

A careful examination, however, of the manner in which the velocity constant varies with pressure over the range 15–800 mm. reveals the existence of a 'segmented' plot of  $1/t_{1/2}$  against  $p_0$ . At 475° C. the 'period of half change' is almost independent of initial pressure with  $p_0 > 250$  mm. Between c. 250 mm. and 40 mm. there is a 'bimolecular' relationship between  $t_{1/2}$  and  $p_0$ , and below c. 40 mm. at least one other such 'bimolecular' region. In this respect, therefore, the reaction falls into line with other similar decompositions recently examined<sup>3</sup>, and can be interpreted as involving the superposition of several independent quasi-unimolecular processes. The energy of activation of the reaction for  $p_0 > 300$  mm. is c. 54,000 cal., and at the lowest pressures (c. 20 mm.) is somewhat lower (c. 50,000 cal.). The rate at the pressure at which the 'falling-off' is first manifest is in accordance with the postulation of a distribution of activation energy among about nine squared terms, which appears a more reasonable value than that previously calculated by Ramsperger<sup>4</sup>.

Increase in the surface-volume ratio of the reaction vessel by about eight times at 470° C. lowers the rate by less than 10 per cent, which, allowing for possible errors, is scarcely significant. The existence of appreciably long reaction chains is therefore unlikely. It has been suggested that isomerisation to acetaldehyde is the first stage in this decomposition, even though no direct evidence for such a mechanism was obtainable. We have been unable to detect the presence of acetaldehyde among the reaction products at any stage of the decomposition over the temperature range employed.

Inert gases (argon, nitrogen, helium and others) retard the decomposition to some extent, but the precise details of this effect require a fuller explanation.

The detailed results will shortly be published elsewhere.

H. W. THOMPSON.  
M. MEISSNER.

Old Chemistry Department,  
Oxford.  
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<sup>1</sup> *J. Amer. Chem. Soc.*, **51**, 2706 (1929).

<sup>2</sup> *NATURE*, **136**, 909 (1935); *J. Amer. Chem. Soc.*, **58**, 534 (1936).

<sup>3</sup> *Proc. Roy. Soc., A*, **141**, 41 (1933); *A*, **146**, 327 (1934).

<sup>4</sup> *Chem. Rev.*, **10**, 27 (1932).

### Antagonistic Effect of Iodides in Baldness and Toxicity due to Thallium Acetate

In earlier reports<sup>1,2</sup>, we stated that potassium iodide administered subcutaneously prevents the loss of hair caused by thallium acetate, as well as reduces considerably the toxicity of the latter.

The object of the present investigation was to find out whether other salts of iodine have the same effect as potassium iodide. We used seven groups of rats, each group consisting of seven rats, except the sixth thallium group which had ten rats. The experiment continued for 69 days. The first four groups (*A*) were receiving daily *per os* for a period of 32 days 0.3 mgm. and for a further 36 days 0.4 mgm. of thallium acetate per 100 gm. weight. In addition, the animals of these four groups received daily subcutaneously 0.5 c.c. of lithium iodide, potassium iodide, sodium iodide and magnesium iodide, respectively (1 c.c. of iodide solution contained 20 mgm. of iodine). The animals of the fifth group (*A*<sub>5</sub>), which were receiving daily the same quantity of thallium acetate *per os* as the previous four groups and also

0.5 c.c. of calcium iodide, very quickly developed dermatitis of the third grade due to the amount of calcium ion administered, and therefore we had to discontinue the calcium iodide. The animals of the sixth group (*B*), which served as controls to the five *A* groups, received *per os* thallium acetate only in the same quantity as the *A* groups. However, during the experiment the animals of Group *B* looked very ill, and in order to save some animals to the end of the experiment we had twice for two days to discontinue the administration of thallium acetate (six out of ten died). The animals of the seventh group (*C*), which served as controls, received no chemicals.

At the end of the experiment, all the animals of the first four groups (*A*) looked healthy, they had increased in weight on the average from 38 gm. to 135 gm. (two rats of the sodium group and one of the magnesium group died during the experiment) and preserved almost completely their coats; however, their hair was softer and lacked the lustre of normal hair. Also the two survivors of Group *A*<sub>5</sub>, which received later on potassium iodide instead of calcium iodide, had the same appearance as the animals of the other *A* groups.

The surviving animals of Group *B* lost their hair almost completely; in a few places the lanugo was seen, and also the feelers were not affected. These animals developed stomatitis, erythema and eczema and at the end of the experiment they looked very poor. The most striking effect of thallium acetate was that all the survivors of this group developed cataracts and other eye defects after 35–50 days of administration of thallium acetate, and the animals became entirely blind. This was never observed in the rats of the *A* groups. Also their average weight showed a smaller increase (from 38 gm. to 115 gm.) and was below the weights of the animals which received the iodides.

The rats of Group *C* increased in weight on the average from 36 gm. to 195 gm. and looked entirely normal.

The results of this experiment indicate that lithium iodide, potassium iodide, sodium iodide and magnesium iodide prevent to a great extent the falling out of hair, reduce considerably the toxicity of thallium acetate and prevent the development of cataracts and other eye defects brought about by thallium acetate. The best results were obtained with lithium and potassium iodide; the results with sodium and magnesium iodide were a little less satisfactory. Calcium iodide would probably have the same biological action as the other iodides used in this experiment, but due to its deleterious effect upon the skin it cannot be used subcutaneously in such experiments.

These results are not in accordance with the results of some scientific workers<sup>3</sup>, who state that potassium iodide does not act as an antidote to thallium (probably not in acute thallium poisoning); in our experiment we obtained satisfactory results with potassium and other iodides as antidotes in chronic thallium poisoning.

O. V. HYKEŠ.  
F. A. DIAKOV.

Department of General  
Biology and Parasitology,  
Veterinary College,  
Brno, Czechoslovakia.  
April 2.

<sup>1</sup> Hykeš and Diakov, *NATURE*, **136**, 685 (1935).

<sup>2</sup> Hykeš and Diakov, *Publications biol. Ecole vétérin. (Biol. spisů V. s. zvěrolék.) Brno*, **14**, 85 (1935).

<sup>3</sup> Hesse, "Handb. exper. Pharmakol.", iii, Bd. 3, T., 2177 (1934).