

diately reestablished, and it was possible to extirpate leisurely the anterior wall of the part of the aorta that had been tubed and to substitute for it a segment of vena cava preserved in cold storage. This operation lasted twenty-four minutes. The tube was then taken out. The animal recovered without incident. He died suddenly of hemorrhage twelve days after the operation. The accident was due to a fault of technique in preserving the veins in cold storage.

These experiments show that operations on the thoracic aorta need not be very dangerous, and that, by Meltzer's method, they are as simple as abdominal operations.

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**The mutual antagonistic life-saving action of barium and magnesium. A demonstration.**

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For rabbits, 1.2 grams of magnesium sulphate per kilo body-weight are invariably fatal in intramuscular injection; they usually die in less than twenty minutes. The rabbit to the right (*A*) received such a dose and has been dead for some time. The rabbit in the middle (*B*) received a similar dose of magnesium and is still alive; it breathes regularly. This animal received also an intravenous injection of barium chloride, which is the cause of its surviving the fatal dose of magnesium.

By a special study we are enabled to state the mode of the antagonistic action of the barium which is this: the fatal action of magnesium is due to a paralysis of respiration and barium counteracts just this effect of magnesium. It differs from the antagonistic action of calcium inasmuch as calcium antagonizes all the effects of magnesium, while barium picks out only the respiration, the animal remaining anesthetized and paralyzed.

This surviving rabbit (*B*) illustrates, however, also another result. The rabbit to the left (*C*) is dead from a dose of barium

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,<sup>1</sup> 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

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<sup>1</sup> Auer and Lewis, *Jour. of the American Med. Assoc.*, 1909, liii, 458.