original condition. It is questionable whether the real primary lesion in such cases is of such character as to be ever demonstrable by physical methods.

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Proteid poisons.

By **VICTOR C. VAUGHAN.**

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We have been able by diverse methods to split proteids, bacterial, vegetable and animal, into poisonous and non-poisonous products. The purpose of this abstract is to state briefly some of the properties of the poisons obtained by the cleavage of proteids.

The poisons obtained from the different proteids are similar but are not identical. All are soluble in both water and absolute alcohol, more freely in the latter than in the former. The aqueous solutions are acid and slowly decompose sodium bicarbonate, forming salts apparently, and these are less poisonous than the free acids. The aqueous solutions give the general color reactions for proteids with the exception of that of Molisch, and some of them give this reaction. However, most of the proteid poisons obtained by cleavage of the proteid molecule contain no carbohydrate and are free from phosphorus.

These poisons when injected into animals intra-abdominally, subcutaneously or intravenously induce characteristic symptoms and when administered in sufficient quantity kill promptly. There is a first stage which may be designated as that of peripheral irritation, which is characterized by restlessness and scratching. In the second stage there is partial paralysis, most marked in the posterior extremities; the third stage is characterized by more or less violent clonic convulsions and in the great majority of instances these terminate in death within half an hour after administration. Animals may show the first and second stages and still recover, but in the great majority the appearance of the convulsive stage indicates a fatal termination. As a rule death or recovery results within one hour and the former may occur within five minutes and,

with an intravenous injection, the time may be even shorter than this. The fatal dose may vary from eight to one hundred milligrams according to the purity of the poison or the mode of administration. While the fatal dose may be small the range between that necessary to induce the first and second stages and that necessary to kill may be wide. With one preparation seventy milligrams was required to kill, but five milligrams developed the first and second stages in pronounced forms.

Death is due to failure of respiration and the heart often continues to beat for some minutes after respiration has ceased. It seems most probable that death is due to the direct action of the poisons on the respiratory center. It is inferred from the readiness with which recovery may follow non-fatal doses that the poison cripples but does not destroy the cells of the respiratory center.

All attempts to produce antitoxins with these proteid poisons have, so far, failed. It is true that repeated treatments of animals with non-fatal doses of the poisons from the colon and typhoid bacilli enable animals to bear from two to four times the ordinarily fatal doses of living cultures of these bacteria, but this seems to be due to an increased resistance rather than to a true immunity. This condition is not specific and may be induced by the poisons obtained from peptone or egg white, as well as with that obtained by cleavage of the homologous bacterium.

Attempts have been made to ascertain the chemical constitution of the proteid poisons by splitting them up with mineral acids but at present these experiments have not yielded satisfactory knowledge and work along this line is being continued. The physiologic action of the proteid poisons leads to the suspicion that they contain a neurin group, but so far we have not been able to demonstrate the presence of such a body.

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Observations on the living developing nerve fiber.

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The immediate object of the following experiments was to obtain a method by which the end of a growing nerve could be