diately following a convulsion there may be a period of post-activity depression as in the case of the metrazol experiments.

A prolonged course of injections of insulin or metrazol in convulsive doses, (30-36 injections over 58-63 days) has not appreciably altered the course of the convulsive response from that observed in previously untreated animals.

The most conspicuous similarity between the actions of metrazol and insulin on cortical potentials is the prolonged phase of random slow wave production, which may possibly be correlated with the period of reduced cerebal blood flow[3] and coma.

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Lead Analyses of Hair as an Indication of Exposure to Lead.

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Meillère[1] believed that the hair acts as a channel of lead excretion from the body. The implications of this finding, if correct, in relation to medical and industrial problems involving serious exposure to lead are obvious. Inasmuch as Meillère made no attempt to determine the extent of contamination of his material, his thesis requires further support before it can be accepted. Previous work in this laboratory[2] has confirmed that of numerous investigators indicating that lead is extremely widespread. It is obvious, therefore, that before this question of true lead content of hair can be studied properly, it is necessary first of all to obtain a complete removal of that which is present as an external contaminant. Experiments were performed for the purpose of revealing how such contaminating lead may be removed. For example, after treatment with ether to remove lipoidal material, hair was washed in soap solution, and then in acetic and nitric acids with the idea that the lead could be removed as the soluble salts, but this technic gave unsatisfactory results. Finally, trials were made of the efficacy of treatment with diphenylthiocarbarzone in chloroform, the reagent found so useful in analysis for lead. When lead in known amounts

[1] Meillère, M. G., *Compt. rend. Soc. de biol.*, 1902, **54**, 1134.

[2] Horwitt, M. K., and Cowgill, G. R., *J. Pharm. Exp. Ther.*, 1937, **61**, 300.

was added to hair to simulate natural conditions of contamination, it was not possible by any of the above methods used to bring the concentration down to what might be regarded as normal levels, such as those characteristic of Group IA in Table II.

The problem of metabolic loss of lead through the hair was then approached by means of experiments on rats. The animals, the hair of which had been clipped, subsisted on a low-lead diet for a period of about 3 weeks, during which an amount of hair sufficient for analysis had grown back; at this time the animals were clipped again, and the hair examined for lead. The rats then received lead, in the form of the acetate, both by mouth and by subcutaneous injection, until a new crop of hair appeared, a period again of approximately 3 weeks. In taking hair for the second analyses, care was exercised to avoid the region where the injections were made. The data obtained are presented in Table I. All analyses were made by the Horwitt-Cowgill method.[3]

TABLE I.

Body wt		Amt of lead given by		Amt of lead in hair in relation to lead administration	
Initial g	Final g	Stomach Tube mg	Injection mg	Before p.p.m.*	After p.p.m.*
280	220	60	45	4	14
380	320	80	60	2	18
148	172	50	41	2	7
86	133	32	36	4	14
110	186	41	45	6	11
90	210	0	0	3	25

*parts per million.

The decrease in weights of the adult animals and the relatively poor growths exhibited by the younger rats indicate that the lead was present in toxic amounts. Although the lead content of the hair increased somewhat after lead administration, the fact that the control animal gave a figure slightly higher than any of the experimental rats constitutes ample evidence that lead is not excreted in the hair in this species. The lack of depression of the control animal and its greater activity doubtless account for its greater contamination.

As it is now evident that lead in hair cannot be regarded as that in process of excretion from the body, any found in the hair must represent external contamination. From this it follows that a high

[3] Horwitt, M. K., and Cowgill, G. R., *J. Biol. Chem.*, 1937, **119**, 553.

content of lead in the hair is sufficient indication that the individual is exposed to high concentrations of lead dust, a form of lead particularly dangerous because it can be inhaled and thus absorbed by the body.[4] To obtain some degree of confirmation of this hypothesis, the hair secured from 30 persons has been analyzed and considered in relation to probable serious exposure to lead. The first group, consisting of 22 persons, includes students, physicians, and individuals from various trades not characterized by undue exposure to lead. The 8 individuals in the second group are paint scrapers, lead smelters, and workers in the scrap metals trade.

TABLE II.

Group	No. of individuals	Lead in hair in parts per million	
		Range	Average
I A	20	10- 41	21
B	2	94-140	117
II	8	135-850	510

Two individuals with no apparent undue exposure to lead proved to have considerably greater concentrations of the metal in the hair than others in the "normal" group. They are indicated by the letter B in the table. From the values presented it might be reasoned that these 2 persons should be classified with Group II, but since they were not knowingly exposed to lead, they have been placed in a separate division. The evidence in the table indicates that exposure to lead dust increases the lead "'content" of the hair considerably. On the basis of the averages presented in the last column of Table II, this increase over the "normal" is about 25 times.

A practical industrial application of these findings would seem to be in the analysis of the hair of workers at intervals to determine not only the degree of their exposure to lead dust, but the efficiency of the devices used and measures taken to reduce the concentration of lead in the air of the plant. As was pointed out at the beginning of this communication, several experiments failed to reveal any satisfactory method by which contaminating lead may be washed from hair.

Summary. 1. The administration of lead salts to young white rats did not lead to an increased amount of the metal in the hair. Therefore the view that lead is "excreted" through the hair is not sup-

[4] Aub, J. C., Fairhall, L. T., Minot, A. S., and Reznikoff, P., *Lead Poisoning. Medicine Monographs,* Vol. VII, Baltimore, Williams and Wilkins Co., 1926. See particularly pp. 45-48.

ported by the results of this study. 2. On the basis of observations made on 30 human beings with variable exposures to lead it is concluded that the amount of lead to which an individual is exposed may be estimated with reasonable accuracy by analysis of the hair for this metal.

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Gonad-Stimulating Abilities of Male and Female Rat Pituitary Glands.*

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Functional differences in the hypophysis of male and female rats have been demonstrated through the use of gonadal transplants. Goodman[1] obtained only follicular growth in ovaries transplanted into the anterior chambers of the eyes of adult males, while corresponding implants into female rats resulted in the production of corpora lutea. Pfeiffer[2,3] confirmed and extended the experiments of Goodman in finding that ovarian grafts in the anterior eye chambers of normal males or males whose testes had been removed and reimplanted ectopically, showed only follicular growth. Evans, Simpson and Pencharz[4] found that transplants of fresh pituitary tissue from normal male rats produced a purely follicular response in the ovaries of immature females. On the other hand, Lipschütz and Reyes[5,6] reported that male hypophysis induced luteinization in 100% of the immature female rats, whereas no lutein tissue developed with the use of female hypophysis.

Since the ovarian response to pituitary implants is determined to a considerable extent by active secretion of the gland after implantation, the present report concerns the ovarian stimulation induced

* Aided in part by a grant from the Research Appropriation of the University of Oklahoma School of Medicine.

1 Goodman, LeRoy, *Anat. Rec.*, 1934, **59**, 223.

2 Pfeiffer, C. A., *Am. J. Anat.*, 1936, **58**, 195.

3 Pfeiffer, C. A., *Anat. Rec.*, 1937, **67**, 159.

4 Evans, H. M., Simpson, M., and Pencharz, R., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 1048.

5 Lipschütz, A., and Reyes, G., *Compt. rend. Soc. de biol.*, 1932, **109**, 1330.

6 Lipschütz, A., *Quart. J. Exp. Physiol.*, 1935, **25**, 109.