the concentration of sugar in the arterial blood was compared with the respiratory quotient.

On some days the subjects fasted; on others they ate from one to five meals, variously spaced. As was to be expected, the respiratory quotients of the fasting subjects fell to values between .78 and .82 and the blood sugar to .08 and .10 per cent. In the subjects who ate, both the respiratory quotient and the blood sugar rose after the meal; but within 2 to 4 hours, if another meal was not taken, it fell again to the fasting level. When this fasting level was reached, it was maintained in the great majority of the subjects with little change for many hours. A few, however, exhibited sudden fluctuations of considerable magnitude in both the respiratory quotient and blood sugar.

Such fluctuations never occurred among the children. All the adults were free from emotional disturbances which might explain them. A search for the cause of the divergent values suggested that it was associated with smoking.

The respiratory quotients and blood sugars before and after smoking were then studied in a number of subjects. The results showed that when the respiratory quotient is above .85 and the blood sugar correspondingly above .13 per cent., the smoking of a cigarette has no appreciable influence upon either. When, however, the respiratory quotient and blood sugar have fallen below these values, and especially when the fasting level has been reached, the smoking of a cigarette is followed by a rise in both. Values are attained within 15 minutes as high as .85 or .90 for the respiratory quotient and .12 or .14 per cent. for the sugar. During the next 30 minutes the values fall gradually to, or slightly below, those observed before the cigarette was smoked.

It is a well-known fact that injection of nicotine into animals is followed by a temporary rise in blood sugar.[2,3,4] But so far as we can find no one has previously reported a similar rise in man resulting from the nicotine of tobacco smoke.

From animal experimentation it is well established that it is the action of nicotine upon the adrenal glands which leads to the hyperglycemia.[3, 5, 6, 7] The rate of discharge of adrenalin is increased; and the liberated adrenalin exerts its characteristic glycolytic action. The glycogen stored in the liver and muscles is converted into sugar. In consequence the concentration of sugar in the blood is increased. Secondary to the rise in sugar the combustion of carbohydrate is increased and can be observed in the increase in the value of the respiratory quotient. But, as already stated, these metabolic effects do not result from smoking when the blood sugar is at a concentration above .13 per cent. as it is for 2 to 3 hours following a meal.

The acceleration of sugar metabolism thus demonstrated affords a possible explanation for the fact that smoking diminishes hunger in many users of tobacco. Hunger appears from our observations and those of other investigators to arise within a definite time after the blood sugar falls to the fasting level. Tobacco smoking, by inducing a hyperglycemia, may thus delay temporarily the development of hunger.

Our observations on the mealtime intervals indicate that the hyperglycemia following a meal definitely relieves the fatigue and irritability that generally develop soon after the fasting level of blood sugar is reached. Smoking by inducing a hyperglycemia temporarily relieves these conditions.

The other effects of smoking, the acceleration of the pulse and the temporary rise in arterial pressure, are presumably, like the increase in sugar concentration, dependent upon discharge of adrenalin.

Our observations demonstrate why tobacco rather than any other substance is used for smoking; the smoker obtains from tobacco repeated minute doses of nicotine.

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