lem. A certain percentage of non-hereditary rumpless specimens can be produced.

Presumably the condition is brought about through a slowing of the growth rate just when the posterior end of the body should be in the state of its most rapid differentiation, a time, according to Stockard,[4] when any arrest is likely to produce lasting effects. That the condition is one of arrested development is further indicated by a rather frequent occurrence of ectopic viscera among these specimens.

These results with eggs of normal stock suggest that the genetic form of the trait may similarly be determined by a differential retardation at some critical moment. The available eggs were not sufficient to test this point, but so far as they go the data are suggestive, inasmuch as from eggs incubated at consistently low temperatures there were 6 normal embryos and one doubtful, while from those incubated wholly or in part at higher temperatures there were 9 normal and 8 rumpless chicks. It is regretted that it has not yet been possible to make tests to determine whether genetically rumpless embryos may have their general metabolism sufficiently slowed during the first week of incubation to reduce the differential to a point that would permit development of normal sacral and caudal vertebrae.

At present something can be done toward making an embryo of normal genotype develop, or fail to develop, a tail. It remains to be determined how a similar control may be exercised over a genetically rumpless embryo.

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Effect of Intravenous Injection of Ethyl Alcohol on Gastric Secretion in Man.*

H. W. NEWMAN AND H. G. MEHRTENS.

From the Division of Neuropsychiatry, Stanford University Medical School.

Of the tests of gastric secretion, that employing alcohol as the stimulus is one more commonly used clinically. The recent use of

[4] Stockard, Charles R., *Am. J. Anat.*, 1921, **28**.

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alcohol by vein as a surgical anesthetic[1, 2] suggested the question as to whether intravenous alcohol would prove as active a stimulus to gastric secretion as that administered by mouth.

In the alcohol test meal as used in practice, 50 cc. of 7% alcohol is introduced into the previously emptied stomach, and the contents aspirated at 15-minute intervals for titration.[3] We have attempted a comparison of the changes in gastric acidity, blood alcohol, and gastric juice alcohol brought about by this procedure with those produced by varying doses of alcohol intravenously. In all cases the solution used in the intravenous test meal was 25% ethyl alcohol in normal saline, injected at a constant rate of 10 cc. per minute. The alcohol determinations were made by the method described by Cannan and Sulzer.[4]

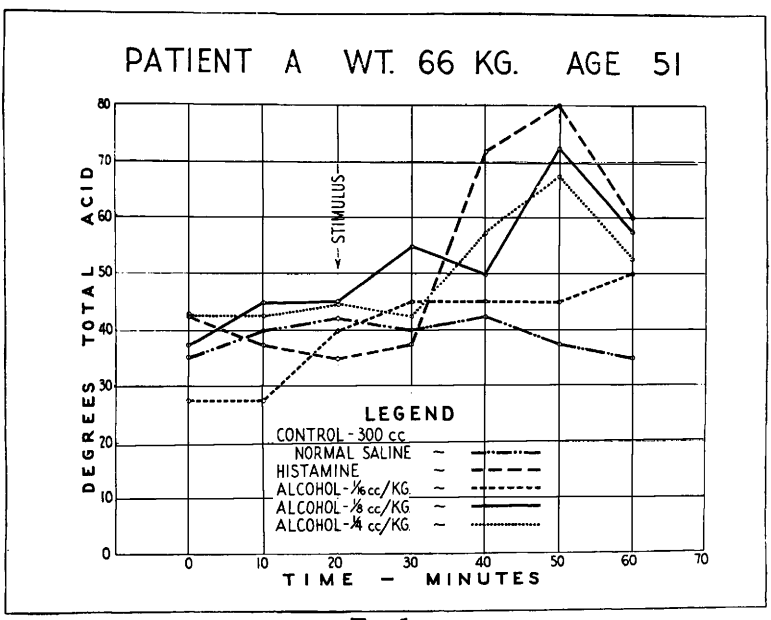

FIG. 1.

Figure 1 shows that in this patient the injection of 300 cc. of normal saline at the prescribed rate as a control had no appreciable effect on gastric acidity, as determined by titration of samples aspirated continuously over successive 10-minute periods. It also shows the response of this same patient to doses of 1/16, 1/8, and 1/4 cc.

[1] Garcia, Miguel, 1929, *E. Mesones. Imp.*, Mexico.

[2] Fohl, Th., *Arch. f. Klin. Chir.*, 1931, **165**, 641.

[3] Cheney, W. F., *Oxford Monographs*, 1928.

[4] Cannan, R. K., and Sulzer, R., *Heart*, 1924, **11**, 141.

of 95% alcohol per kg. body weight intravenously as the 25% solution, and also to 0.01 mg. histamine per kg. subcutaneously. It will be noted that 1/8 cc. of alcohol per kg. gives a maximal response, but that the response is not so great as that to histamine.

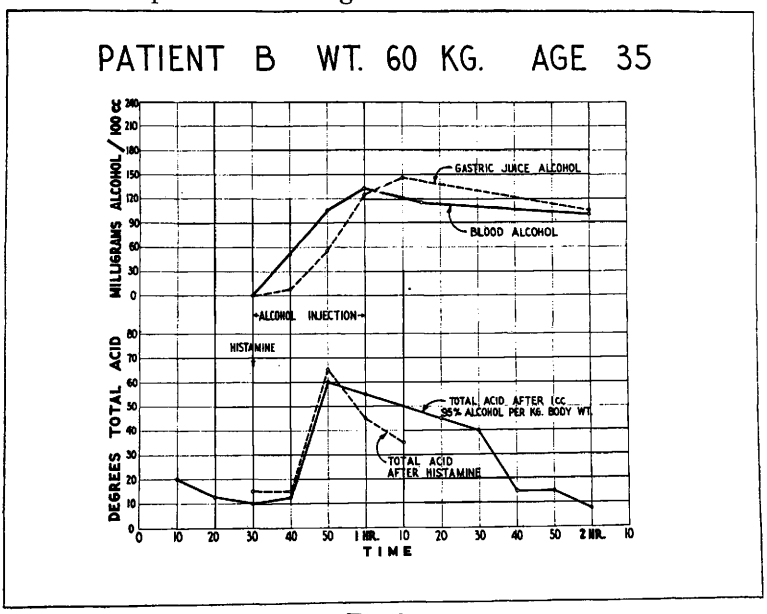

FIG. 2.

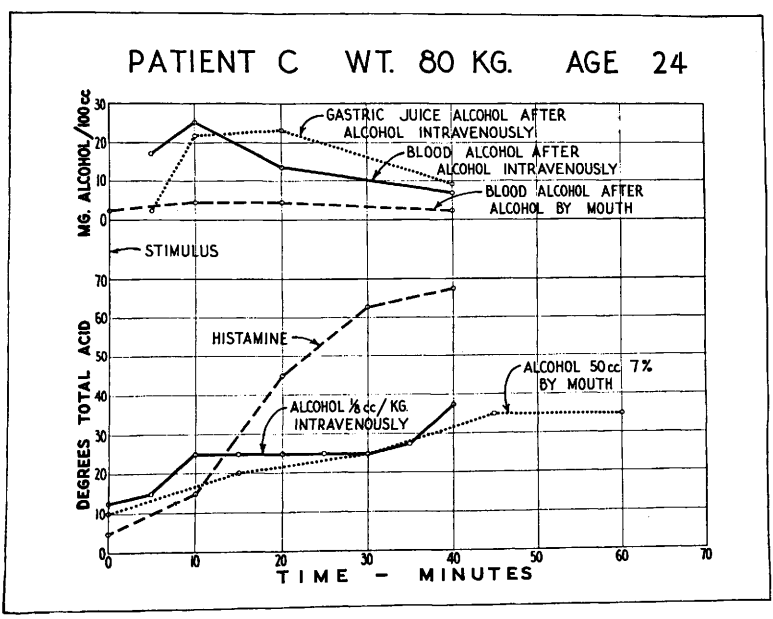

FIG. 3.

Figure 2 shows the response of another patient to 1 cc. of 95% alcohol per kg. intravenously as the 25% solution. Here again the response does not equal that to histamine. The alcohol content of the gastric juice closely follows that of the blood.

Figure 3 shows the response of a third patient to the oral alcohol test meal, to 1/8 cc. of 95% alcohol intravenously, and to histamine; as well as the blood alcohol curve during the oral and the intravenous meal. There is no significant difference evident in the responses to alcohol by the 2 routes, indicating that the mechanism in both cases may be the same. Here the response to histamine far exceeds that to alcohol orally or intravenously.

The foregoing work merely indicates the field opened up by this new method of approach. We will report an investigation of psychological and psychiatric problems by this method in future papers.

Conclusions. 1. Alcohol intravenously in man produces an increase in gastric acidity. 2. 1/8 cc. of 95% alcohol per kg. body weight intravenously as a 25% solution in normal saline is as effective a stimulant of gastric secretion as is the standard 50 cc. of 7% alcohol by mouth. 3. The response to alcohol intravenously up to 1 cc. per kg. is not as great as that to histamine. 4. The alcohol content of the gastric juice closely follows that of the blood. 5. The blood alcohol during the oral alcohol test meal is much less than after the least effective dose by vein. 6. The gastric juice alcohol after the intravenous injection is minute compared to the 7% administered by mouth. 7. From 5 and 6 above, it would seem possible that the seat of action of alcohol as a stimulus to gastric secretion lies neither in the general circulation, nor at the surface of the gastric mucosa, but somewhere between the two. Further investigation of this point is in progress.