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It is difficult to resist the conclusion, in the face of these results coupled with those of Bacharach and Zacho, that vitamin P deficiency can exist, at least so far as guinea pigs are concerned, and that citrin contains a substance or substances (vitamin P), the effect of which is to return the capillary resistance to normal.

The pressures given were recorded on a spring manometer corrected against a mercury manometer.

I am indebted to Messrs. Roche Products for the supply of citrin and vitamin C used in these experiments.

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## Human Poliomyelitis in the Developing Chick Embryo

SEVERAL attempts by different authors<sup>1</sup> to propagate human poliomyelitis virus in the developing chick embryo have been entirely negative. In these experiments the technique employed was that elaborated by Burnet, the eggs being inoculated on the 10-12th day of incubation. Recently, however, I reported<sup>2</sup> successful transfer to, and serial passage n, chick embryos of mouse poliomyelitis virus (Theiler's virus). In conformity with the observation by Kligler and Bernkopf<sup>3</sup> on rabies virus, it was found that only young embryos-5-7 days oldwere susceptible to infection. The virus was restricted to the central nervous system, and no lesions were detectable.

Although the conditions were most unfavourable for the continuation and extension of these experiments, in that the hatching eggs obtainable were of low quality, the mortality rate of the embryos being very high, and the supply of monkeys was depleted and could not be restored, it was considered worth while to try the same technique on human poliomyelitis virus. The result of the first attempt was promising, although not entirely convincing. As the experiments had to be discontinued for the time being, the present observation will, however, be reported.

The strain of virus used was the L strain, adapted to monkeys and of a uniformly high virulence. A 10 per cent suspension of glycerolated monkey cord was inoculated in 0.05 ml. amounts in four eggs on the sixth day of incubation. One embryo died on the day after inoculation; the remaining ones were all alive and showed normal development at the termination of the experiment. On the 10th day after inoculation the eggs were opened and the brains of the embryos harvested. 0.5 ml. of a pooled 10 per cent brain suspension was inoculated intracerebrally

in a Rhesus monkey, an old animal with traces of a previous cage paralysis. Rectal temperature readings were taken daily for four weeks. With the exception of a slight fever during the first five days after inoculation, nothing abnormal was observed. The temperature of this animal was rather low, not exceeding 37.7°C. The result was considered negative. On the 70th day after inoculation, however, the animal displayed certain disturbances, irritability and slight tremor, and its fur was ruffled. On the following day the temperature was  $38 \cdot 4^{\circ}$  C. and rose to 39.9° the day after. On the 73rd day the temperature fell to  $36.4^{\circ}$  in the evening and was  $35.8^{\circ}$  on the morning of the 74th day. Paresis of the legs became evident on the 71st day. On the 74th day a quadri-plegia had developed. The animal was sacrificed. No gross lesions were observed at autopsy. Microscopic examination revealed typical lesions in the pons region, medulla oblongata, and all levels of the spinal cord, consisting of perivascular cuffing, scattered infiltration in the grey matter, and neuronophagia.

No other diagnosis than poliomyelitis presents itself. With the exception of this experiment, no work on human poliomyelitis had been carried out in the laboratory for more than six months previously. Accidental infection of the animal must, therefore, be considered most unlikely. In these circumstances the experiment gives suggestive evidence that human poliomyelitis virus can be propagated in the central nervous system of young chick embryos. The unusually long incubation period in back passage to monkey indicates that the concentration of virus remains on a low level.

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Products Formed in Pyrethrin Concentrates during Storage

In the course of work on the structure of the pyrethrins, concentrates rich in pyrethrins I and II were stored in the dark for some months. It was then found that the concentrates were no longer completely soluble in petroleum ether and, surprisingly, that the insoluble residues showed a high 'apparent' pyrethrin content when assayed both by the Wilcoxon-Holaday and Seil methods<sup>1</sup>, in spite of their low toxicity to house flies in comparison with freshly prepared pyrethrum extracts at equivalent concentrations. Thus two typical residues showed (Sample A) pyrethrin I,  $13 \cdot 5$  per cent w/w, pyrethrin II, 64.7 per cent w/w (Wilcoxon-Holaday), and pyrethrin I, 15.1 per cent, pyrethrin II, 65.1 per cent (Seil), and (Sample B) pyrethrin I, 43.1 per cent, pyrethrin II, 7.5 per cent (Wilcoxon-Holaday), and pyrethrin I, 47.5 per cent, pyrethrin II, 7.2 per cent (Seil), yet 1 per cent solutions of these residues in acetone were less effective against house flies under exactly comparable conditions than a 0.1 per cent