Table 1. Significance of the differences among ECS conditions at the three intensities of foot shock on day 2.

Training-ECS interval (sec)	Training-ECS interval (sec)			
	10	160	640	1600
	Foot shock, 0.3 ma			
No ECS	.01	.07	NS*	NS
10		.05	.05	.05
160			NS	NS
640				NS
	Foot shock, 1.5 ma			
No ECS	.01	.02	NS	NS
10		NS	.01	.01
160			.025	.05
640				NS
	Foot shock, 2.8 ma			
No ECS	.01	NS	NS	NS
10		.05	.01	.01
160			NS	NS
640				NS

* Not significant.

after the response when no FS is given, response latencies on day 2 are shorter ($P < .01$) than those of mice given no FS and no ECS (Fig. 1). (All comparisons reported between two groups were made with median tests.) This result suggests that ECS is not acting as an aversive stimulus in this situation, or latencies of the first group would have been longer than those of the second.

Table 1 contains, for day 2, the significance levels of the differences in response latencies between the ECS conditions for each foot-shock intensity. An ECS delivered 10 seconds after FS disrupted the avoidance response at all three FS intensities. An ECS given 160 seconds after FS disrupted only the groups given 0.3- and 1.5-ma foot shocks whereas an ECS given 640 or 1600 seconds later did not cause a significant disruption in any of the groups. The response latency of the group given 2.8-ma foot shock increased when ECS was delayed 160 seconds, whereas there was no increase in that of the group given a 1.5-ma foot shock until the ECS was delayed 640 seconds. The group given 0.3-ma foot shock never shows a single large increase in response latency. When ECS is delayed 1600 seconds, all FS groups have response latencies very similar to those when no ECS is given. It seems then that, as the FS intensity increased, the interval during which ECS caused a significant disruption of the learned response decreased.

Failure in previous studies (*5*) to find this interaction between FS intensity and effectiveness of ECS in causing performance decrement seems to have been due to the use of only a single short training-ECS interval. Our study shows that, with short training-ECS intervals, ECS is effective in producing performance decrement over a wide range of reinforcement intensities, whereas with long training-ECS intervals only responses followed by low intensities of foot shock are disrupted. This result seems compatible with both the results of Kopp *et al.* (*4*) who used a 0.32-ma, 0.8-second FS and obtained disruption with long training-ECS intervals, and with the data of Chorover and Schiller (*1*) who used a 0.75-ma FS with durations of 0.5 to 4.0 seconds and obtained disruption only at short training-ECS intervals when long FS durations were used.

There seem to be some performance changes which are independent of FS intensity and some which are not. These data may support a two-part theory of memory consolidation. One component of the trace might be based on simple contiguity of the conditioned stimulus and the unconditioned stimulus; this component does not vary with reinforcement magnitude and is not subject to disruption by ECS. The second component of the trace seems to be time dependent (and may be a performance factor) and varies both in size and in resistance to disruption with reinforcement magnitude.

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On the Discovery of Actin

The initial isolation and characterization of actin is commonly ascribed to F. B. Straub (*1*). Actin actually was isolated and characterized under the name "myosin-ferment" 55 years before Straub's studies were published.

W. D. Halliburton, at the time a physician acting as assistant professor of physiology at University College in London, was attempting to extend Kühne's observations on frog muscle proteins (*2*) to those of the rabbit and cat. Halliburton's observations (*3*) included the following comments, captioned "The preparation and properties of myosin-ferment."

> We now turn to the full consideration of the ferment which brings about the coagulation of myosin, and to which allusion has been several times made in the foregoing pages.
>
> I have prepared three specimens in all; and the method of preparation is almost precisely that adopted by Schmidt in the preparation of the fibrin-ferment from blood.
>
> Muscle was first allowed to undergo rigor; it was then chopped up into small pieces and kept under absolute alcohol for a long time.
>
> The first preparation made from cat's muscle was kept under alcohol for ten months; and two preparations from rabbit's muscle were kept under alcohol for three months.
>
> The pieces of muscle after having been thus treated were then dried over sulphuric acid, and powdered. An aqueous extract of this powder contained the myosin-ferment, as shown by the fact that dilution of muscle-plasma, or a solution of myosin with it brought about coagulation much more quickly than dilution with distilled water.
>
> The chemical properties of the aqueous extract were as follows:
>
> 1. Alcohol gave a precipitate soluble in water.
> 2. Boiling gave no precipitate.
> 3. The xanthoproteic reaction showed that a small amount of proteid was present.
> 4. Nitric acid gave a slight precipitate in the cold; this disappeared on boiling, and reappeared on cooling.

This description and other experiments described in Halliburton's long article indicate that he was dealing with the protein now known as actin. It also seems clear that Halliburton was able to distinguish operationally between myosin A (actin-free myosin) and myosin B (natural actomyosin).

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