

of the filtrate was markedly diminished (as compared with weakly reacting normal individuals) or was negative. During the early period of the disease the reaction might be distinct, in the second or third week the reaction was usually diminished or negative. After convalescence the reaction began again to become positive. The ability of the organism to neutralize the local action of the typhoid filtrate was apparently evanescent, and confined to the actual period of the disease.

At the present time we prefer not to discuss the many possible theories which are suggested by these results, but simply to call attention to the fact that this reaction is apparently not similar or identical with the skin reactions to the so called true toxins, such as diphtheria toxin or scarlet fever toxin.

This is a preliminary report.

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Observations on Fat Embolism.

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Fat embolism, occurring as the result of trauma to fat depots, can be demonstrated clinically and experimentally. Trauma is usually considered the sole cause. However, fat embolism has been found in many non-traumatic conditions. Dogs will survive aseptic injections of cotton-seed oil in amounts per kilo body weight that are twice the amount of fat per kilo body weight in the marrow of the human femur.

It is obvious, therefore, that there must be some other etiology than trauma for some cases of fat embolism. The most logical other source for fat embolism is, of course, the normal plasma emulsion of fat.

Experiments are reported showing that certain artificial emulsions can be broken down by the addition of necrotic muscle extract, pepsin and histamine, besides other substances. This breakdown varies in speed and degree inversely with the fineness of the emulsion. It has not been demonstrated in the physiological plasma emulsion of a digestive lipemia.

However, the well-known destruction of this emulsion by fat solvents was applied by the intravenous and mask administration of

ether to dogs in the lipemic phase following a fat meal. Ninety-five per cent of animals so treated showed definite fat embolism in the lungs, as compared to 21 per cent of controls which were killed by many different procedures.

It is emphasized that no significance has heretofore been placed on the physical state of the normal fat in the blood-stream. This is the first experimental production of fat embolism by other than traumatic means. The possible relationship of clinical fat embolism to the presence of protein split products in the blood-stream is suggested by the breaking down of artificial emulsions by these substances.

This is a preliminary report.

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Action of Aldehydes on Histamine and on Active, Histamine-like Substances Produced by Gas-Bacillus.

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It was shown in a preceding paper¹ that the contraction induced by histamine, or the histamine-like substance produced by the gas bacillus, in a section of guinea pig intestine, or uterus suspended in Tyrode solution, could be relaxed immediately by the addition of 0.1 cc. neutral formalin solution. The most plausible explanation for this phenomenon is a definite chemical reaction between the amino group of the histamine molecule and the aldehyde according to the equation

$$R.CH_2 CH_2 NH_2 + H.CHO = R.CH_2 CH_2 N : CH_2 + H_2O$$

whereby the ethyl amine group is removed.

It follows that other aldehydes might react in a similar manner, if this explanation be correct.

A series of aldehydes have consequently been tested, using the technique described in the preceding contribution.¹ These comprised, besides formaldehyde, already mentioned, acetaldehyde and its polymer paraldehyde, aldol, glycol aldehyde, glyceric aldehyde and methyl glyoxal, the ketones, acetone and dihydroxyacetone, and acetophenone.