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Blood Sugar Level and Influence of Hyperventilation on Slow Activity in Electroencephalogram.

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Interest has recently been focused on the relationship which seems to exist between slow potential changes appearing in the electroencephalogram (EEG) on hyperventilation and alterations in blood sugar level.^{1, 2} If a direct relationship between the two does exist, it will be necessary systematically to control the blood sugar level in all hyperventilation studies. It is not easy to maintain such a control, and before the electroencephalographer becomes burdened with this precaution, it seemed desirable to investigate the possible relationship further.

Methods. One physically healthy, schizophrenic patient was observed on 10 different occasions* over a period of 10 weeks. Three glucose and 7 insulin (3, 5, 10 U) tolerance tests were given. Preceding the collection of each blood sample an EEG was obtained in the following manner: 5 meters of control record were followed by a 2-minute hyperventilation (at a regular rate of 30 deep respirations per minute), and the record allowed to return to its initial level. In this way, many hyperventilation records at various blood sugar levels were obtained.

Slow activity in the EEG was considered to include all waves with a frequency below 8 per second, and was qualified as total slow activity in the 7-meter period, which included the hyperventilation and recovery portions of the record.

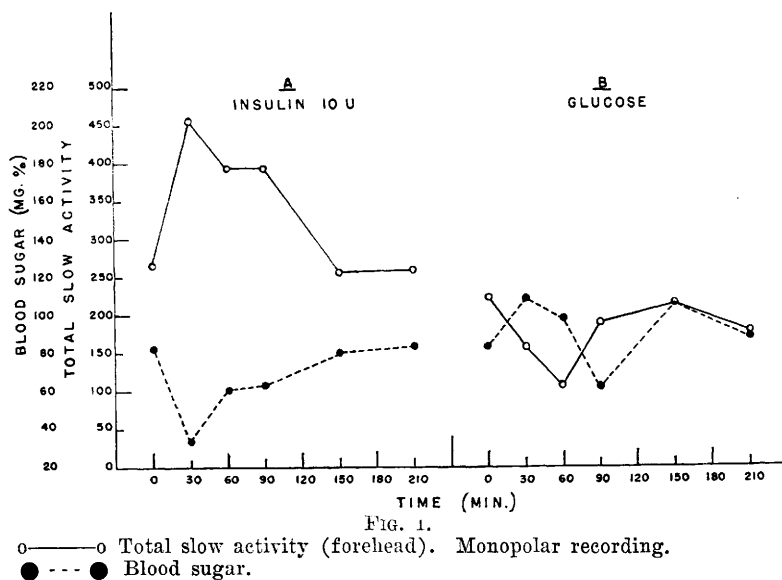
Results. Fig. 1 A shows a definite inverse relationship between blood sugar and slow activity. Fig. 1 B, however, is typical of the remainder of our data. Here slow activity is inversely related to blood sugar only at 30 and at 90 minutes. In addition, it may be noted that the minimum amount of slow activity does not correspond with the maximum blood sugar value.

When all the blood sugar values obtained were plotted against the corresponding amounts of slow activity, an apparent relation-

¹ Davis, H., and Wallace, W. M., *Am. J. Physiol.*, 1941, **133**, 258P.

² Engel, G. L., and Margolin, S. G., *Arch. Neurol. Psychiat.*, 1941, **45**, 890.

* The patient was in the basal state on each occasion (fasting for at least 15 hours previous to the experimental session).



ship was found which held for blood sugars up to about 120 mg % (Fig. 2). This relationship, however, is only a rough trend, since the scatter of the data is great. For example, in individual instances blood sugars from 27 to 110 mg % all give corresponding total slow activity of about 220.

Discussion. It is apparent that decreasing blood sugar below 120 mg % alters the amount of slow activity appearing in the EEG

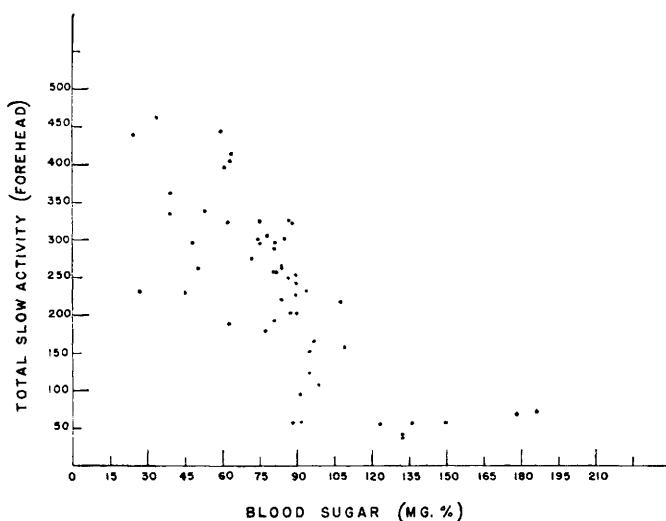


FIG. 2.

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.