to serious variations in geometry.

Summary. A method for the determination of Iodine-131 in urine is described. A dipping counter mounted on a modified microscope base using a blackened test tube with a sample large enough to cover the sensitive area of the tube was used. Counting of gamma radiations is quite reproducible by this procedure.

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Effect of Procaine Hydrochloride on Response of the Heart to Epinephrine During Cyclopropane Anaesthesia. (17362)

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While studying the production of ventricular fibrillation in dogs by the intravenous administration of epinephrine during cyclopropane anaesthesia, observations were made on the effect of procaine HCl on the cardiac irregularities. Its effect on the rate and rhythm of the heart during cyclopropane anaesthesia and its effect on the cardiac response to epinephrine were noted.

Method. A modification of Meek's technic was used.[1] Thirty minutes after premedication with 1.0 mg/kg morphine sulphate and 0.04 mg/kg atropine sulphate, 18 male and female dogs (4-10 kg in weight) were induced with a cyclopropane and oxygen mixture, intubated, and allowed a further 30 minutes to equilibrate with the cyclopropane-oxygen mixture administered by a to-and-fro carbon dioxide absorber. The mixture was adjusted to produce stage III, plane 3 anaesthesia. A total of 78 injections of epinephrine 0.01-0.03 mg/kg were made, taking 20 seconds for each injection, and with at least 20 minutes between injections. Procaine HCl 5-15 mg/kg in 5 cc saline was given in 50 seconds, 5 minutes before 32 of the epinephrine injections. On 7 occasions 50 mg/kg of procaine HCl were given and not followed by epinephrine. Electrocardiograms were recorded continuously during and following each injection. When ventricular fibrillation occurred, artificial respiration with 100% oxygen was instituted, thoracotomy performed, and cardiac resuscitation attempted using manual massage and intracordial injections of procaine HCl 5-15 mg/kg.

Effect on Cardiac Rate and Rhythm. The cardiac rate was increased in all but two experiments when procaine HCl was given. (Table I). The degree of acceleration present 5 minutes after the injection increased with the dose employed (Table II). On 24 occasions irregularities were present at the time procaine HCl was injected, and with the exception of one sinus arrhythmia these changed to a regular sinus rhythm. (Table I).

Modification of Cardiac Response to Epinephrine. The response of the heart to epinephrine was notably different after the administration of 5-15 mg/kg of procaine HCl. Epinephrine was given 46 times without previous procaine HCl and 32 times following this agent. The occurrence of various rhythms following the epinephrine is shown in Table III. Calculation of "p" value[2] reveals a significant increase in the number of normal rhythms, a significant decrease in the number of cases with a sinus arrhythmia component in the rhythm, and no significant change in the incidence of either extrasystoles or of ventricular tachycardia.

An attempt was made to determine whether increased vagal tone was contributing to the electrocardiographic picture obtained following epinephrine injections. Signs taken to mean increased vagal tone were initial slowing, A-V block, and suppression of the sinus

[1] Meek, W. J., Hathaway, H. R., Orth, O. S., *J. Pharm. Exp. Therapy*, 1937, **61**, 240.

[2] Mainland, D., *Can. J. Research*, 1948, E, **26**, 1.

TABLE I.
Effects of Intravenous Procaine HCl (5-15 mg/kg) on Cardiac Rate and Rhythm Under Cyclopropane.

Rhythm before procaine HCl	No. of exp.	After procaine HCl	
		Reg. sinus rhythm	Acceleration
Regular sinus rhythm	8	8	7
Sinus arrhythmia	18	17	18
Heart block	2	2	2
Bigeminal rhythm	3	3	3
Irregular bradycardia	1	1	0
	32	31	30

TABLE II.
Cardiac Acceleration with Procaine HCl.

Dose, mg/kg	No. of exp.	Avg rate when procaine given	Avg rate 5 min. after procaine
5	12	73.7	93.0
15	20	73.2	119.0
50	7	62.4	138.5

TABLE III.
Rhythms Produced by Intravenous Epinephrine.

	No. of injects.	Rhythms observed			
		Reg. sinus	Vent. tachy.	Ex. systol	Sinus arr.
Epinephrine alone	46	2	17	17	9
Epinephrine after procaine HCl	32	9	10	10	0
"p" values		<0.01	>0.7	>0.7	<0.05

node with a slow nodal or ventricular rhythm. Evidence for an increase in vagal tone was absent in only ten of 46 records where epinephrine was given alone, but was absent in 29 of 32 records where epinephrine followed procaine HCl. The p value for this difference is <0.001.

Ventricular fibrillation occurred in 2 of 17 animals when epinephrine was administered alone, and in 3 of 12 when epinephrine was preceded by 5 mg/kg of procaine HCl. These 3 animals had all received the same dose of epinephrine in previous experiments with the onset of ventricular extrasystoles or ventricular tachycardia only. We were unable to restore a normal rhythm to the fibrillating ventricles using manual massage, artificial ventilation, and intracordial procaine HCl, 15 mg/kg.

Comment. Ectopic rhythms may arise following the depression of the normal pacemaker or as a result of impaired intracardiac conduction. The administration of epinephrine may produce this effect reflexly through pressor-vagal reflexes with increase in vagal tone. Changes in vagal tone accompanying the phases of respiration are regarded as the cause of sinus arrhythmia. The increase in cardiac rate following procaine HCl, the absence of sinus arrhythmia, and the failure to demonstrate an increase in vagal tone following epinephrine injection all point to an anti-vagal action of procaine HCl in these experiments.

Burstein *et al.*[3] have reported that by the administration of procaine HCl (5 mg/kg) intravenously, cyclopropane-epinephrine induced ventricular fibrillation may be prevented from occurring, and that the fibrillating heart may be restored to a normal sinus rhythm. We feel that their own observations

[3] Burstein, C. L., Marangoni, B. A., DeGraff, A. C., and Rovenstine, E. A., *Anesthesiology*, 1940, **1**, 167.

do not justify their conclusions, and our results are in disagreement on both points. Not only have we failed to show a protecting effect by procaine HCl, but like Stutzman *et al.*,[4] we have failed to revive the fibrillating heart by cardiac massage and the intracordial injection of 15 mg/kg of procaine HCl.

Allen *et al.*[5] have demonstrated a protecting action of procaine HCl in doses of 16 mg/kg against cyclopropane-epinephrine induced ventricular tachycardia, and feel that such protection is brought about by depression of the ventricular muscle. In their work the epinephrine and procaine HCl were given together, while in our experiments the procaine HCl preceded the epinephrine by 5 minutes. Our inability to duplicate their results may indicate that any such protection of the myocardium is a transient one, particularly when larger doses of epinephrine are employed.

Conclusions and Summary. Intravenous procaine HCl increases cardiac rate and restores a regular sinus rhythm to those hearts showing irregularity during cyclopropane anaesthesia. This may account for the favorable clinical impressions on the use of procaine HCl.

A protecting action against cyclopropane-epinephrine induced ventricular tachycardia and fibrillation with doses of procaine HCl reasonable for prophylactic purposes during anaesthesia could not be demonstrated. In our opinion such protection has not yet been demonstrated by others.

Under the conditions of the experiment the predominating effect of procaine HCl given intravenously was an anti-vagal one.

4 Stutzman, J. W., Allen, C. R., and Orth, O. S., *Anesthesiology*, 1945, **6**, 57.

5 Allen, C. R., Stutzman, J. W., Slocum, H. C., and Orth, O. S., *Anesthesiology*, 1941, **2**, 503.

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Response of the Syrian Hamster to Intradermal Injection of Modified Newcastle Disease Virus. (17363)

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The adaptation of the California strain (No. 11,914) of Newcastle disease virus to the Syrian hamster with 300 intracerebral serial passages and with 9 intranasal serial passages has been reported.[1] Various subcultures of hamster brain material, up to the 200th[2] intracerebral serial passage, were injected intradermally into 4-week-old hamsters (average weight 28 g). No evidence of Newcastle virus infection was noted, although hamsters injected intracerebrally with the same brain suspension showed typical symptoms. No attempts were made to establish Newcastle disease by intradermal injection from the 200th through the 299th intracerebral passages.

The modified hamster virus was first successfully established intradermally by the inoculation of 0.1 cc of a 10% brain suspension of the 300th intracerebral passage in the abdominal region of four 4-week-old hamsters (average weight 28 g). Simultaneously 4 hamsters of the same age and weight were inoculated similarly with 0.1 cc of a 10% brain suspension of the 14th intranasal passage. Both groups of hamsters showed signs of involuntary motor reaction, evidence of salivation, and paralysis in 5 to 10 days.

1 Reagan, R. L., Lillie, M. G., Smith, D. C., and Brueckner, A. L., PROC. SOC. EXP. BIOL. AND MED., 1949, **71**, 293.

2 Reagan, R. L., Lillie, M. G., Hauser, J. E., and Brueckner, A. L., PROC. SOC. EXP. BIOL. AND MED., 1948, **68**, 293.