chloride similar to the one administered to the surviving animal (*B*). This means that the magnesium antagonizes the fatal effect of barium. We are not ready to state definitely in what way this action of magnesium is exerted. The poisonous effect of barium is due to its action upon various functions and magnesium antagonizes some of them. We are engaged in the study of the particulars of the subject.

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A demonstration of the cause of acute anaphylactic death in guinea pigs.

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In a preliminary communication,[1] we pointed out among other things, (1) that the acute anaphylactic death of guinea pigs was due to asphyxia; (2) that this asphyxia was caused by the development of a stenosis in the pulmonary air passages, so that practically no air enters or leaves the lung in spite of violent diaphragmatic contractions, the lungs remaining distended even after opening of the chest; (3) that this stenosis was caused by a *peripheral* action of the second or toxic injection, for the same stenosis and striking lung picture was obtained after destruction of the cord and medulla, artificial respiration being maintained; (4) that this stenosis was probably caused by a tetanic contraction of the muscles of the bronchioli.

In the demonstration, a sensitized guinea pig was immobilized by curarin and artificial respiration instituted. This respiration was of such a strength that the chest expanded and collapsed well. Within from one to two minutes after the injection of one cubic centimeter of normal horse serum, the respiratory oscillations of the chest gradually became less and less and finally stopped, although the respiration machine delivered the air with the same rate, strength and amount as before; now the chest remained motionless in an inspiratory condition, thus demonstrating that the

[1] Auer and Lewis, *Jour. of the American Med. Assoc.*, 1909, liii, 458.

entering air found an obstruction or stenosis shortly after the injection of the toxic dose.

Autopsy showed the typical picture of the lungs: trachea is clear, lungs are well distended, almost forming a cast of the thoracic cavity; there is failure to collapse upon opening the chest and excising the lungs; pieces cut off from the lung do not collapse, but show on pressure a good amount of air and practically no fluid; blood in lungs and heart is black.

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Anaphylactic "shock" in the dog.[1]

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The observation concerning the blood pressure here offered is not original in that the condition of low blood pressure in anaphylactic shock has previously been described by Biedl and Kraus.[2] The phenomena of anaphylactic "shock" in the dog are, however, so different from anaphylactic death in the guinea pig that it seemed to Drs. Auer and Lewis[3] and ourselves desirable, that our work, though as yet incomplete, should be presented at this time. In the dog the chief disturbance which can be demonstrated by physiological methods is a sharp fall in blood pressure (50 to 70 mm. Hg) which continues for hours, resembling in this respect shock due to other conditions. This is unaccompanied by disturbance in heart rate or by respiratory disturbance, other than that due to the medullary anemia consequent upon the low arterial pressure. From this condition the dog eventually recovers. Death has not been observed in our experiments and Biedl and Kraus state that the animals recover. The recovery from the low level of pressure is very slow, frequently no change being observed in half an hour; at other times the upward trend begins in less time.

[1] Aided by a grant from the Rockefeller Institute for Medical Research.

[2] Biedl, A., and Kraus, R., Experimentelle Studien über Anaphylaxie, *Wiener klin. Woch.*, 1909, xxii, 363.

[3] Auer, J., and Lewis, P. A., Acute Anaphylactic Death in Guinea Pigs. Its Cause and Possible Prevention; a Preliminary Note. *Jour. of the American Med. Assn.*, 1909, liii, 458.