A few of these penetrated the muscle and attained a diameter of 1.5 cm. However, all of them healed in 2 or 3 weeks without secondary infection. Seven of the treated animals showed varying grades of fatty degeneration of the liver, while this condition was found in only one control guinea pig. However, because of the small numbers involved, we do not wish to draw conclusions from this latter observation.

Discussion. From the evidence presented above, we think it reasonable to conclude that promin exerts a retarding effect on the course of experimental tuberculosis in guinea pigs. Under the conditions of our experiments, this beneficial effect is manifested by a retardation of the spread and multiplication of tubercle bacilli in the animals. While these results are far superior to those which we have obtained with any other therapeutic agent in experimental tuberculosis, they fall far short of the wonderful effects reported by Feldman and his coworkers. These differences may be explained in part by variations in experimental procedure in such external and internal factors as route of administration of the drug, organism used, and breed of guinea pig. However, an adequate chemotherapeutic agent for humans should be able to cope with bacteria of wide differences in virulence, and should be of help in individuals of various constitutional makeups and from a wide variety of environments. Nevertheless the results reported, in our opinion, indicate that promin should have an exhaustive and prolonged study in the treatment of human tuberculosis.

Summary and Conclusions. 1. Promin exerts a bacteriostatic effect on tubercle bacilli *in vitro*. 2. Promin exerts a retarding effect on guinea pig tuberculosis, but does not cure the disease. 3. The effects of promin on human tuberculosis are being investigated.

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Complementing Action of Eserine and Acid in Neurohumoral Activation.

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Since eserine permits a greater accumulation of acetylcholine at junctional points of activated ganglion cells, muscle fibers and other

end organs, and granting the same for increased acidity, it is to be anticipated that eserine and carbon dioxide would complement each other in their effects upon the body.[1-6] This preconceived idea was put to a simple test in anesthetized dogs connected with rebreathing tanks for alternate administration of room air and carbon dioxide mixtures.

The cardio-inhibitory reflex initiated by faradic stimulation of the superior laryngeal nerve served as the physiological indicator. The superior laryngeal nerve was stimulated for a period of 5 seconds at intervals of 2 minutes with faradic shocks of uniform intensity.

It was demonstrated, by the administration of a 10% mixture of carbon dioxide in 40% oxygen, that hypercapnia increased the reflexogenic inhibition of the heart. Intravenous injection of eserine superimposed upon a continuing hypercapnia increased and prolonged the reflexogenic cardio-inhibition still more. Return to room air while the effects of eserine were still evident produced an abrupt diminution in the degree of cardio-inhibition. These results permit 4 conclusions. 1. Hypercapnia potentiates the cardio-inhibitory reflex initiated by stimulation of the superior laryngeal nerve. 2. Eserine produces a comparable potentiation. 3. The potentiation produced by eserine is addible to that of hypercapnia. 4. The potentiating action of hypercapnia is subtractible from that of eserine.

Because the frequency of the heart beat is probably the end effect of the influences of many junctional stations, parasympathetic, sympathetic and central, a quantitative allocation of the acid-neurohumoral effects at the several stations is at present impossible. Nor are the effects of acid to be limited to its preservative action on physiologically deposited acetylcholine alone. There are at least two other possibilities, which we should like to suggest in a tentative way. One is the increased elimination of potassium during asphyxia.[7,8] The intimate interrelation of the potassium ion and acetylcholine has been described.[9,10] The other is the depressant action of cH upon the

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physiological action of adrenaline.[11] The latter offers most interesting possibilities in the dual sympathetic and parasympathetic controls of the body of which cardiac regulation is an example. It strongly suggests a reciprocating chemical control built upon physiological variation of the hydrogen ion concentration. Specifically, a rising cH increasing the parasympathetic inhibitory action might be supported by a diminishing intensity of sympathetic excitatory action, and a fall in cH would be associated with a converse reciprocal interaction. Central neuroreciprocal action between the excitatory and inhibitory cardiac centers would be complemented by an outlying chemical reciprocation.

On the assumption that carbon dioxide can substitute for eserine and related compounds for producing experimental effects in those systems of the body where acetylcholine is normally deposited, it is deemed advisable to reinvestigate many of the fundamental researches in neurohumoral physiology. In the light of normal and pathological fluctuations in acid-base equilibrium and the practical applications which may arise, this course is imperative. The valuable studies of Cannon and Rosenblueth[12] and their associates on the five stages of stimulation where physostigmine has such profound effects is but a single example. Will carbon dioxide produce comparable effects to those of physostigmine on these phases of stimulation? Is an increasing muscle cH a possible explanation of the fifth stage? These are merely illustrations of the many questions that may be raised to test the application of the acid neurohumoral mechanism of activation in the body.

13598

Influence of Intensity of White Light upon Pupil Diameter of the Human and of the Rabbit.

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Hecht and Pirenne[1] attempted to measure the minimum visual threshold of the nocturnal long-eared owl by comparing the inten-

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