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The Sensitizing Dose in Respiratory Anaphylaxis (Asthma)*

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In our early studies on respiratory anaphylaxis[1] it was shown that guinea pigs could be sensitized by the inhalation of a dry antigenic dust. The animals were placed in a properly ventilated glass cage on a false wire floor. Dry horse dander was spread on the true floor of the cage. A nozzle, inserted through a hole near the bottom, was attached to a pump which blew in intermittent puffs of air, throwing the dust into a fine cloud. The air in the cage was kept moderately filled with the dust. The animals were exposed for 1 to 2 hours daily over a period of 6 to 12 days.

In our initial experiments[1] we were primarily interested in demonstrating that animals could be sensitized with a dry dust in a manner simulating the natural contact of a human being. Having demonstrated this, we desired to determine how long a period of exposure was necessary to sensitize animals by this route.

Two series of experiments were carried out in which the animals (300 to 400 gm.) were exposed in the above manner for varying intervals of time. Hypersensitiveness was proven by an intravenous injection (0.3 to 0.5 cc.) of horse dander extract after an incubation period of about 3 weeks.

In our first series we had 22 animals. After intravenous injection, of the 4 that were exposed for 1 hour, 2 were negative, 1 presented marked dyspnea with recovery and 1 presented marked dyspnea, suffusion of the eyes, collapse and recovery; the 5 exposed for 2 hours were all negative; the 3 exposed for 3 hours were all negative; of the 5 exposed for 4 hours, 1 was negative, 1 showed suggestive symptoms of anaphylaxis and 3 died in typical anaphylactic shock; and of 5 exposed for 5 hours, 4 died in typical anaphylactic shock and 1 showed marked anaphylaxis, convulsive movements, dyspnea and collapse with recovery.

With this group therefore it was only those animals which received 4 to 5 hours of contact with the dust that were sufficiently sensitized to die in anaphylactic shock, although we have an indica-

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[1] Ratner, B., Jackson, H. C., and Gruehl, H. L., PROC. SOC. EXP. BIOL. AND MED., 1925, xxiii, 17; *Am. J. Dis. Child.*, 1927, xxxiv, 23.

tion that even after 1 hour's exposure there were moderate signs of sensitization.

In view of the evidence that animals might become sensitized after 1 hour's exposure, we carried out another series of experiments on 37 animals. After intravenous injection, of 4 animals exposed for ¼ hour, 1 showed suggestive symptoms, 3 were negative; of 7 exposed for ½ hour, 4 showed moderate dyspnea, and 3 were negative; 5 exposed for 1 hour all died a typical anaphylactic death; of 4 exposed for 2 hours, 1 was negative and 3 died in typical anaphylaxis; of 9 animals exposed for 3 hours, 7 died in typical anaphylactic shock, 1 showed dyspnea, suffusion of the eyes, moderate collapse with recovery and 1 was negative; of 5 animals exposed for 4 hours, 4 died and 1 showed moderate anaphylaxis with recovery; all 3 animals exposed for 5 hours died in typical anaphylactic shock.

Therefore a suggestion of sensitiveness may be brought about by the inhalation method in as short an exposure as ¼ hour. Definite sensitization may be established by 1 hour's exposure in certain animals. Other animals apparently cannot be sensitized at all.

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Effect of Hydroquinone in Vitamin A Deficiency.

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Since the discovery about 1913 that a lack of vitamin A in the diet would cause ophthalmia in rats, attempts have been made to show that this pathological condition might also be due to other factors than the absence of this vitamin. The so-called "salt ophthalmia" of McCullom, Simmonds and Becker[1, 2] has been shown by McCullom, Simmonds and Becker[3] and Jones[4] to be due to an oxidative destruction of vitamin A in the diet by ferrous sulfate if the

[1] McCullom, E. V., Simmonds, N., and Becker, J. E., *J. Biol. Chem.*, 1922, liii, 313.

[2] McCullom, E. V., Simmonds, N., and Becker, J. E., *J. Biol. Chem.*, 1925, lxiv, 161.

[3] McCullom, E. V., Simmonds, N., and Becker, J. E., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 952.

[4] Jones, J. H., *J. Biol. Chem.*, 1927, lxxv, 139.