tem,[8] while those which died despite the treatment showed similar pathologic changes as the untreated mice except that the cytoplasmic basophilic substance of the nerve cells appeared better preserved.

Discussion. The described experiments indicate that malononitrile has a certain value in the treatment of mouse poliomyelitis. If lower concentrations of the virus were used, treatment in the preparalytic stage prevented death in more than one-half of the animals and prolonged the incubation period nearly threefold. While death following huge doses of the virus could not be prevented by the drug, the life span between paralysis and death was prolonged also in these cases.

When malononitrile was given after the appearance of paralysis, in about one-third of the animals significant regression of the clinical symptoms was observed. Complete recovery was observed in solitary cases but the animals died after cessation of the treatment.

There are various ways in which a chemical compound may act against the poliomyelitis virus. It may have a prophylactic effect either due to the action upon the virus before it reaches the nervous system, or it may render the nerve cells unsuitable for the multiplication of the virus. The arrangement of these experiments precludes the possibility of a direct inactivation of the virus before it reached the nervous system. Malononitrile, however, may render the intracellular environment unsuitable for continued development of the virus by its protective effect upon the nucleic acids. It may also help to restore the damaged function by increasing the ribonucleic acid content of the attacked cells. While a direct action upon the virus within the cell is not probable, the possibility that this compound acts by interfering with an enzyme system of the host, essential for the multiplication of the virus, has to be considered.

Summary. Malononitrile treatment preserved the life of more than one-half of the mice infected with a low concentration of the Lansing type poliomyelitis virus, but only prolonged the life span of mice infected with large doses of the virus. Mice treated with malononitrile after the onset of the paralysis showed markedly prolonged survival time. In about one-third of the animals partial or total recovery of the paralytic symptoms was observed; the mice, however, died after cessation of the treatment.

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[8] Lillie, D. R., and Armstrong, C., *Publ. Health Rep.*, 1940, **55**, 718.

Change of Muscular Excitability by Eserine, Acetone and Methyl-alcohol. (17317)

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The potentiation of acetylcholine by eserine, acetone, and methyl alcohol being known, we are interested in finding out their influence on the electrical excitability of the muscle.

The rheobase and chronaxie were determined with condenser discharge. The rectus muscle of the toad was placed in a glass muscle chamber, moistened with Ringer's solution, and observed under dissecting microscope. After the control reading had been taken, the rectus muscle was immersed in a bath of 8 cc of Ringer's solution, bubbled with air, and treated with 10 γ eserine for 30 minutes, or 0.05 cc acetone or methyl-alcohol for 3 minutes, and, in addition, Ringer's solution was alone used without adding any reagent for blank control. Following the treatment, both were redetermined under conditions as before.

TABLE I.
Excitability of Rectus Muscle Increased by Eserine, Acetone, and Methyl Alcohol.

		Rheobase				
		Before treatment		After treatment		
Treatment	No. of observations	Range, v.	Avg, v.	Range, v.	Avg, v.	Difference, v.
Eserine	10	.58- .71	.669 ± .014	.34- .58	.485 ± .024	.184 ± .028
Acetone	10	.65- .78	.717 ± .014	.46- .66	.583 ± .020	.134 ± .024
Methyl-alcohol	10	.65- .77	.705 ± .012	.44- .66	.569 ± .019	.136 ± .022
Ringer's solution (Blank control)	10	.92-1.34	1.106 ± .041	.90-1.33	1.085 ± .044	.021 ± .060

		Chronaxie				
		Before treatment		After treatment		
Treatment	No. of observations	Range, msec.	Avg, msec.	Range, msec.	Avg, msec.	Difference, msec.
Eserine	10	1.30-7.46	3.649 ± .563	1.11- 8.14	4.007 ± .782	.358 ± .964
Acetone	10	.40-8.55	3.086 ± .710	1.54-11.0	3.859 ± .830	.773 ± 1.092
Methyl-alcohol	10	.77-6.22	4.104 ± .535	1.72- 6.75	4.810 ± .532	.706 ± .754
Ringer's solution (Blank control)	10	.87-7.96	4.064 ± .788	1.91- 9.64	5.308 ± .877	1.244 ± 1.187

It was found that rheobase was uniformly decreased by all these agents (Table I), indicating that the excitability of the muscle was definitely increased according to Chao.[1] Similar treatment with Ringer's solution gave no significant change.

The chronaxie showed insignificant change, and we agree with Chao[1] that it is not a valid measure of excitability in such a case.

Summary. With the threshold intensity as a criterion, the excitability of the rectus muscle is definitely increased by eserine, acetone and methyl-alcohol.

1 Chao, I., *The Science Reports of National Tsing Hua Univ. Series B*, **2**, 183.

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Detection of Acrolein by Qualitative Immunochemical Analysis. (17318)

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In a previous report[1] it was shown that acrolein can produce a condition in experimental animals similar in its clinical and pathologic manifestations to that accepted as "shock". The problem of the detection of acrolein in blood or tissue by some specific method next presented itself. Acrolein is found to be a chemically active and highly unstable compound that does not retain its characteristics for any length of time. These two properties alone would make it difficult to detect by the usual qualitative methods of organic analysis, especially when acrolein is present in small quantities. Secondly, as will be shown, there is experimental evidence indicating that acrolein combines directly with protein, and no longer exists in a free state. It thus becomes obvious that the usual qualitative chemical methods of detection cannot be used.

Another possible method for the detection was suggested by a postulated chemical com-

1 Kamen, G. F., PROC. SOC. EXP. BIOL. AND MED., 1943, **52**, 363.