such long afferent loops invite stomal ulcer.

A large number of experiments in each group would undoubtedly be helpful in resolving the importance of each of the factors scrutinized in this study. In addition, the animals not dying of spontaneous perforation of a stomal ulcer should be allowed to survive longer before sacrifice.

It is not unlikely that, employment of additional modes of attack may help to separate out more definitely, the component important parts in the predisposition to stomal ulcer presented by the long afferent duodenojejunal loop. Three such methods are now being applied to the problem in this laboratory: (1) assay of the secretin potency of intestinal mucosa from varying levels of the bowel in both dog and man in dogs with pancreatic fistula. (2) Determination of the loss in titratable alkalinity, if any, of the content of the long afferent duodenojejunal loop as delivered at the afferent gastrojejunal stoma (3) Experiments in which the sensitivity of the mucosa of various segments of the intestine is examined by allowing hydrochloric acid to drip upon isolated surfaces.

Conclusions. It is difficult to separate out with finality the role of the various factors contributing to the development of stomal ulcer attending employment of a long afferent loop in the operation of complete intragastric drainage of the content of the duodenal loop. The secretin factor can not be divorced completely from the considerations of the "distance" factor. Experiment No. 12 suggests rather definitely that, the "sensitivity" factor is not as important as the other two.

The evidence garnered in this study lends strong confirmation to the deductions arrived at in the previous two studies indicating that, a long afferent duodenojejunal loop invites stomal ulcer in any gastric operation carried out on the Billroth II plan of procedure.

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Effect of 2, Methyl Amino Heptane Upon Cardiac Automaticity During Cyclopropane Anesthesia in Dogs.

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Epinephrine and certain related aromatic amines, when used during cyclopropane anesthesia, cause pronounced cardiac irregularities. Ventricular premature beats, ventricular paroxysmal tachycardia, auricular and ventricular fibrillation are the usual manifestations.[1,2] Recently a group of aliphatic amines has been prepared which possess vasopressor activity and other pharmacologic effects of epinephrine. Studies on cardiac automaticity during cyclopropane anesthesia using these aliphatic amines have not been reported. One of these, 2-methyl amino heptane (EA-1) is useful to overcome circulatory disturbances during spinal anesthesia.[3,4] Inasmuch as cyclopropane is frequently used in conjunction with spinal anesthesia and may be used when this amine has been administered, it is essential to determine if it causes disturbances in cardiac rhythm and if these disturbances are similar to and as severe as those caused by epinephrine.

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Method. Twenty-three observations were made on 5 dogs. The dogs were premedicated with morphine 1 mg/kilo and scopolamine .04 mg/kilo administered intravenously. Anesthesia was maintained at the point of intercostal paralysis with cyclopropane-oxygen mixtures using rebreathing by means of the carbon dioxide absorption technic. The amine dissolved in .5 cc of sterile water was given intravenously in one single injection over a period of 30 seconds. Studies were made on each dog before premedication, after premedication and after cyclopropane with premedication. The drug was administered from 15 to 30 minutes after the induction of cyclopropane anesthesia. Electrocardiographic tracings, standard leads 1, 2, 3, and lead 4 (esophageal) were taken one minute after completion of the injection at which time the pressor effect is usually established. Tracings were also made 10 minutes after the injection because the pressor action begins to disappear at this time. In 3 dogs, arterial cannulation was performed and kymographic tracings of blood pressure were correlated with the electrocardiographic studies. When the blood pressure had returned to the control level, following the use of EA-1, epinephrine .01 mg/kilo was then given and electrocardiograms taken. In dogs IV and V, carotid cannulation was not done and the order of injection reversed—

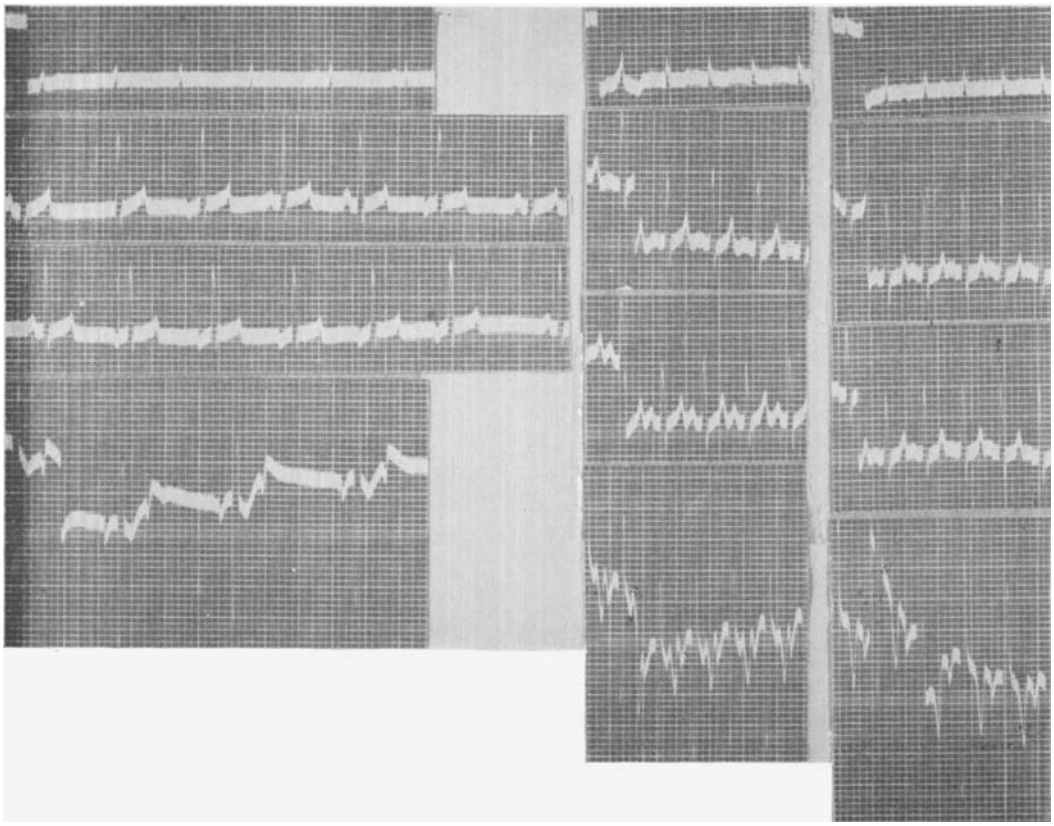
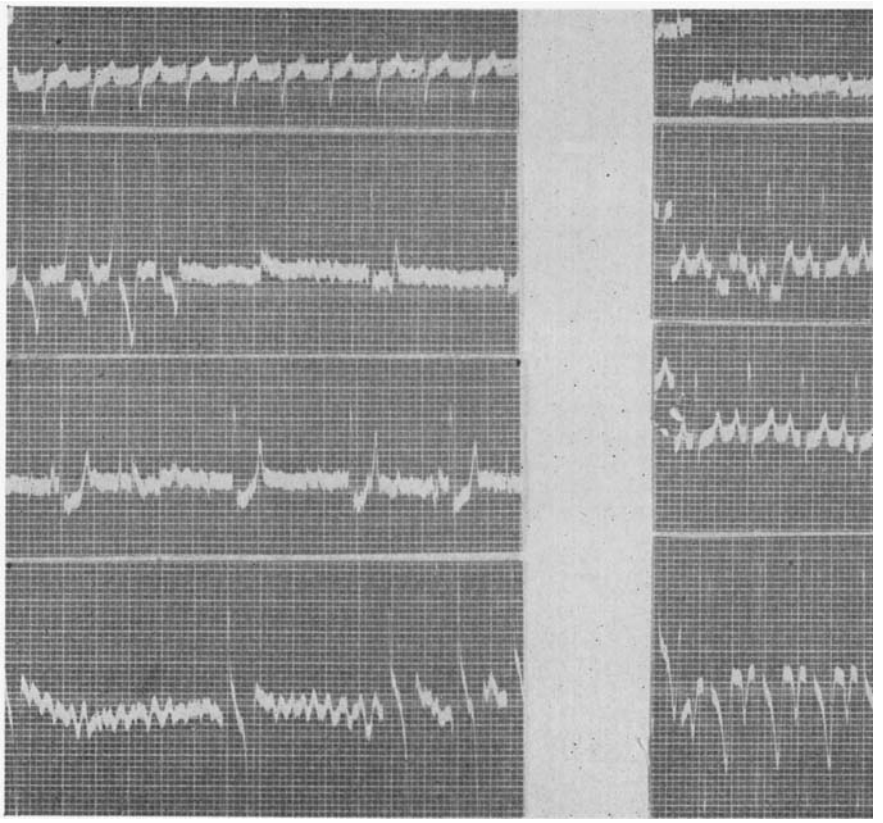


FIG. 1-A.—Dog I.—Control after cyclopropane and premedication. Sinus arrhythmia with change in P waves which may be due to shift in pacemaker or change in position of heart. (This is normal in the dog.)

FIG. 1-B.—Dog I.—One minute after EA-1. Sinus tachycardia; one to one conduction. Intraventricular conduction normal.

FIG. 1-C.—Dog I.—Ten minutes after EA-1. Rate of sinus tachycardia slightly increased. No other change.



A B

FIG. 2-A.—Dog. I.—One minute after epinephrine. During the first part of the ECG there is a regular, rapid, ventricular rate possibly ventricular paroxysmal tachycardia. During this period, the auricles may be fibrillating. The tachycardia ceases abruptly in lead 2 and the mechanism for the rest of the ECG is auricular fibrillation with varying A-V conduction.

FIG. 2-B.—Dog I.—Ten minutes after epinephrine. A sinus mechanism with arrhythmia is restored.

epinephrine being given before EA-1. In dog I, the effects of excessive doses of EA-1 (2.5 mg/kilo) were also studied.

Results. In controls without premedication or anesthesia 1.0 mg/kilo of EA-1 (therapeutic dose) produced neither notable changes in conduction nor abnormal rhythms. Likewise, no notable changes followed its administration after morphine and scopolamine. In dogs I, IV and V, the administration of EA-1 during cyclopropane anesthesia with or without premedication consistently produced sinus or auricular paroxysmal tachycardia but no other disturbance of rhythm or conduction. (Fig. 1).

In dog II under anesthesia, EA-1 produced the most profound effects in the whole series of observations. Auricular and ventricular premature beats, intraventricular block with variations in the form of QRS complexes, were observed. In dog III under anesthesia, EA-1 produced fleeting ventricularparoxysmal tachycardia in one instance. This was not produced during subsequent studies.

Epinephrine in these same 5 dogs .01 mg/kilo to .03 mg/kilo produced marked changes of conduction and rhythm *viz.*, from auricular and ventricular premature beats, ventricular paroxysmal tachycardia and auricular and ventricular fibrillation. (Fig. 2).

Cyclopropane anesthesia caused an increase in heart rate which was further increased by the administration of EA-1. No correlation was observed between ECG and

blood pressure changes. The EA-1 did not reverse the effects of epinephrine or prevent their appearance.

Comments. EA-1, in doses ranging as high as 2.5 mg/kilo, will cause disturbances of cardiac rhythm in the unanesthetized dog. Auricular paroxysmal tachycardia, auricular and ventricular premature beats are the most commonly observed disturbances. Therapeutic doses cause no such changes in the unanesthetized dog. However, in the heart sensitized by cyclopropane anesthesia, therapeutic doses can produce sufficient stimulation to cause disturbances in rhythm. These disturbances affect the auricles principally and appear to exert little effect on the ventricular automatic tissue.

Summary. In 5 dogs premedicated with morphine and scopolamine and anesthetized with cyclopropane, 2-methyl amino heptane (EA-1) caused sinus and auricular paroxysmal tachycardia, and ventricular premature beats. Ventricular paroxysmal tachycardia and auricular and ventricular fibrillations were not observed.

The writers are indebted to Dr. James Gouax of the Heart Station, Charity Hospital for interpretation of the electrocardiograms and to Dr. Ray Parmely for assistance in anesthetizing the dogs.

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Inhibition of the Deciduomal Response by Preexisting Deciduomata in the Mouse*†

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Loeb[1,2] discovered that surgical traumatization of the uterus during periods of luteal activity was followed by growth and differentiation of the endometrium into a structure which he called a deciduoma. In more recent years the observations of Krehbiel[3] and Atkinson[4] on the morphology and physiology of deciduomata have demonstrated that the reaction is the experimental equivalent of the naturally occurring maternal placenta.

It has been reported by Selye and McKeown[5] that deciduoma formation cannot be elicited after placentation in the rat. Moreover, these authors found that if the pregnant horn of the unilaterally pregnant animal is removed, the remaining non-pregnant horn will quickly regain its sensitivity to traumatization. Atkinson[6] demonstrated that this uterine desensitization to deciduoma formation is not due to changes in the estrogen-progestin balance in the pregnant mouse.

The phenomenon of uterine desensitization is difficult to analyze in the pregnant animal due to the complexity of the factors involved. However, it is possible to evaluate the role of the maternal placenta alone through the study of its experimental counterpart, the deciduoma. The present experiments were designed to determine whether the presence of deciduomata in one uterine horn would influence the responsiveness of the contralateral horn.

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