recover from the effects of anesthesia, each of them received an intravenous injection of 0.05 mg. of ergotamine tartrate. In every instance these injections failed to elicit defecation. However, upon intravenous injection of 0.5 mg. of pilocarpine hydrochloride defecation followed 3, 5, and 9 minutes after administration.

Not wishing to deny the probability of peripheral purging effects of ergotamine and other ergot principles on the intestine, we are even ready to attribute all our delayed or late defecation responses to such an action. We must insist, however, that the extraordinary prompt response to small optimum doses is due to central action, as such responses are inhibited by application of ergotamine tartrate to the medulla, by administration of large doses of morphine, and by simultaneous vagotomy and high transection of the cord.

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Relative Susceptibility of Adult and Young Mice to Asphyxiation.

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It is generally stated that in carbon monoxide asphyxiation the smaller and the younger individuals, with the more active metabolism, tend to approach saturation more rapidly, and that small individuals therefore succumb sooner than large individuals. Four parts of carbon monoxide in 1000 parts of air are considered fatal to man after an exposure of less than one hour and presumably much more fatal to smaller animals such as mice.

Following the observation by one of us (R.C.A.) that the young of a family of wild mice survived while the parents died when exposed to illuminating gas, a series of experiments with 73 white mice was carried out.

The data in Table I show that, contrary to current belief, the younger survive longer. In pure concentrations of chemically inert gases (nitrogen, hydrogen and argon) the young survive exposures from 3 to 6 times as long as those which prove fatal to adults.

The lethal periods of exposure for adult and young mice, in the case of pure CO_2 and CO lie so close together that it is difficult to observe a survival of the young. This is partly due to the fact

TABLE I.

Gas	No. Adults	No. Young	Wt.	Time of Exposure	Results
			gm.	sec.	
Nitrogen	3		24-26	25- 40	Died during exposure
		3	3- 4.5	25-150	Survived
Argon	1		28	37	Died during exposure
		1	5	120	Survived
Hydrogen	3		22-28	18- 20	Died during exposure
		5	3- 5	18-135	Survived
		2	3.5-4	120	Died after exposure
CO_2	5		21-26	20- 36	Died during exposure
		5	3- 5.5	20- 33	Survived
		4	3.5-5	36- 60	Died after exposure
Illuminating Gas	2		22-23	18	Died during exposure
		3	4- 5	18- 40	Survived
CO	3		16-23	19- 22	Died during exposure
		4	3.5-6	19- 22	Died after exposure
	5		15-28	8- 15	Died after exposure
		3	2- 5	8- 12	Survived
0.4% CO	5		25-28		Died in 8, 19, 21, 42 and 60 min.
		3	5- 6		Died in 180, 225 and 255 min.
		4	1.5		Died in 270, 300, 315 and 360 min.
		4	2.5		Alive after 180 min., dead in 210 min.
	1		20	1 hr.	Survived
		4	2- 5	1 hr.	Survived

NOTE: In nearly every experiment shown in these data an adult and a young were exposed simultaneously in the same chamber.

that when adult and young animals were simultaneously exposed the gassing was continued until all muscle twitching in the animal which succumbed first had ceased. The young in such cases were still alive when exposure to the gas was discontinued but died in air within a few minutes after the termination of the experiment. When the time of exposure is sufficiently shortened the young survive indefinitely and the adults die immediately after removal from the gas.

The difference in time of survival between adult and young in illuminating gas is even more pronounced than in the case of pure CO.

In a 0.4% concentration of CO in air the time of exposure required to cause the death of the young is from 3 to almost 50 times as long as that necessary to cause the adult to die. In 0.4% CO 2-day-old mice similarly will outlive some which are 7 days old.

It was found that in no instance does a young animal die during one hour's exposure to 0.4% CO, which concentration is considered fatal to man for that period. In but one instance did an adult mouse survive for an hour under these conditions.

It developed in our experiments with the various gases, that in no instance, when young and old were exposed simultaneously, did the adult survive the young.

No determinations of the comparative blood CO pictures of these animals under conditions of these experiments have been made. It is, therefore, not possible to offer any physiological explanation of this survival phenomenon.

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Migration of Anthocyan Pigment in Plant Cells During the Flow of Electric Current, and Reversal by Acids and Alkalies.

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Visible effects of the flow of direct current through the cells of *Tradescantia* and other plants bearing anthocyan indicators have been known since the studies of Kühne.[1] They are generally accepted[2, 3, 4] as evidence of a change of acidity at the opposite ends of each cell, in accordance with the well known behavior of these pigments as indicators. A repetition of such experiments, with a variety of plants (from about a dozen families), leads the writer to a different interpretation, namely, a migration of the pigment within the cell.

The typical results with one group of plants including *Tradescantia (Zebrina)*, *Commelina*, *Rhoeo* (see also second group), *Lippia, Plantago, Sonchus*, beet and turnip, are as follows:

1. With uninjured cells, distant electrodes making contact to the tissue through agar, and low applied potential (1.5 to 6 volts in the epidermal strips as used) there is no apparent effect of current flow, the pigment remaining uniformly distributed throughout the cells and unchanged in hue.

2. With higher potentials (10 to 20 volts, probably causing some injury), or with low potentials in cells injured by chloroform or otherwise, there is a marked migration of pigment in most cells of

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[3] Mast, S. O. *Z. vergl. Physiol.*, 1931, **15**, 309.

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