convulsions.
		5:20 P.M.				Died in convulsions.