

on hyperventilation. Our data show, however, that the relationship is not a simple one, since it is not possible to predict the amount of slow activity from the sugar value alone. As in all biological processes, there are many factors at work at the same time. In this case it is not at present possible to decide whether these factors cloud any direct relationship that sugar may have, or whether blood sugar level is not of primary importance but is capable of stimulating some other mechanism which controls the production of slow potentials in the EEG on hyperventilation. Until the other factors concerned with slow activity are identified, it is desirable to have the blood sugar as near as possible to 120 mg %.

It is doubtful that fasting is a highly significant variable in the appearance of slow rhythms after hyperventilation.³ In our experience, on some occasions fasting resulted in the appearance of slow waves on hyperventilation, but was without effect at other times.

Summary. Lowering blood sugar below 120 mg % may influence the response of the electroencephalogram to hyperventilation. Consequently it is advisable to keep blood sugar at a level of 120 mg % when the electroencephalogram is recorded during overventilation.

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Inhibition of Experimental Auricular Fibrillation by Procaine and Other Substances.*

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Shen and Simon¹ and van Dongen² have demonstrated that procaine (novocaine) inhibits the development of arrhythmias and ven-

³ Liberson, W. T., and Strauss, H., *Proc. Soc. Exp. Biol. and Med.*, 1941, **48**, 670.

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¹ Shen, T. C. R., and Simon, M. A., *Compt. rend. de la Soc. de Biol.*, 1938, **127**, 1457.

² van Dongen, K., *Arch. internat. de pharmacodyn. et de therap.*, 1936, **53**, 80; 1936, **54**, 252; 1938, **60**, 207.

tricular fibrillation which may occur upon the injection of epinephrine into cats and rabbits anesthetized with chloroform. Burstein, Marangoni and Rovinstine^{3, 4} have found that it exerts the same effect upon arrhythmias and ventricular fibrillation produced by epinephrine during cyclopropane anesthesia in dogs. Van Dongen² has shown also that procaine inhibits the development of both auricular and ventricular fibrillation from electrical stimulation of these chambers in cats and rabbits.

Since auricular fibrillation is much less persistent in cats and rabbits than it is in dogs and conditions in the latter approach more closely the clinical conditions in human beings we have investigated the effects of procaine and related substances on experimental auricular fibrillation in dogs.

All our animals were anesthetized with intraperitoneal injections of sodium pentobarbital (35 mg per kg).

Auricular fibrillation was produced in either of two ways: (1) Small fish hook electrodes were inserted into the walls of the right auricle and the auricle was stimulated for 5 seconds with the smallest stimulus from the secondary of a Harvard induction coil which would induce persistent fibrillation when the stimulus was repeated 3 times in rapid succession. The electrodes were made by soldering a very small fish hook to a piece of wire. This wire was passed through a piece of glass tube and then soldered to the end of a safety pin whose other end was fixed with sealing wax to the proximal end of the glass tube. The safety pin thus acted as a spring to clamp the fish hook tightly against the distal end of the glass tube. A piece of annunciator wire soldered to the safety pin connected the electrode with the pole of the induction coil. (2) We also produced auricular fibrillation by dropping acetyl beta methyl choline chloride solution on to the wall of the atrium by the method of Nahum and Hoff.⁵

A similarly constructed fish hook with glass tube and spring attachment was inserted in the wall of the right atrium. The lumen of this glass tube was 8 mm in diameter, and a small wad of cotton was fixed to its lower end so as to be in contact with the wall of the atrium. Five drops of a 1:500 solution of acetyl beta methyl choline chloride solution was then dropped into this tube and when it came

³ Burstein, C. L., and Marangani, B. A., *PROC. SOC. EXP. BIOL. AND MED.*, 1940, **43**, 210.

⁴ Marangani, B. A., Burstein, C. L., and Rovinstine, C. A., *PROC. SOC. EXP. BIOL. AND MED.*, 1940, **44**, 594.

⁵ Nahum, L. H., and Hoff, H. E., *Am. J. Physiol.*, 1940, **129**, P 428.

into contact with the atrial muscle auricular fibrillation resulted. This fibrillation lasted from 11 to 12 minutes and could be reproduced with about the same duration.

In 5 dogs we have found that procaine in doses of 10 to 20 mg per kilo intravenously stopped fibrillation produced by the minimal effective faradic stimulation of the atria, but this resistance to fibrillation passed off rapidly, often within 5 minutes. Larger doses, 40 to 80 mg per kilo, prevented even maximal faradic stimulation from producing auricular fibrillation. In 4 dogs auricular fibrillation induced by dropping 5 drops of 1:500 solution of acetyl beta methyl choline chloride solution on to the atria was stopped within 35 seconds by the intravenous injection of 1 to 6 mg of procaine per kilo. The effect of the smaller doses of procaine was very transitory because the application of the same stimulus 5 minutes after cessation of the fibrillation usually caused the fibrillation to reappear.

At least 2 other local anesthetics seem to exert similar effects in inhibiting auricular fibrillation. In 3 dogs p-butyl amino-benzoyl-dimethyl amino-ethanol hydrochloride (pontocaine hydrochloride) in doses of from 0.7 to 12 mg per kg stopped auricular fibrillation produced by the minimal effective faradic stimuli, and in 2 dogs we obtained the same effect with 2-butyloxyquinoline carboxylic acid-4-diethylethylene diamide hydrochloride (nupercaine hydrochloride) in doses of from 1 to 3.75 mg per kg respectively.

Summary. At least three local anesthetics, procaine, pontocaine and nupercaine can inhibit experimental auricular fibrillation in dogs.

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Toxicity of Antiseptics for the Chick Embryo.*

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The resistance of the chick embryo to toxic substances is a subject which has been little explored¹ in spite of the fact that the embryo is well adapted to a variety of bacteriological and physiological experiments. The developing egg consists of rapidly growing em-

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¹ Witlin, Bernard, *Proc. Soc. Exp. Biol. and Med.*, 1942, **49**, 27.