

anti-bodies from the serum. If these are supplied, an animal passes directly into the anaphylactic condition again, and this reversal could conceivably be repeatedly renewed.

CONCLUSIONS.

1. Guinea-pigs which have been rendered actively anaphylactic by a preliminary injection of foreign proteid (horse serum, egg albumen), and have then been made anti-anaphylactic by the injection of a sub-lethal dose of this proteid, may be immediately re-sensitized by the introduction of serum from a pig sensitized, or one immunized to the same proteid. Death is then produced, with typical anaphylactic symptoms by the intravenous injection of an amount of this proteid which to normal pigs is non-toxic.

2. Actively anti-anaphylactic pigs may be re-sensitized with serum derived from a rabbit immunized against the same proteid.

3. Passively sensitized pigs, rendered anti-anaphylactic by the usual methods, can be re-sensitized by the re-introduction of an immune serum.

4. A pig simultaneously sensitized to two different foreign proteids, may be rendered anti-anaphylactic to one of these by the usual methods, while its sensitiveness to the other remains unimpaired. This demonstrates that anti-anaphylaxis cannot be due to exhaustion of complement, since complement is amply present.

5. The introduction of normal guinea-pig's serum into an anti-anaphylactic animal fails to re-sensitize. Some factor other than complement, therefore, must be introduced in re-sensitization.

6. The absence of available anaphylactic antibodies appears to be the cause of the refractory condition known as anti-anaphylaxis, and re-sensitization is due to their re-introduction.

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The toxicity of foreign leucocytes.

By **WILFRED H. MANWARING.**

[From the Rockefeller Institute for Medical Research.]

The injection of from 0.7 c.c. to 1.0 c.c. of rabbit leucocytes into the cerebral meninges of dogs is apparently invariably fatal.

The animals show no immediate symptoms; but about two hours later there is beginning incoördination, rigidity and respiratory distress, increasing to collapse about the fourth hour, and death about the sixth. An occasional dog survives till the twenty-fourth hour. Autopsy usually shows extensive local hemorrhagic, inflammatory and necrotic changes.

Horse leucocytes similarly injected produce symptoms of the same general nature, though less severe. About two thirds of the dogs injected with horse leucocytes recover. A reinjection of these dogs, however, or the injection of dogs whose meninges have been previously injured by tubercle bacilli, is almost invariably fatal.

The injection of 0.5 c.c. of rabbit leucocytes into the spinal meninges of monkeys produces slight symptoms from which most of the monkeys recover. Larger amounts are usually fatal.

Horse leucocytes are less toxic for monkeys, producing few if any symptoms, even when injected in 1.0 c.c. doses. The toxicity of both leucocytes however increases on repeated injection, the third injection often being fatal. Autopsy in such cases often shows edema of the lungs as the apparent immediate cause of death.

These tests have a bearing on the possible therapeutic uses of leucocytes in meningeal infections.

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The occurrence, and the significance, of tyrosinase in the reproductive organs of certain amphibians.

By **ROSS AIKEN GORTNER.**

[*From the Laboratory of Biological Chemistry of the Station for Experimental Evolution, The Carnegie Institution of Washington.*]

Tyrosinase—the enzyme which oxidizes tyrosin to produce a black, insoluble, pigment-like compound—has been shown by Phisalix (*C. R. Soc. Biol.*, 50, p. 793, 1898) to occur in the skin of the European frog, *Rana esculenta*. Gessard, later (*ibid.*, 56, p. 285, 1904), shows that the same enzyme occurs in the skin of the toad, *Bufo vulgaris*, and in the frog, *Rana temporaria*. In discussing the