ready had occurred. In about 60 per cent of the experiments formaldehyde caused relaxation of the contracted strip.

Experiments done with horse serum and horse dander extract showed similar results.

Occasionally smooth muscle was found irritable and spontaneous contractions occurred. Formaldehyde added to these strips had no appreciable effect.

From these experiments it is evident that formaldehyde may prevent and also relax smooth muscle contraction induced in anaphylaxis. Moreover, sensitized muscle exposed to formaldehyde and then failing to react to homologous antigen, will not react subsequently to such antigen, although contractility is not impaired. This effect simulated desensitization.

This is a preliminary report.

[1] Kendall, Arthur I., PROC. SOC. EXP. BIOL. AND MED., 1927, xxiv, 316.

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Formation of Methylglyoxal From Hexose Phosphate by Tissues.

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Methyl glyoxal has long been hypothecated as a probable intermediate in the formation of lactic acid in tissues, for which hypothesis the existence of glyoxalase activity is strong but indirect evidence.

Until very recently methyl glyoxal has never been detected in tissues. Toenniessen and Fisher[1] now report that hexose phosphate with muscle in the presence of pancreas (anti-glyoxalase) forms methyl glyoxal which they were able to identify by the p-nitrophenyl-di-hydrazone. In attempting to repeat this work I find that if muscle, or, better yet, liver extract when incubated with toluol for 24 hours, then hexose phosphate added and the mixture incubated for another day, the solution contains a substance which reacts like methyl glyoxal. It may be determined by the colorimetric method, is destroyed by excess alkali as is methyl glyoxal, and it gives a red di-hydrozone with p-nitro-phenyl-hydrazine which after recrystallization from pyridin melts with decomposition at about 283° (uncorrected). The di-hydrazone made from methyl glyoxal has the same crystal form and melting point. This evidence con-

firms the observation of Toeniessen and Fisher and indicates that the substance is actually methyl glyoxal; but conclusion is withheld until a larger quantity of the substance is prepared and submitted for further analysis. The importance of this observation makes very desirable a detailed investigation of the reactions involved. Hexose phosphate alone, liver (or muscle) extract alone, or heated extract plus hexose phosphate does not yield the glyoxal-like substance.

The formation of methylglyoxal under the conditions stated perhaps indicates that the conversion of hexose phosphate to lactic acid takes place in steps, hexose phosphate → methyl glyoxal → lactic acid. The glyoxalase, responsible for the second step is largely destroyed during the first day of autolysis, while the ferment presumably responsible for the first step (phosphatase) is apparently still active.

[1] Toenniessen and Fischer, *Z. f. physiol. Chem.*, 1926, clxi, 254.

3623

Protective Mechanism Against Acoustic Insult in the Mouse.

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Practically all experimental animals except the small rodents show evidence of cochlear degeneration when exposed to prolonged acoustic insult. Experimental degenerations of the cochlea have been employed as a method of determining specific pitch areas in the organ of Corti. There is, however, a difference in opinion on the causal factor of the lesion. The majority hold it is due to an excessive stimulation dependent on the pitch and intensity and is, therefore, a criterion of a specific area of reception. The minority holds the degeneration does not appear until the intrinsic muscles have been fatigued and that it may represent a result of clatter in the ossicular apparatus. This would mean the lesion is not dependent on the pitch. If the mouse really escapes intact under conditions which cause lesions in other experimental animals, then an additional protective mechanism in the middle ear must be sought. Small rodents show a rather remarkable departure from the usual middle ear picture. The stapes is traversed by a large artery which must certainly act as an efficient and tireless damping mechanism.