

TABLE I.

°C.	Moina macrocopa		Pseudosida bidentata		Simocephalus, all species		Daphnia pulex (type)		Daphnia longispina		Daphnia magna		Daphnia pulex (984)	
	time in hours	Q <sub>10</sub>	time in hours	Q <sub>10</sub>	time in hours	Q <sub>10</sub>	time in hours	Q <sub>10</sub>	time in hours	Q <sub>10</sub>	time in hours	Q <sub>10</sub>	time in hours	Q <sub>10</sub>
13	296.8		517.6		381.0		309.7		284.5					
20	114.2	3.96	246.0	2.90	208.5	2.36	156.8	2.64	187.0	1.82				
25	66.9	2.39	134.3	2.03	129.1	1.81	100.8	1.70	138.0	1.48	203.1		170.3	
30	49.5	1.76	121.2	1.63	115.0	1.15	92.1		126.0		146.0	1.31	120.4	1.19
35	38.0		82.4		112.5		Will not live and reproduce.							

the species show a value of the temperature coefficient which is above 2.00; two with a value near 1.75; one with a value near 1.50; and two with a value near 1.25. The reality of these differences indicated by the temperature coefficient for development is emphasized by comparing the lethal temperatures of the adults of these same species. The lethal temperatures, determined arbitrarily by suddenly immersing the animals in water of the desired temperature for one minute, are: *M. macrocopa* 48°, *P. bidentata* 48°, *Simocephalus* species 43°, *D. pulex* typical 44°, *D. longispina* 42°, *D. magna* 41°, and *D. pulex* "984" 41°. The species are seen to fall approximately into the same groups as determined by the temperature coefficient for development of the younger animals.

<sup>1</sup> Brown, L. A., *J. Gen. Physiol.*, 1926-27, x, 111.

<sup>2</sup> Kanitz, A., *Temperatur und Lebensvorgänge*, 1915, Berlin.

<sup>3</sup> Przibram, H., *Temperatur und Temperaturen*, 1923, Leipzig und Wien.

<sup>4</sup> Banta, A. M., *Z. Indukt. Abstammungs u. Vererbungslehre*, 1925, xl, 28.

<sup>5</sup> Schrader, F., *Z. Indukt. Abstammungs u. Vererbungslehre*, 1925, xl, 1.

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### The Effect of CO<sub>2</sub> Administration Upon Parathyroid Tetany.

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It has been known for some years that certain mineral acids such as HCl, and acid producing salts such as CaCl<sub>2</sub> and NH<sub>4</sub>Cl are effective agents in the treatment of infantile tetany, and have also been successfully employed in cases of experimental tetany.

The effect of injecting HCl and CaCl<sub>2</sub> or NH<sub>4</sub>Cl is the production of an acidosis. It seems from a survey of the literature on the subject, that the gross manifestations of tetany, even the most violent convulsions, are rather promptly relieved by slight changes in the reaction of the blood toward the acid side, and that such changes influence in some manner the calcium content of the blood.

In view of these facts it was considered worthwhile to test the effect of administering CO<sub>2</sub> to dogs in tetany, (1) in relation to relief of symptoms and (2) the effect upon the serum calcium level.

Small rat and fox terriers were used as experimental animals. They were thyroparathyroidectomized and permitted to develop tetany. When the animals were prostrate they were bled and the serum Ca, CO<sub>2</sub> capacity, CO<sub>2</sub> content and pH determined. Following bleeding, the dogs were immediately placed in a specially constructed CO<sub>2</sub> chamber and sufficient gas administered over a 20 to 40 minute interval to produce unconsciousness.

The rapidity with which the muscle tremors, jerks and convulsions disappear following CO<sub>2</sub> treatment is remarkable. Within a few minutes following treatment the animal shows a complete return to norm. The length of the recovery period varies considerably. Some animals do not again exhibit tetany symptoms for 12 hours, others show tetany within a few hours. If such animals are again placed in the CO<sub>2</sub> chamber they again temporarily recover. No effort was made to keep the animals alive indefinitely, since we were primarily interested in the effects produced by the acid intoxication.

The dogs were again bled for Ca, CO<sub>2</sub> capacity, CO<sub>2</sub> content and pH after removal from the CO<sub>2</sub> chamber. The data showed no change in the serum Ca but a very marked drop in the CO<sub>2</sub> capacity, CO<sub>2</sub> content, and pH of the blood, evidence indicative of acid intoxication. It was evident from the CO<sub>2</sub> change in the blood that the acidosis was not due to the CO<sub>2</sub> *per se*, but probably to a rise in organic acid. Since the CO<sub>2</sub> treatment was really a simple method of asphyxiation it seemed probable that lactic acid was the responsible agent.

Further investigation showed the surmise to be correct. The rise in the blood lactic acid following CO<sub>2</sub> treatment is striking and consistent, and in our opinion is sufficient to account for the degree of acidosis observed. The normal lactic acid content of both normal and operated dogs in tetany is approximately 27.5 mg. per 100 cc. Following recovery from tetany after CO<sub>2</sub> treatment lactic acid values of over 100 mg. per 100 cc. were encountered. The decreased CO<sub>2</sub> capacity and CO<sub>2</sub> content of the blood of CO<sub>2</sub> treated dogs, is obviously due to the increase in lactic acid with consequent blowing

off of CO<sub>2</sub>. The data from one of our experimental dogs are given below.

TABLE I.

Oct. 30	Lactic acid mg. %	CO <sub>2</sub> content vols. %	CO <sub>2</sub> capac- ity vols. %	pH	Remarks
10:00 A. M.	28.7	33.3	43.9	7.44	Violent tetany
10:30 A. M.	56.5	22.2	27.6	7.25	CO <sub>2</sub> for 20 min Recovery
3:40 P. M.	27.9	35.0	42.7	7.45	Tetany
4:05 P. M.	88.9	29.2	32.7	7.29	CO <sub>2</sub> for 20 min Recovery

Following return of the lactic acid to a normal level, *i. e.*, (about 27.5 mg. per 100 cc. blood) tetany symptoms again appear. The evidence at hand seems to indicate that the change in the blood reaction toward the acid side relieves tetany by rendering serum Ca more diffusible, and also probably stimulates excretion of the excess phosphorus. The total serum Ca in all of our animals remained unchanged by the CO<sub>2</sub> treatment, despite the fact that the animal appeared normal and was free from tetany.

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#### Survival Period in the Pregnant and Lactating Cat Following Adrenal Extirpation.

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H. A. Stewart<sup>1</sup> first called attention to what he considered to be an unusual prolongation of the life-span of the pregnant cat following adrenal removal. He states "In most instances the pregnant and lactating animals survived the extirpation of the adrenal glands longer than normal, non-pregnant or male animals." The average prolongation in good cases of non-injected pregnant cats was 7.2 days, whereas the average prolongation of male and non-pregnant females was only 2.2 days.

Stewart and Rogoff,<sup>2</sup> working with dogs, cited cases of pregnant bitches which lived for considerable periods following bilateral adrenal removal. Several animals survived throughout the gestation period, and one animal gave birth to a litter of pups. These authors do not attempt to explain the reason for such prolongation of the survival period of adrenalectomized dogs, but merely suggest that possibly the *corpus luteum* may be involved.