

used, and high concentrations of the drug were found in the liver and spleen, the data indicate that the urinary coproporphyrin output is not increased by the administration of atabrine. It may be noted that, in comparable experiments with the sulfonamides, Rimington and Hemmings⁵ were able to demonstrate a marked porphyrinuria.

Conclusion. The absorption of acidic solutions of the coproporphyrins at 4000 Å is 25 times that observed at 5480 Å, suggesting that measurements should be made at the former wavelength.

Daily oral administration of toxic doses of atabrine does not produce an increased urinary coproporphyrin output in the rat.

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Effects of Exposure to Oxygen at High Barometric Pressure on Higher Functions of the C.N.S.*

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Observations have been previously made by one of us (J. W. B.) that intermittent exposure of rats to high oxygen pressure not only causes motor dysfunction but also what appears to be a disruption of some of the higher functional activities of the C.N.S. and that these alterations persist for considerable periods after the animal's return to the normal atmosphere. This suggested that less severe exposures to increased O₂ pressure might appreciably affect certain mental processes without causing any gross manifestations in motor function. Experiments were therefore carried out to determine what effect, if any, successive short exposures to O₂ at increased pressure made over a period of several days might have on learning and memory in rats.

The pressure chamber employed was essentially that described earlier (Bean¹). A continuous circulation of O₂ and removal of CO₂ was maintained by means of a power blower and a canister of soda lime. The O₂ pressure used was 65 pounds (gauge); the temperature was held at 25°C. The experi-

mental animals selected were young adult rats; throughout their exposures to high O₂ pressure they were continuously under the observation of the operator stationed outside the chamber. Convulsive seizures were avoided as far as possible by shortness of the exposures (about 15 minutes) and decompression was carried out so as to insure freedom from the formation of oxygen emboli. Learning and memory tests were made with the Lashley Maze III (Lashley²). In the preliminary trials it was found that the O₂ exposures so affected some of the rats that they frequently failed to run the maze for several hours after their removal from the pressure chamber. For this reason an interval of one day was provided between the removal of the animals from the chamber and their subsequent test in the maze.

Learning. The facility shown in learning the maze was determined by two measures, *viz.*, the number of trials, and the number of errors, made before reaching the learning criterion. No significant difference was found in either respect between those animals which had been exposed to increased O₂ pressure and in the control animals which had been kept at normal atmospheric conditions.

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¹ Bean, J. W., *Proc. Soc. Exp. Biol. and Med.*, 1929, **26**, 832.

² Lashley, Karl Spencer, *Brain Mechanisms and Intelligence*, Chicago, University of Chicago Press, 1929.

Memory and Retention. To test for memory and retention a group of animals learned the maze and was then divided into two matched groups, experimental and control. The experimental group was subjected to increased O₂ pressure in 16 successive exposures over a period of 10 days. The control group was treated in the same way except for the exposure to oxygen. One day after the last oxygen exposure of the experimental group, both it and the control group, were tested for maze retention. It was found that the experimental group made an average of 52.9 errors in relearning the maze, whereas the control group made on average of only 2.7 errors. This difference is strikingly significant. Thus

the memory and retention of the maze previously learned was definitely impaired in the experimental group of animals as a result of their exposure to the increased O₂ pressure.

Conclusion. The data were interpreted to mean (1) that successive intermittent exposures to O₂ at high barometric pressure had no effect on the ability to learn a maze habit; (2) that the retention and memory of a maze previously mastered was adversely affected to a striking degree by intermittent exposures to increased O₂ pressure; (3) that certain mental processes may suffer deleterious effects as a result of exposure to increased O₂ pressure even when such exposure causes no evident motor dysfunction.

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Similarity of Response of Thymus and Lymph Nodes to Administration of Adrenocorticotrophic Hormone in the Rat.*

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Enlargement of the thymus and lymphoid tissues has been noted experimentally and clinically¹ as the result of adrenalectomy or hypofunction of the adrenal cortex. On the other hand, adrenal cortical extracts² and the administration of adrenocorticotrophic hormone³ have been shown to cause thymic involution. Since it has been shown experimentally⁴ that lymph nodes respond to adrenalectomy in the rat in the same manner as does the thymus, it is of interest to note that adminis-

tration of purified adrenocorticotrophic hormone causes a similar involutionary change in lymph nodes as well as thymus.

Methods. The reaction of thymus and lymph nodes to purified sheep adrenocorticotrophic hormone (ACT)⁵ was established in the following experiments. (1) Action of ACT in the normal rat. (2) Action of ACT in the hypophysectomized rat. (3) Action of ACT in the adrenalectomized rat maintained in good condition on 1% NaCl. All animals employed were 26-day-old male rats at onset of the experiment. The treated animals were given a total dose, daily, of 1 mg ACT by intraperitoneal injection in 3 injections distributed through the 8-hour-day (2 injections on Saturday, 1 injection on Sunday). Injections were begun on the day of operation. All animals were maintained under identical nutritional and environmental con-

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¹ Moon, V. H., *Shock*, Lea and Febiger, Philadelphia, 1942, 198-203.

² Ingle, D. J., Higgins, G. M., and Kendall, E. C., *Anat. Rec.*, 1938, 71, 363.

³ Evans, H. M., Moon, H. D., Simpson, M. E., and Lyons, W. R., *Proc. Soc. Exp. Biol. and Med.*, 1938, 38, 419.

⁴ Reinhardt, W. O., and Holmes, R. O., *Ibid.*, 1940, 45, 267.

⁵ Li, C. H., Simpson, M. E., and Evans, H. M., *Science*, 1942, 96, 450.