hepatic sugar. At the end of the experiment this liver was found to contain only 6.10 mg. of glycogen.

These results seem to point to the conclusion that the effect of thyroxin upon the carbohydrate of the liver is probably an indirect one. The manner in which this influence is brought about is not yet clear.

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Respiratory Anaphylaxis in Guinea Pig Due to Castor Bean Dust.*

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Last year we[1] demonstrated for the first time that normal guinea pigs, when exposed to an organic dust such as horse dander, could become sensitized through inhalation. Guinea pigs, thus sensitized, when again placed in contact with the same dust after a suitable incubation period, demonstrated unmistakable signs of anaphylaxis which we termed "respiratory anaphylaxis" and which we believed to be identical with bronchial asthma in the human being. This was offered as a method for studying asthma experimentally in the animal.

Recently, Figley and Elrod[2] reported the occurrence of a large number of cases of asthma resulting from the inhalation of castor bean dust "pomace" thrown into the air from the pipes of a castor oil factory. To determine whether pomace was an anaphylactogen and could be the cause of asthma, we proceeded to study the problem of pomace asthma in the guinea pig. The pomace came from the above castor oil factory.

Thirty-seven normal guinea pigs were placed for several hours in glass cages in which pomace was kept in circulation by currents of air. After an incubation period of about 3 weeks, 2 of these animals were given an intravenous injection of 0.5 cc. of a crystal clear alkaline extract of this pomace. Both animals showed definite anaphylaxis, one dying within a few minutes and demonstrating

* This work is being carried on under "The Crane Research Fund for the Study of Allergic Diseases in Children."

[1] Ratner, B., Jackson, H. C., and Gruehl, H. L., *Am. J. Dis. Child.*, 1927, xxxiv, 23.

[2] Figley, Karl D., and Elrod, Robert H., *J. Am. Med. Assn.*, 1928, xc, 79.

typical anaphylactic lungs, and the other going into collapse with marked dyspnea and finally recovering. These 2 animals established the fact that sensitization could be accomplished by inhalation of pomace.

Of the remaining 35 animals, 17 died 6 to 7 days after the sensitizing inhalation of pomace, with definite evidences of ricin poisoning. However, besides the classical abdominal pathology of ricin poisoning, we noted in many of the animals a severe hemorrhagic condition of the lungs. We first attributed this lung condition to the entrance of ricin through the nasal passages but we obtained similar results later when the pomace extract was injected.

After an incubation period of 2 to 3 weeks, the remaining 18 animals were again placed in the cage to determine whether we could demonstrate the symptoms of respiratory anaphylaxis (bronchial asthma). Unmistakable signs of respiratory anaphylaxis were shown by these animals. Later, however, 10 of the 18 died from ricin poisoning with the pathological findings noted above. The remaining 8 were injected intravenously with pomace extract and showed typical symptoms of anaphylaxis.

In testing the toxicity of the pomace extract used, we readily discovered that the normal animal died from typical ricin poisoning in about 12 hours after an intravenous injection of the extract. It was interesting, however, to note that the primary toxicity was never manifested immediately after injection and never in the same manner in which anaphylaxis was manifested. Further control tests with ricin gave results identical with those obtained with pomace.

From the 8 cases noted above which showed typical anaphylaxis on injection of pomace extract, a definite immunity to ricin poisoning was demonstrated. In 3 of these animals there was no evidence of ricin poisoning even after prolonged contact with the pomace.

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Effects of Experimental Block of the Amphibian Nervous System.

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Experiments previously reported[1] have shown that the cut ends of the spinal cord can be prevented from reuniting by the removal

[1] Nicholas, J. S., *Anat. Rec.*, 1928, xxxviii.