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.

Received August 29, 1949. P.S.E.B.M., 1949, **72**.

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.

TABLE I.
Intradermal Passage of a Strain of Hamster-Adapted Newcastle Virus.

Material used	Site of inoculation	Passage No.	No. animals inoculated	No. animals showing symptoms	No. days after inoculation symptoms appeared
0.1 cc of 10% suspension of 14th intranasal pasage	Intradermally in abdominal area	1	4	4	5–7–7–10
		2	6	3	3–4–4
		3	6	2	5–6
		4	6	4	5–6–6–6
		5	6	4	5–6–6–7
		6	3	3	4–4–4
		7	6	3	4–5–5
		8	6	6	4–4–4–5–5–5
		9	4	4	4–5–5–5
		10	4	4	3–4–5–5
0.1 cc of 10% suspension of 300th intracerebral passage	Intradermally in abdominal area	1	4	4	5–6–6–10
		2	4	4	5–5–5–5
		3	4	4	5–6–6–6
		4	3	3	3–5–6
		5	4	4	3–5–5–6
		6	4	4	3–4–5–5

These symptoms are similar to those occurring in hamsters infected intracerebrally, although salivation has occurred regularly only in later passages. Brains were removed aseptically from the moribund hamsters of each series, ground with alundum, and diluted to a 10% suspension with physiological saline. One-tenth cc of this suspension from each series was injected intradermally into hamsters. By using this technic, the 300th intracerebral brain material was carried up to 6 passages intradermally, and the 14th nasal brain material was carried up to 10 passages intradermally, as shown in Table I. These subinoculations are being continued at the present time.

To determine the distribution of Newcastle virus in the affected hamsters of each series, blood, liver, spleen, kidneys, lungs, spinal cord, and brain were injected into embryonated eggs. Each organ was removed aseptically, ground with alundum and diluted to a 10% suspension with physiological saline. One-tenth cc of citrated blood and of each organ suspension was injected into the allantoic sac of each of six 8-day embryonated White Leghorn eggs. Because of possible bacterial contamination, the lungs, kidneys, liver, and spleen were treated with streptomycin in the ratio of 1 mg per cc of suspension and with penicillin in the ratio of 500 units per cc of suspension before egg inoculation.

Virus was isolated from the brain and spinal cord of hamsters of each series. Virus from the brain and cord of each series was completely neutralized by positive Newcastle chicken serum but it was not affected by normal chicken serum. Virus was not isolated in embryonated chicken eggs from the blood or other organs of either series.

Day-old chicks were injected intramuscularly with 0.2 cc of a 10% suspension of brain material from each series. All chicks showed symptoms of Newcastle disease from 3 to 10 days after injection. Six-week-old chickens were injected intramuscularly with the same amount of a 10% suspension of brain material from each series but showed no evidence of Newcastle disease.

Discussion. In the subinoculations up to the 200th intracerebral hamsters passage, hamsters were not infected intradermally. Upon trial of the 300th intracerebral passage and of the 14th intranasal passage, hamsters were infected intradermally, when brain material was used as the inoculum. The 300th intracerebral passage was carried through 6 subinoculations intradermally, and the 14th intranasal passage was carried 10 subinoculations intradermally.

With hamsters as the test animals, virus neutralization tests were conducted, using brain suspensions of the 3rd intradermal passage of the 300th intracerebral series, and

TABLE II.
Titration of Hamster-Adapted Newcastle Virus in Hamsters.

Virus	Dilution 10-1	10-2	10-3	10-4	10-5	Titer
Hamster brain 5th intradermal passage originally from 14th nasal passage	*4/4	4/4	4/4	0/4	0/4	$10^{-3.5}$
Hamster brain 3rd intradermal passage originally from 300th intracerebral passage	3/3	4/4	2/4	0/4	0/3	10^{-3}

* Numerator denotes No. of deaths.
Denominator denotes No. of animals inoculated.

the 5th intradermal passage of the 14th intranasal series. Specific immune chicken serum completely neutralized the intradermal brain virus from both series, whereas normal chicken serum had no effect.

The virus titered 10^{-3} in the first series (300th intracerebral) and $10^{-3.5}$ in the second series (14th intranasal), according to the Reed-Muench calculation.[3] Details are given in Table II. Paralysis of the pharynx, as evidenced by excessive salivation with wetting of the mouth and chin, is now a distinct symptom in hamsters.

It has been observed in other modified forms of this virus that day-old chicks are susceptible, whereas older individuals are resistant to infection.

Summary. After the 300th intracerebral and the 14th intranasal hamster passage, Newcastle disease virus, California strain No. 11,914 can successfully infect hamsters by intradermal injection. The virus in the brain material from each series was passed intradermally in hamsters. Virus was isolated from the brain and cord of hamsters of both series upon inoculation of embryonated chicken eggs. The results of the neutralization tests confirm the virus to be that of Newcastle disease. Hamsters infected intracerebrally, intranasally, and intradermally show similar symptoms, such as involuntary motor reactions, excessive salivation from pharyngeal paralysis, general paralysis, prostration, and death.

Received September 14, 1949. P.S.E.B.M., 1949, **72**.

[3] Reed, L. J., and Muench, H., *Am. J. Hyg.*, 1938, **27**, 493.

Effect of Oxythiamine on the Growth of Chicks.* (17364)

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Bergel and Todd[1] reported the synthesis of oxythiamine.‡ They found that 1 mg of this compound possessed no antineuritic ac-

* This work was undertaken in cooperation with the Office of Naval Research, Navy Department, Washington, D.C., and was aided by a grant to Cornell University by the Nutrition Foundation, Inc., New York.

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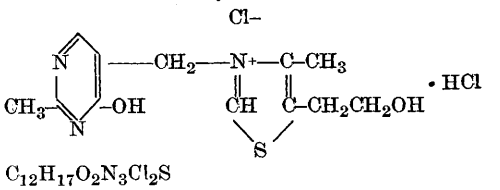
[1] Bergel, F., and Todd, A. R., *J. Chem. Soc.*, 1937, 1504.

‡ The formula of oxythiamine is



$C_{12}H_{17}O_2N_3Cl_2S$