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Probability of Signal Detection in a Vigilance Task

Abstract. It is hypothesized that the probability of detecting a signal in a vigilance task depends upon its temporal location with respect to the preceding series of signals. Probability of detection should be at a maximum when the signal occurs after a temporal interval which is equivalent to the mean of the intervals between the preceding signals detected. The experimental results support this hypothesis.

In advancing an expectancy theory of vigilance I have hypothesized that "the probability of detection of a signal in a vigilance task is greatest when the signal occurs after an interval which is equivalent to the mean of the inter-signal intervals preceding the interval in question: detection probability is low immediately after a signal, increases as the mean inter-signal interval of the preceding series is approached, and if not reinforced by the occurrence of a signal, again decreases" (*1*). In other words, a peaked symmetrical distribution of detection probabilities is hypothesized.

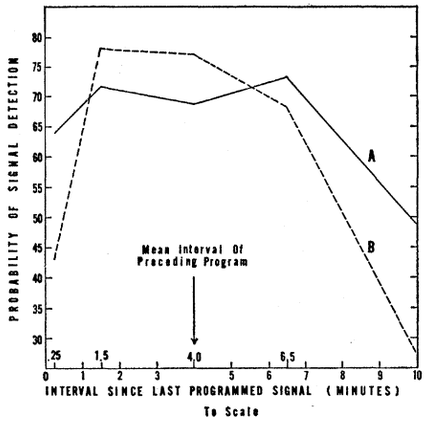

Fig. 1. Probability of signal detection and percentage of signals detected as a function of the length of the interval between the last programmed signal and the "test" signal.

This report describes a study undertaken to verify the hypothesis. The general plan was to expose subjects to a series of eight signals over a period of 32 minutes. The signals were of sufficiently small magnitude to ensure that not all observers would detect all signals. Inter-signal intervals employed were 0.5, 1.5, 2.5, 3.5, 4.5, 5.5, 6.5, and 7.5 minutes. The mean of this series is 4 minutes, though an interval of 4 minutes was not included. Different groups of subjects (simultaneously tested in individual isolation booths) were presented with the intervals in different random orders.

The eight signals of the 32-minute program were followed without a break by a ninth "test" signal which was presented after an interval of 0.25, 1.5, 4.0, 6.5, or 10 minutes, and the percentage of observers detecting the "test" signal was computed.

The test employed was a clock-test. The clocks (one per booth) were electric, 8 inches in diameter, with a black face and a single black second hand having the tip painted white for 1 inch. The hand revolved continuously, once per minute, except when a signal occurred. Signals were defined as 0.30-second stoppages of the clock hand. The subjects' task was to depress a hand-held microswitch whenever a signal was detected.

To test the hypothesis, it was important that subjects not be misled concerning the lengths of the inter-signal intervals but at the same time it was necessary to employ programmed signals of small magnitude in order to encourage missed detections to the subsequent "test" signal. To surmount this problem a small bright light above the clock appeared for a period of 1.0 second immediately following any undetected signal.

Eighty-six subjects, paid housewives, were employed. Each undertook the task twice, each time with a differently randomized program, each program having the "test" signal after a different interval. The total of 172 possible "test" responses is far from ideal but nevertheless served the purpose. The plan called for simultaneous testing of groups of seven subjects but several appointment cancellations at the last moment resulted in an allocation of the 172 possible responses to the "test" signal in the uneven numbers of 39, 28, 38, 26, and 41 to the five "test" signal intervals respectively.

The data in Fig. 1 show probability of signal detection (percentage of signals detected) as a function of the length of interval between the last programmed signal and the "test" signal. Curve *A* represents data from all 86 subjects and consequently includes those who missed none of the programmed signals, that is, those who found the task "easy," as well as those who missed a large number of the programmed signals, that is, found the task "difficult." The drop in detection probability from 6.5 to 10 minutes is significant at the .05 level of confidence, but as a number of subjects are represented at both points this is probably a conservative figure.

Curve *B*, on the other hand, represents data exclusive of those from subjects who missed 0, 1, or 2 programmed signals, and of those from subjects who missed 6 or 7 programmed signals. (No subject missed all 8 signals.) Curve *B* is representative, then, of subjects who missed 3, 4, or 5, or in the region of 50 percent of the programmed signals, and were consequently considered the most sensitive instruments for testing the hypothesis. In addition, any possible correlation effects consequent to plotting two data points for a single subject have been removed: no subject is represented in curve *B* more than once.

It is apparent that curve *B* is grossly representative of the distribution hypothesized. A statistical analysis was undertaken to determine whether the probability of detection at 0.25 and 10 minutes differed significantly from that at 4 minutes. Because of the small sample upon which these three points of curve *B* are based, a total of 36 subjects, the data for 0.25 and 10 minutes were collapsed and chi square computed for the resulting 2 × 2 contingency table, after applying Yates's correction for continuity (*2*). Chi square was computed to be 5.5307, which is significant beyond the .02 point.

While the middle plotted point of curve *B* cannot be considered peaked with respect to those adjacent, the general shape of the complete curve is considered to be in support of the hypothesis.

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Action of 1,1,Dichloro-2-p-chlorophenyl-2-o-chlorophenylethane on Dog Adrenal Cortex

Abstract. A single intravenous injection of *op'*DDD (1,1,dichloro-2-*p*-chlorophenyl-2-*o*-chlorophenylethane) has an acute effect on the adrenal cortex of the dog. Within 2 hours after intravenous injection of the drug, there is a decrease in the in vitro response of the adrenal cortex to stimulation by adrenocorticotrophic hormone and an inhibition of glucose-6-phosphate dehydrogenase activity. The inhibition of glucose-6-phosphate dehydrogenase activity might explain the effect of *op'*DDD on corticosteroid production.

Since Nelson and Woodward reported that DDD [1,1,dichloro-2,2-bis(chlorophenyl) ethane] causes atrophy of the adrenal cortex (*1*), there have been numerous reports dealing with the therapeutic possibilities of this adrenocorticolytic drug. However, the mechanism of action, as far as we know, has not yet been described. We have found that a single injection of *op'*DDD (1,1,dichloro-2-*p*-chlorophenyl-2-*o*-chlorophenylethane) causes a reduction in the response to in vitro stimulation with ACTH (adrenocorticotrophic hormone) and a partial inhibition of adrenal glucose-6-phosphate dehydrogenase. The inhibition of this enzyme suggests one possible mechanism of action for the drug.

Table 1. Effect of *op'*DDD on in vitro response of adrenal gland to ACTH.

Porter and Silber chromogens (μg/100 mg wet weight)		*t*	*p*
Control dogs	Treated dogs		
	No additions		
3.0	1.3		
	5.4		
7.9	3.2		
7.5	1.8		
7.9	1.1		
14.2	5.4		
4.2	1.4		
Mean ± S.E.:			
7.6 ± 1.69	2.8 ± 0.72	2.62	.022
	ACTH (0.2 unit per flask)		
21.1	1.9		
21.1	7.7		
15.1	4.4		
12.3	1.4		
21.4	7.0		
28.9	8.9		
13.5	1.4		
Mean ± S.E.:			
19.0 ± 2.20	4.7 ± 1.21	5.7	< .001

Eighteen mongrel dogs weighing 12 to 15 kg were used; nine were injected with *op'*DDD (*2*) (60 mg/kg body weight), and the other nine received only solvent [6 ml of ethanol and propylene glycol (1:1)]. After 2 hours, the adrenals were removed under pentobarbital anesthesia and cleaned of adherent fat; one adrenal of each dog was sliced, placed in Warburg flasks (40 to 60 mg of tissue per flask), and incubated for 1 hour in 3 ml of Krebs-Ringer solution with bicarbonate. Then the slices were incubated for another hour in a medium of Krebs-Ringer solution, glucose, and bicarbonate with nothing added or with ACTH added (0.2 unit per flask). After this final incubation, the medium was removed and Porter and Silber chromogens were determined (*3*). The activity of glucose-6-phosphate dehydrogenase was determined (*4*) in cell-free extracts prepared from the adrenals that had not been incubated.

The results (Table 1) show that a single intravenous injection of *op'*DDD decreases the in vitro corticosteroid response of the adrenals to ACTH. We feel that this response is a further confirmation of Nichols' work (*5*) and that it is evidence for a specific site of action of *op'*DDD. Table 2 shows that the activity of glucose-6-phosphate dehydrogenase is partially inhibited in dogs injected with *op'*DDD. The activity of 6-phosphogluconic dehydrogenase and the formation of lactic acid were not influenced by *op'*DDD (*6*).

Dogs treated with DDD for 5 days, besides showing the well-established diminution of Porter and Silber chromogens, show a decrease in the urinary excretion of 17-keto-steroids (*7*). A possible interpretation of this decrease is that the biosynthetic pathways of steroids were blocked at an early stage. The inhibition of glucose-6-phosphate dehydrogenase would be a confirmation of this hypothesis, since the inhibition would result in decreased production of reduced triphosphopyridine nucleotide, which is necessary for the breakdown of the cholesterol side chain (*8*). Moreover, glucose-6-phosphate dehydrogenase, which is very active in the adrenal cortex (*9*), is preferentially located in the inner zones (*10*); and it is in these zones that *op'*DDD has most of its effect (*11*).

Table 2. Effect of *op'*DDD on glucose-6-phosphate dehydrogenase activity of adrenal gland. The unit of activity is change in optical density of 0.001 per milligram of nitrogen per minute, at 340 mμ.

Activity		*t*	*p*
Control	*op'*DDD		
844	515		
700	590		
886	400		
565	214		
792	410		
813	473		
913	507		
1275	340		
1287	498		
Mean ± S.E.:			
897 ± 80.4	438 ± 37.3	5.18	< .001

When *op'*DDD was added in vitro to the Warburg flasks, instead of being injected in vivo, it had no effect (*6*). This lack of response in vitro suggests that the *op'*DDD did not reach the intracellular space or that *op'*DDD must be converted into another active product or that the dosage was insufficient (0.8 μmole per flask), because of poor solubility of the drug in the medium. Experiments to elucidate these possibilities are being carried out (*12*).

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