COMPONENT RESPONSE DIMINUTION IN COMPOUND ELECTRODERMAL CONDITIONING: HUMAN SUBJECTS

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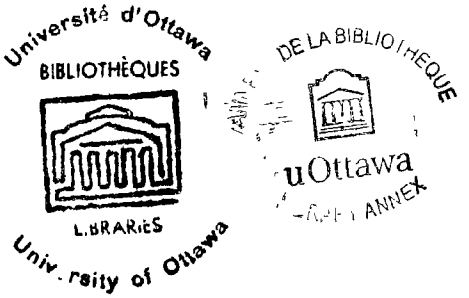
Thesis presented to the School of Graduate Studies of the University of Ottawa as partial fulfillment of the requirements for the degree of Doctor of Philosophy



Ottawa, Canada, 1976

ABSTRACT

In the past, perceptual integration learning in compound conditioning had been inferred from the increased differentiation that occurred between the test responses of the compound CS and component stimuli, with greater amounts of prior compound overtraining. However, when it was demonstrated, in a between-subject experiment, that the component responses also increased with compound overtraining, component CR diminution across independent comparisons became the sole criterion from which component configuring could be inferred. Unfortunately, the only conditioning experiment which had demonstrated component CR diminution according to this standard contained a methodological shortcoming. In that work, the component CR diminution obtained was not without residual conditioned properties, and may have been determined by the disruptive effects of orienting reflexes evoked by the component test stimuli overtrained in compound. Therefore, the present compound conditioning experiment used long delay electrodermal conditioning which provided an opportunity to observe any systematic relationship between orienting reflexes (first interval GSRs) and anticipatory (second interval GSRs) or conditioned responses (third interval GSRs) evoked by a component stimulus in testing. In addition to two experimental groups given different amounts of compound conditioning, a control comparison was employed to assess whether or not the

component stimuli of the overtrained group still possessed conditioned properties. Other experiments which had shown compound-component CR differentiation, in the absence of component CR diminution or while demonstrating an increase in component response magnitudes, failed to test for component conditioned properties, thereby overlooking the crucial test for configurational learning.

Two groups of human subjects received either 10 or 30 compound GSR conditioning trials interpolated between adaptation training and the configurational test phase of experimentation. The control treatment received random presentations with 30 compound and 30 US stimuli. In adaptation training and configural testing, varied schedules containing the compound stimulus (light + tone) and component stimuli (light, tone) were presented and later expressed as two trial blocks, each containing an equal number of compound and component presentations.

The comparison of first interval GSRs revealed no conditioning treatment differences for any stimulus condition in adaptation or configurational testing. Both phases of experimentation showed first interval GSR magnitudes for the compound to exceed the light reactions, which in turn surpassed those for the tone. First interval GSRs for all stimulus conditions in adaptation tended toward habituation in the second of two trial blocks, while in testing the overall

level of responding was higher than in adaptation, and responses here did not decrease over trial blocks.

In adaptation training, no differences for second interval GSRs emerged, but in testing, the compound second interval responses of the experimental treatments were similar in magnitude and surpassed those of the control comparison. Test GSRs for the light component in the undertrained group exceeded both the overtrained second interval responses and the control value, which were equivalent. For the tone component, second interval GSRs were indistinguishable between the experimental and control treatments. The within-group compound versus component comparisons for the experimental subjects were significant in favor of the compound, with the component responses equal. No differences between stimulus conditions were apparent for the control treatment. After the first and second interval GSRs, evoked by the component stimuli, were correlated to determine the existence of a systematic relationship, none was found.

Third interval GSRs for stimulus conditions within or between conditioning treatments failed to differ during the adaptation phase, but overall response levels decreased with trial blocks. In testing, combined stimulus condition responding for the asymptotic and overtrained treatments yielded a conditioning effect within the first trial block, but no differences relative to the control group were apparent by trial block 2. The compound third interval GSRs for both

experimental treatments in testing were similar, but superior to the control standard. The component between-group comparisons contributed no differences. Within experimental groups the compound third interval GSR magnitudes varied significantly above the component GSRs of equal strength. Stimulus conditions within the control group remained equal. In spite of the treatment differences found for compound third responses in testing, statistical comparisons which involved both the adaptation and the test data revealed that such treatment variations did not exceed identical comparisons in the adaptation phase of experimentation.

In all, only the second interval response furnished a clear compound-component differential that could be related to compound overtraining. Since the component second responses were observed not to vary from the control level following overtraining and did not share a systematic relationship with the first response, it was concluded that the second response exhibited configurational learning.

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INTRODUCTION

Simultaneous Compound Conditioning

In Razran's reviews of the Russian literature (1939, 1961, 1965a, 1971), configural conditioning (compound conditioning with overtraining) was operationally defined as the simultaneous or successive presentation of two or more stimuli followed by reinforcement (US). Intermittent test trials allowed the comparison of CRs evoked by the compound CS and component stimuli. These tests have revealed component CR magnitudes to be inferior to the compound CRs and to decrease in strength to "nil" with increased amounts of prior configural overtraining (1971, p. 220). The diminution of component CRs, during simultaneous compound overtraining, has been shown to follow three phases (Razran, 1965a, p. 232): phase I, when CR actions to the components are weaker than CRs to the compound; intermediate phase II, when only the weakest components totally lose their CR effectiveness; final phase III, when all component CRs drop out and only compound CRs remain. Razran (1971, pp. 263-266) interpreted configural conditioning effects (increased compound-component CR differentiation due to component CR diminution during compound conditioning overtraining, in the absence of non-reinforced component training trials) as the manifestation of a rudimentary form of perceptual learning structured upon the sensory integration of component stimuli. In these

experiments, which had used animals as research subjects, it was assumed that the compound CS (configure) was perceived as a new independent entity apart from the embedded components and, as a result, only the compound became the effective CS. According to this view, component CR diminution was the result of sensory integration learning without component contrasting. Three facts supported this assumption: (1) discrimination learning plays no role in configural conditioning effects (see below, p. 7), (2) association areas of the brain are necessary for sensory conditioning (Thompson & Kramer, 1965; Razran, 1971, pp. 200-203), and (3) configural conditioning effects (Razran, 1971, pp. 213-214), but do not support component or discrimination conditioning.

In opposition to Razran, Baker (1968) asserted that component CR diminution was the result of component contrasting and could not be attributed to the emergence of percept learning, or the synthesis of sensory components. He explained the Russian data (Razran, 1939, 1961, 1965a) in terms of the experimental designs used. It was pointed out that the Russian research employed repeated intermittent test trials throughout compound CR acquisition and overtraining, which was tantamount to differential conditioning between the compound and component stimuli. Baker concluded that, if only a single configurational test followed different degrees of prior compound conditioning in a between-subjects design, component CR diminution would not occur. Baker's position

rested upon evidence reported in Baker (1969). In that research two experimental designs were employed to train rabbits on a hopping avoidance task signalled by a 2.0 sec simultaneous compound CS (light + tone). The reinforcement was a 0.1 sec shock which immediately followed CS offset. In the within-subjects design, each animal received 1350 conditioning trials. Following the first 50 acquisition trials on day 1, five blocks of 20 trials each were given per day, and a triad of test presentations containing the compound and component stimuli was interspersed within each succeeding block. In this situation, results identical to the Russians' were obtained. The same experiment was also arranged in a between-subjects design which contained different levels of compound training with 0, 100, 400, 1300 conditioning trials. One hundred trials a day were given to each group up to the appropriate trial limits, save condition 0. Twenty-four hours thereafter, each level of training was divided into three subgroups to provide four sets of independent compound-component CR comparisons. All subgroups then received 50 additional compound conditioning trials immediately followed by two days of extinction tests with either the compound CS, or component stimuli. With this design, both the compound and component test CRs increased as a function of prior training, with the compound CRs becoming increasingly more frequent than component CRs

across the different levels of prior compound conditioning. In spite of a greater compound-component CR differentiation with increased amounts of prior training, Baker's results still offered strong evidence against Razran's configural hypothesis. According to the configurational explanation, component CR diminution should have occurred across groups given increasing amounts of prior configural training, but Baker's data showed an increase in component CR strength. Baker concluded that compound-component CR test differences in the between-subjects design were the result of a generalization decrement between training and testing conditions, and that component CR diminution only occurred with repeated intermittent test trials or differential conditioning.

In addition to Baker's work, Wickens, Nield, Tuber and Wickens (1970) contrasted the degree of compound-component differentiation in cats following compound classical conditioning with or without intermittent component test trials. Two groups of subjects were presented with a simultaneous compound CS (light + tone) 2.0 sec in duration. The US onset occurred immediately after CS offset and consisted of a 60.0 msec paw shock. One set of subjects received 12 replications of a standard training schedule with 36 compound conditioning trials and 21 nonreinforced (intermittent) test trials. Each replication took five successive days to complete. The first four days each contained 9 conditioning trials and 3 interspersed test trials with the compound and

component stimuli. Day 5 terminated each replication and was composed of 9 test trials, 3 for each kind of presentation (compound and component stimuli). Two days separated the replications, and the order of test trials was counterbalanced across all subjects. A second set of subjects was presented with 13 similar replications but with one variation, now all interspersed test trials would contain the compound CS. Following the training replications, six days of extinction testing were given to those subjects who had received component test trials. In extinction testing, 9 nonreinforced trials were presented daily, 3 for each stimulus condition. Subjects, who had not received component test trials during training, were given five days of identical configural testing.

An analysis of the percentage of CRs evoked by the compound and component stimuli during interspersed test trials revealed a clear compound-component differentiation after 7 replications of training. However, the frequency of component CRs across replications 8 through 12 did not decrease. When responses in extinction were used to evaluate compound-component differentiation, subjects given interspersed component tests in training, yielded more CRs to the compound than to component stimuli. For subjects given no component test trials during conditioning (only nonreinforced compound presentations), compound-component

discrimination in extinction was marginal. Even though no component CR diminution occurred across nonreinforced component trials during compound conditioning, these investigators held that compound CR dominance reflected configural conditioning and not the summation of two component CRs. In any case, although Baker found compound CR dominance with or without component testing in conditioning, the greater degree of compound-component differentiation with component contrasting is at least in line with Baker's position.

A similar failure to obtain compound-component discrimination, with only compound test presentations amidst compound conditioning trials, has also been reported by Morrow, Seiffert and Kramer (1970). In a GSR experiment, human subjects were given a 0.1 sec shock to the fingertips 0.1 sec prior to the offset of a 0.6 sec simultaneous compound CS (light + tone). Each subject accepted five days of compound conditioning. Sixteen conditioning trials for each day were interspersed with 4 compound test presentations, while another such test trial always preceded the first conditioning trial. On day 6 the conditioned subjects received 2 component test trials just prior to 18 compound trials in extinction. For the component tests, a light then tone were presented to half of the subjects, while the reamining half received the reverse trial sequence. An explicitly unpaired compound/US condition furnished control

comparisons. Although the primary goal of this experiment was to assess the compound CRs (GSRs) against control equivalents, only the analyses of test trial difference scores between the compound and component GSRs of the experimental and control groups need concern us here. Three analyses of difference scores (compound stimuli minus component stimuli) were carried out. The first consisted of differences between the first elemental trial on day 6 and the initial compound trial on day 5. In the second, the mean magnitudes of component trials were subtracted from the last compound test trial on day 5. For the third, the mean magnitude of the component trials was subtracted from the mean magnitude of the first two compound trials on day 6. In the end, only the first analysis indicated compound CR dominance and therefore the attainment of configurational conditioning was denied.

Razran (1971, p. 215) argued on logical and empirical grounds that the configural effect (component CR diminution) reviewed in his work could not have been the result of discrimination learning between compound and component stimuli. He made the following points summarized below:

1. Compound and component stimuli share identical neural events and that differences in stimulus value between compound and component test trials are unnecessary for the production of the configural effect. (Increasing component intensities during testing has no effect.)

2. Animals with lesions that abolish the configural effect are not impaired with regard to component or differential conditioning.

3. Animals below the phylogenetic levels of birds and mammals are not capable of acquiring configural conditioning, while discrimination learning is well within their capacity.

4. Configural conditioning takes many more trials than simple differential conditioning.

5. Differential contrasting between compound and component stimuli during configural conditioning yields a negligible advantage.

6. Unreinforced intermittent test trials during configural conditioning are far too few to explain the dramatic diminution of component CRs, given the high resistance to extinction of conventionally overtrained conditioned stimuli.

Two recent experiments conducted in the United States, using between-subject designs (no intermittent component test trials during compound conditioning), provided convincing evidence for Razran's configurational theory. Guth (1967), in the first phase of his experiment, trained rats to bar press for water during a simultaneous compound Sd (a + b, or light + tone). These subjects were then given extinction training with component a during a second phase. In the final phase of the experiment, extinction tests were given to independent subgroups with either the compound Sda+b or component b. Those subjects who had received the light as component a during extinction training were given the tone for component b in extinction testing and vice versa. This allowed for two independent operant response

comparisons between Sd$\underline{a+b}$ versus $\underline{b}$. In addition, Guth trained a control group to press for water when both components $\underline{a}$ and $\underline{b}$ were presented separately as Sd_1 and Sd_2. Appropriate subgroups were then given extinction training and testing as above. During acquisition, all Sds ($\underline{a+b}$, $\underline{a}$, $\underline{b}$) were equated for the duration of stimulus exposure and amount of reinforcement. Therefore, in acquisition sessions of equal length, Sd$\underline{a+b}$ presentations were separated by SΔ periods, but no such periods intervened between Sd$\underline{a}$ and Sd$\underline{b}$ onsets. During extinction testing, subjects previously trained with the simultaneous Sd, gave significantly higher response rates to the compound $\underline{a+b}$ than to component $\underline{b}$. In the successive Sd condition, no differences between the compound and component subgroups were noted. The author concluded that the $\underline{a+b}$ versus $\underline{b}$ test differential for the simultaneous Sd condition could not have been the result of a nonintegrated summation of the two components. He reasoned, that if this were so, the successive Sd treatment should also have shown the same $\underline{a+b}$ versus $\underline{b}$ subgroup test differential as both acquisition conditions were equated for response strength to the component Sds. Therefore, it was assumed that a configure (Sd$\underline{a+b}$) must have been linked to bar pressing in the simultaneous condition, and extinction training to $\underline{a}$ could not weaken the response strength attached to this Sd.

To date, Booth and Hammond (1971) have produced the strongest support for the configurational theory. In their study, rats were trained to bar press for water. Following acquisition, three independent subgroups (4T, 12T, 40T) had either 4, 12, or 40 CS-shock pairings applied for bar-press suppression training. The CS was a 3 min simultaneous compound stimulus (flashing light + tone) which terminated with a 1.0 sec foot shock. Four CS-shock presentations were given per day, and so group 4T received one day of suppression training, group 12T three days, and group 40T ten days. At the end of conditioning, configural testing began. During this phase of the experiment, each subject received triads of nonreinforced trials (no shock), which contained randomized permutations of the compound and component stimuli. Each stimulus type was deleted upon the attainment of an extinction criterion for par-press suppression. As part of the data analysis, compound and component suppression ratios $\left(\frac{B}{A+B}\right)$ were reported for the first two triads in testing. For the second test triad a comparison of compound and component ratios within groups 4T and 12T failed to uncover any differences. But in the overtrained treatment (40T), the compound stimulus impeded responding to a greater extent than the component stimuli. When each stimulus condition was examined between groups 12T and 40T, the component stimuli for the overtrained treatment effected significantly less

suppression than those of treatment 12T. Compound stimulus performance levels were indistinguishable. Statistical tests for configuring within triad 1 differed from those of triad 2, in that the tone exhibited less suppression than the compound stimulus in group 12T and comparable suppression to the compound stimulus in group 40T. Finally, the degree of suppression to the tone did not differ across groups 12T and 40T. However, when an additional group of subjects was given 60 trials of compound suppression training, compound-component differentiation was complete in both test sequences (Razran's personal communication with the authors, p. 220, 1971).[1] These measures indicated that compound overtraining alone could produce compound-component differentiation with component CR diminution.

Serial Compound Conditioning

In serial configural conditioning, the compound CS was composed of two (or more) nonoverlapping stimuli presented in immediate succession. Reinforcement (US) coincided with the termination of the second component. As with simultaneous configural conditioning, Razran (1965a, p. 235) outlined three phases of development associated with the acquisition of a classically conditioned configure (two components only):

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1 This experiment has been successfully replicated by Dr. T. Gray of Sir George Williams University. Personal communication with Dr. Jane Stewart, Chairman, August 1974.

phase 1, when only CR reactions to the second component are evident; phase 2, when the first component evokes only orienting reactions and the second the CR; phase 3, when CRs to both CS components are active and equal but CRs to the second in compound are enhanced compared to CS_2 test responses (CS_1-CS_2 CRs = CS_1CR > CS_2CR). Razran (1965a, p. 235) construed superior CS_2 responses during compound trials to be the result of sensory integration learning (configuring). CS_1 was alleged to elicit the sensory reactions of CS_2 prior to CS_2 onset. Nevertheless, the Russian phases of configural CR development were based on experiments which had employed an unavoidable shock US and a relatively long compound CS. In connection with this circumstance, Baker (1968, pp. 616-617) cited evidence (Baru 1962 translation) which demonstrated that in any component conditioning situation, involving a long delay CS followed by an unavoidable shock US, the resulting CR latencies would grow progressively shorter until all CRs fell within the first portion of the CS. This same shift in CR latency became evident in the third phase of configural conditioning. These facts led Baker (1968) to propose that the phases of configural conditioning might simply reflect a long delay CS effect, not configuring. Based on this assumption, Baker deduced that in any serial compound conditioning experiment, "it appears possible to predict which component of a successive compound will be effective from the latency data for that situation using a single CS (p. 616)."

Guided by this conclusion, Baker (1968) juxtaposed the serial compound conditioning literature with studies that had compared the CRs of similarly extended compound and component stimuli. A related review of the serial compound literature follows.

Sergeev (1961) classically conditioned two dogs with six serial compound stimuli to each. All compound stimuli were composed of an auditory stimulus as CS_1 (one of three tones or one of three metronome rates) and a succeeding cutaneous stimulus for CS_2 (one of three cutaneous vibrators at different body locations). CS_1 and CS_2 had durations of 10.0 sec and together formed a serial compound CS that spanned 20.0 sec. All six compound CSs were presented in random order throughout any one conditioning session and food reinforcements immediately followed CS_2 offsets. One hundred and eighty to 210 conditioning trials were given with each compound CS, and only isolated test trials (one or two per month) with CS_1 and CS_2 were interspersed during acquisition. Each test stimulus had a changed duration of 20.0 sec. Following acquisition, extinction training with the compound CSs began and during the different stages of extinction identical test trials to those above were again interspersed. Test trial results during acquisition indicated that CS_1 evoked 30 - 40% of the mean magnitude for salivations to the compounds, while CS_2 from 70 - 100%. Test trials during extinction showed that CS_2 responses

manifested inhibition prior to CS_1 salivary reactions and remained weaker throughout extinction. Component test trials during an additional compound conditioning re-acquisition period yielded similar results to those found during compound extinction, namely, CS_1 reconditioned first and then CS_2.

Williams (1965) contrasted three classical conditioning treatments using a within-subjects design and four dogs. Each animal was simultaneously conditioned with a serial compound CS, a short delay CS, and a long delay CS. All stimulus conditions contained 22.0 secs and were considered to have first and second interval periods. The first interval periods were 16.0 sec in length and the second interval periods 6.0 sec. For the serial compound condition, CS_1 filled the first interval, while CS_2 occupied the second. In the short delay treatment, the first interval was vacant, but the second held CS_2. The first and second interval periods for the long delay CS condition were possessed by a third stimulus, CS_3. The conditional stimuli (CS_1 CS_2 CS_3) between dogs had varying identities (either a steady tone, intermittent tone, clicking sound), but CS_2 in the serial and short delay treatment always shared the same stimulus. During the experiment, this organization allowed the subjects to discriminate between the stimulus treatments. All subjects received salivary conditioning for the CS types with the US (a food pellet) presented within the last

two seconds of the second intervals. During conditioning tests, each animal was exposed to only two stimulus conditions within a single session. Two subjects had their serial and short delay CRs reported, while the responses of the third formed a serial compound versus long delay comparison. The CRs of the last dog provided all of these measures. Although the fourth animal had its short and long delay CRs expressed, their relationship was not emphasized. Examination of the first and second interval portions of the serial compound and long delay conditions showed that the second intervals held greater CRs in terms of salivation rates. Both stimulus situations elicited greater CRs than the short delay treatment with respect to the first interval period, but the long delay CRs exceeded those of the serial condition. However, the second component of the serial compound evoked superior CRs relative to those in the corresponding intervals of the short and long delay treatments. Added to Williams' analyses was the observation that in one dog, with the long delay versus short delay comparison available, greater CRs were found in the short delay second interval than in the comparable period of the long delay treatment. Tsi-Tsiao (1959) confirmed this relationship in a within-subjects experiment when the degree of salivation in dogs was compared between a 15.0 sec short delay CS condition and the compatible 15.0 sec period of a 60.0 sec long delay CS treatment.

Taken with Sergeev's test trial results during acquisition, the greater CR magnitudes during the second interval periods of the serial and long delay conditions would appear to support Baker's contention that serial conditioning was a simple extension of long delay component conditioning (Pavlov, 1927, Lecture VI). Williams, in interpreting his data, rejected such a position on the grounds that the first interval response of the serial condition was depressed and the second enhanced relative to the same periods in the long delay treatment. Instead, he related the superior serial CRs in the second interval to a positive induction effect, or the sudden release of excitation upon CS_1 offset. Contrary to Williams, Baker (1968) insisted that the enhanced second interval CRs in the serial treatment were simply due to a greater separation between the inhibition of delay and pursuant excitation. If the response magnitudes of the short delay stimulus are considered, the superiority of the second interval responses in the serial condition are consistent with Williams' view. This same result also supported the third stage of configural conditioning (CS_1 producing the sensory effects of CS_2); but within the serial treatment inferior first interval responses relative to those of the second run counter to it. Conversely, the dominance of CS_2 responses in Sergeev's test results during acquisition would seem to go against phase 3 of configuring, since CS_1 should

have evoked the sensory reactions of the compound to a far greater extent than CS_2. By the same reasoning, the greater response strength of CS_1 during compound extinction (at least in its early stages) was consistent with it. However, any evidence taken from Sergeev's work must be viewed with caution as no controls were included to cancel the possible influence of stimulus modality during conditioning.

The most adequate complement of evidence for the configurational perspective of serial compound conditioning can be drawn from a three-part experiment on compound conditioned reinforcement by Thomas, Berman, Serednesky and Lyons (1968). In experiment II, a serial two component visual compound (disc-projected green light and line stimuli balanced across S_1-S_2) was presented to pigeons just prior to a single food reinforcement earned with each disc peck (2050 food reinforcements dispensed on a CRF schedule). The compound conditioned reinforcer was 2.0 sec in length and consisted of components 1.0 sec long. During 16 days of disc pecking performance each subject received conditioned reinforcement test sessions after 50, 650, 1250, and 2050 compound-food pairings. In testing, the subjects were required to disc peck for component presentations having durations of 1.0 sec and compound presentations which now consisted of a 0.5 sec exposure of S_1 immediately followed by a 0.5 sec exposure of S_2. Upon the completion of the experiment it was found that only in the last test segment did the compound support more disc pecks

than either of the components which did not differ. More importantly, it was demonstrated that S_1 only acquired conditioned reinforcement properties after S_2, but both component stimuli declined in strength with increased conditioning. To evaluate the effects of continuous non-reinforcement to the components (compound versus component contrasting) and the differences in stimulus value between compound and component test presentations, a third experiment was performed. In experiment III, the basic conditioned reinforcement procedure was maintained, but important variations were added. During conditioning, a two component visual compound (two line stimuli at 90° and 60°) was reinforced on half of its presentations (2130 reinforced presentations out of 4185) and only a single conditioned reinforcement test was given upon the completion of training. In training and testing, the compound conditioned reinforcer (S_1-S_2) filled 1.0 sec with the components enduring for 0.5 sec each, but the isolated component reinforcers (S_1, S_2) had durations of 1.0 sec each when tested in the final stage of the experiment. Two additional control compounds were included in testing. One was a reversed sequence (S_2-S_1) and in the other S_1 was followed by a novel stimulus (S_1-S_N). As earlier, the compound conditioned reinforcer (S_1-S_2) supported significantly more disc pecks in testing than all other control stimuli, whose response values were equivalent. The compound (S_1-S_2) versus component and control sequence differentials in experiment III agreed with the configurational

theory, since these differences could not be attributed to variations in stimulus value (i.e., S_1-S_2>S_2-S_1 = S_1 = S_2), nor component contrasting during conditioning. Projected against this evidence, the initial acquisition of conditioned properties by S_2 with the subsequent attainment by S_1 (in experiment II) underscored the reliability of the first two Russian phases of serial compound conditioning and their claimed relation to configurational learning. Nevertheless, the equality of component test performances (S_1 = S_2) was not consistent with the Russian third phase of serial configuring. The level of performance for S_1 should have approximated that of the compound and hence excelled S_2. However, component CR equality and the diminution of component performance levels observed in experiment II resembled the Russian's third stage of simultaneous configural conditioning.

Baker (1969) denied that configuring had occured in Thomas et al. (1968) because of the compound-component performance differential in experiment II being greater than in experiment III, despite 2050 compound presentations in the former and 4185 in the latter (2130 of which were reinforced). Perhaps Baker was focusing on Razran's belief that the sensory integration within configural conditioning was the same as in perceptual learning external to it. Regardless, the absolute levels of component performance in experiment III were less than in experiment II and, as Baker mentioned, the extent of

discrimination between the compound and component stimuli in experiments II and III may be linked to the changes in stimulus quality or reinforcement schedule. At this point, Baker (1972) provided the literature with an experiment similar to Thomas et al. (1968), but couched in a between-subjects design. Included were serial compound conditions with partially overlapping components, and therefore, this work is outlined below in the conclusion of the next section.

Serial Compound Conditioning with Overlapping Components

To investigate the degree of component CR strength that would accrue during compound classical conditioning, Wickens (1959) conditioned nine groups of human subjects in a GSR experiment. Throughout compound conditioning acquisition, CS_1 preceded, overlapped, and then coterminated with CS_2, just prior to the onset of a 100.0 msec shock UCS. CS_2 had a duration of 500.0 msec in all stimulus conditions, whereas CS_1 preceded CS_2 by either 0, 100, 200, 300, 400, 500, 1000, 2000, or 4100 msec. After ten compound conditioning trials, half of the subjects from each stimulus condition were given component test trials with the long stimulus, while the remaining halves received the short component. In conditioning, a light and a tone were counterbalanced within each treatment, such that in testing the component stimulus was always the tone. To analyse compound conditioning effects on

the test CRs of the long stimuli, control groups for each CS_1 duration were exposed to ten component conditioning trials (with the tone) prior to testing. Wickens found that during component testing, CS_1 evoked increasingly greater CRs from the 100.0 msec precedent treatment through to the 400.0 msec condition, but thereafter the CRs declined. The identical short duration stimuli (CS_2) produced CRs which waned in strength across the same experimental groups, but then these CRs increased. In the 100.0 msec and 1000.0 msec conditions, CS_2 responses were greater than those of CS_1, but in the 400.0 msec treatment this relationship was reversed. When both types of component reactions were plotted against their corresponding precedent conditions, their curves portrayed mirror images of each other. Control component CRs were similar to CS_2 responses in that they decreased in magnitude across comparable precedent conditions. Wickens, Geham and Sullivan (1959) reported essentially the same pattern of GSRs following an identical experiment, except for slight changes in the CS_2 precedent conditions. In testing, the long component CRs ascended throughout the 100.0, 200.0, 300.0, 420.0 msec interval conditions, while CS_2 responses descended inversely. As before, CS_1 control GSRs declined from the 100.0 msec group to a 540.0 msec interval condition. In addition to human subjects, Wickens, Born and Wickens (1963) extended the use of this kind of

experiment to cats, but with a repeated measures design. Six subjects were given GSR conditioning with three compound stimuli having CS_1 preceding CS_2 by either 150.0, 500.0, or 700.0 msec. CS_2 was always 600.0 msec in length, and a 50.0 msec paw shock served as the US. All stimuli (light, tone, US) terminated together. Over the course of three weeks, all animals received one week of compound conditioning with testing for each of the three stimulus conditions. Throughout the first three days of a single week, 30 conditioning trials (10 per day) were interspersed with four test trials to the compound and component stimuli (12 in all). On days 4 and 5, four test trials were given with each kind of stimulation (24 in all). Modalities of stimulation and order of test trial presentations were equated for each interstimulus condition, along with the order of conditioning for stimulus treatments. Only the test data from days 4 and 5 were analysed and the outcome was representative of the equivalent human research. The short test stimulus of the 150.0 and 700.0 msec precedent intervals elicited significantly greater GSRs relative to the corresponding long components, but as anticipated, the reverse was true for the 500.0 msec precedent condition. Compound stimulus responses for each precedent treatment did not differ from each other or from the dominant component response within each condition. Inferior component CRs in each stimulus condition did vary significantly from the appropriate compound GSRs. Here, and in other studies

(Wickens, 1959; Wickens et al., 1959; Wickens, 1965), the preponderant CS_2 responses (i.e., in the 150.0 and 700.0 msec levels) were assumed by the authors to be the result of component conditioning effects similar to those revealed by the CS_1 control groups in Wickens (1959) and Wickens et al. (1959). In those studies a single stimulus acquired a greater response strength when conditioned with a CS-US delay interval of 500.0 msec (i.e., point of 0, precedence) than with others having smaller or larger values. Contrary to these observations, CS_1 produced superior CRs to CS_2 (and CS_1 control stimuli in Wickens, 1959; Wickens et al., 1959) after these components had been presented in compound with a 500.0 msec CS_2 precedence. In this experimental condition, it was postulated that sensory conditioning between the component stimuli had emerged, and that an internalized representation of the stimulus complex had become the effective CS. During conditioning it was now assumed that CS_1 acquired the capacity to evoke the internalized complex and, therefore, the "compound" CR. CS_2 was thought to lack this ability due to its backward relation to the internalized sensory sequence. In this way the dominant responses associated with CS_1 in the 500.0 msec condition (and the 400.0 msec treatment of Wickens, 1959) could be accepted even though this result went against the previous component control data. At this point, it is appropriate to note that the component reactions associated with sensory conditioning were in perfect agreement with the

Russian construction regarding the third phase serial configuring (i.e., CS_1-CS_2CRs = CS_1CR>CS_2CR).

To ensure that the GSR findings above were not the result of CS_2 precedent interval effects on component disparity reactions (Grings, 1969) that might have occurred in testing, Allen, Hill and Wickens (1963) converted the previous designs into a habituation experiment through the use of a neon light as a surrogate US. The CS_2 precedent conditions became 150.0, 450.0, and 700.0 msec to maximize treatment differences, but all other aspects remained the same as in Wickens (1959) and Wickens et al. (1959). It was reasoned that if the conditioning process in past works was not related to component test differences, similar results might occur in post-habituation tests with the component stimuli. Following habituation training and component testing in this experiment, GSR values for each subject's last two acquisition trials were expressed as a $\bar{X}$ score and these were then subtracted from a subject's component test responses. Difference scores demonstrated that the long component manifested greater ORs in the 150.0 msec treatment, while the short component yielded superior ORs in the 450.0 msec condition. No component differences were noted for the 700.0 msec group. From this experiment it seemed clear that disparity reactions to the component stimuli did occur, but they were in the opposite direction to the component CRs observed in the conditioning experiments. Therefore, it

was concluded that the precedence conditions act on both the disparity reactions and component CRs, but in conditioning research, the conditioning effects dominate. Additional evidence against a disparity reaction interpretation of Wickens' compound conditioning data is contained in Wickens' latest effort in this area (Wickens, 1973). In this experiment, a paw withdrawal response in cats, known to be insensitive to orienting behavior, was conditioned to three compound stimuli of the CS_2 precedent variety. Component tests which followed overtraining (12 trials per day for 42 days) revealed identical results to those found in prior GSR experiments.

Since Wickens' first investigations with partially overlapping compound stimuli, other researchers have followed his lead, but instrumental responses were used to demonstrate CS_1-CS_2 interval effects. Baker and Schoeninger (1969) conditioned rabbits to perform a hopping response to avoid foot shock. The avoidance CS was a compound stimulus in which a tone always preceded, overlapped, and terminated with a 2.0 sec light. The US immediately followed the compound CS. Independent groups of subjects were assigned to one of six CS_2 precedence conditions with values of 0.0, 0.5, 1.0, 2.0, 4.0, and 8.0 sec. One of three replications run with this design was conditioned with a distributed training schedule of 20 trials per day to an avoidance criterion. Twenty-four hours thereafter half of the subjects in each precedent

treatment were given 20 extinction trials a day to criterion with CS_1. The remaining halves received CS_2. Upon the attainment of extinction criterion, each half was then tested in extinction with the other component stimulus. The second replication was treated the same as the first, with the exception that all subjects received extinction testing with CS_1 first, then CS_2. A third replication was trained to avoidance criterion with a massed conditioning schedule containing 100 trials per day. Immediately after avoidance training, this set of subjects was extinguished with CS_1 and then CS_2 within a single test session. Therefore, in this experiment, training schedule and intersession delay between conditioning and extinction were confounded. After the first and second replications were pooled (with CS_1 responses corrected for order of testing in half the subjects of replication I), analysis of the number of CS_1 avoidance responses in the first 20 extinction trials showed that the 4.0 sec precedent treatment yielded a greater resistance to extinction relative to the 0.0 and 1.0 sec groups. Conditions 0.5, 2.0, and 8.0 sec responded less than treatment 4.0 sec, but not by a significant margin. In the third replication, it was the 0.5 sec treatment that produced significantly more CS_1 responses in extinction than any of the other groups which did not differ. A significant replication X precedent interval interaction disclosed that, for the 0.5 sec precedent condition, the third replication had a greater response

frequency in extinction when compared to the pooled replication. Within the 4.0 sec precedent treatment, the pooled replication exhibited more avoidance responses than the third. No other differences were noted between the sets of subjects. To explain these results, Baker, guided by Wickens (1959), hypothesized that CS_1 in the 0.5 sec precedence treatment for either schedule drew the greatest response strength through sensory conditioning. This was born out in part by the immediate extinction tests given to subjects treated with the massed schedule. The failure of subjects who were trained with the distributed schedule to show this result was related to the effects of the 24-hour delay between training and extinction testing. During this time, Baker suggested that the weak CS_1-CS_2 links of the 4.0 sec group increased while the stronger ones of the 0.5 sec condition decreased to inferior levels. According to this rationale, it only appeared that with the 24-hour test delay the best stimulus control for CS_1 (through sensory conditioning) had occured in the 4.0 sec interval condition.

Confirmation that the CS_1-CS_2 intervals in Baker's study had, in fact, determined the acquisition of varying levels of sensory conditioning (and hence CS_1 efficacy) came from two earlier experiments on sensory preconditioning. Both utilized the same kind of compound stimuli for preconditioning as those employed by the previous compound conditioning research. For preconditioning, Hoffeld, Thompson and Brogden

(1958) presented groups of cats with compound stimuli having CS_2 precedent intervals of 0.0, 0.5, 1.2, 2.0, and 4.0 sec. Following component conditioning with CS_2, sensory preconditioning tests with CS_1 were given. Similarly, Wickens and Cross (1963) trained and tested groups of human subjects. Their CS_2 precedent treatments during preconditioning were 0.0, 100.0, 400.0, and 600.0 msec. CS_1 tests showed that in Hoffeld et al. (1958) preconditioning in the 4.0 sec treatment surpassed all others, while in Wickens et al. (1963), it was the 400.0 msec condition which excelled. Since the latter research occurred in a massed practice situation (immediate CS_1 testing) and the former with distributed sessions (24-hour CS_1 testing), these data fit with Baker's.

Egger and Miller (1962) conditioned two groups of rats (A, B) using the identical compound CS. The durations of CS_1 and CS_2 were 2.0 sec and 1.5 sec, respectively. CS_1 preceded, overlapped, and coterminated with CS_2. Food served as the US and was delivered 0.25 sec prior to the offset of the compound CS. All subjects were presented with 135 conditioning trials over a period of five days, but group B received an additional 110 nonreinforced trials with CS_1 during the training period. The authors predicted that in condition A, CS_1 would acquire a greater response strength than CS_2 even though both component stimuli signalled the US. It was assumed that CS_2 possessed redundant information and would condition less than CS_1. Conversely,

it was anticipated that CS_2 within treatment B would control responding to a greater extent than CS_1. This expectation was based on the presumption that CS_2 was the only reliable predictor of the US in treatment B, as CS_1 would have been partially reinforced. In order to observe differences in component stimulus control 24 hours following compound conditioning, each component of the compound CS was presented as a conditioned reinforcer for a previously established bar pressing response. Brief bar press extinction periods preceded the component test sessions which were balanced between all subjects for type of component test presentation. All conditioned reinforcements were 1.0 sec in duration. The conditioned reinforcement test sessions showed that when bar press performances were compared within each experimental condition, CS_1 supported more responses than CS_2, provided that no nonreinforced CS_1 presentations were administered during conditioning. Independent group comparisons disclosed that treatment A emitted more test responses for CS_1, while the opposite was true for CS_2. When the dominant CS_1 test performance in A was compared with the dominant CS_2 test performance in B, CS_1 was found to be more effective than CS_2. Pseudo-conditioning control conditions, designed to resemble treatments A and B, did not yield any differences within or between groups. When these pseudo-conditioning groups were pooled and compared to treatments A and B, CS_1 and CS_2 control performance levels only fell

short of the CS_1 response level in treatment A. Based on the greater CS_2 reinforcement efficacy for group B relative to A, it was concluded that, in treatment A, the enhanced responses associated with CS_1 were the result of this stimulus acquiring the most information value during compound conditioning. The reliability of these comparisons were corroborated in a similar experiment by Egger and Miller (1963). Seligman (1966) replicated the work of Egger and Miller (1962) using a bar press suppression experiment which employed conditioned reinforcers as punishment. Initially, all subjects were trained to bar press for food pellets on a V.I.-1 schedule. Twenty-four hours after the stabilization of bar pressing, the lever was retracted and the subjects were then divided into three experimental treatments for Pavlovian fear conditioning. All conditions received the identical compound CS as in Egger and Miller (1962), but instead of appetitive reinforcement, a 0.5 sec foot shock was the US. Two experimental conditions were the same as treatments A and B above, in that both were presented with 10 compound conditioning trials, but B also received 10 nonreinforced trials with CS_1. The third, an explicitly unpaired control, contained 10 compound CS and 10 shock exposures. The day after conditioning, the bar was replaced and appetitive V.I.-1 responding was re-established. Following a brief warm-up period, half of the subjects in each group were then tested with CS_1 as an aversive reinforcer contingent

upon each bar press. The remaining halves pressed for CS_2. Each conditioned reinforcement was 1.5 sec in length. Bar presses occurring within the first five minutes following the initial conditioned reinforcement were expressed as a percentage of the last five minutes of warm-up responding. This constituted a measure of bar press suppression. Contrary to Egger and Miller, both experimental treatments manifested greater suppression than the control condition regardless of the type of aversive reinforcement. As expected, CS_1 in treatment A effected more suppression than CS_2 in treatment A, and CS_1 of treatment B. Although these results supported the informational hypothesis, CS_2 in treatment A impeded bar press behavior to a greater extent than in treatment B. Further, bar press performance related to CS_2 did not differ from that of CS_1 when subjects were pretreated with treatment B. These latter findings went against the informational hypothesis and would preclude its generalizability to situations in which appetitive and aversive reinforcements are used during different stages of training.

In a similar effort to examine the dynamics of component CRs in compound conditioning, Baker (1972) conditioned three independent groups of rats using a serial compound CS (seq. condition) and two variations (OL conditions) of a serial compound CS with partially overlapping components. All compound stimuli lasted for 3.0 sec, and in the overlapping treatments, the components terminated together. Both

components of the serial compound CS had durations of 1.5 sec. For one overlapping compound condition (OL, 2.5), CS_1 had a duration of 3.0 sec, and CS_2, 2.5 sec, while in the other (OL, 1.5) CS_1 endured for 3.0 sec, and CS_2, 1.5 sec. White noise and the simultaneous onset of a white, red, and green light were the component stimuli; and complete counterbalancing was implemented for component order within each compound. The USs consisted of food pellets which were dispensed upon the completion of compound CS exposures. Each stimulus treatment was partitioned into four independent subgroups (12 in all) which received either 50, 150, 450, or 900 CS-US pairings. Throughout conditioning, 25 conditioning trials were given per day in the absence of a response lever. On days following the completion of the appropriate amounts of classical pairings, each stimulus treatment had a previously acquired bar pressing response re-established to furnish a warm-up period prior to component testing. After 10 minutes of warm-up bar pressing, food reinforcements were discontinued and replaced by the component stimuli, which were then presented to each subject as conditioned reinforcers in one of two test segments of five minutes each. Kind of conditioned reinforcement was equated across the test segments. During testing, the durations of the components were 1.5 sec and amount of bar presses for them provided an index of CR strength. Prior to a final analysis of the data, scores for each component stimulus were

transformed according to a mathematical model designed to separate component effects from those of the test segments. A previous and conventional analysis showed that these variables had interacted dramatically. The analysis of transformed scores is outlined below (Figure 1). Of the 50 trial subgroups, CS_1 in the OL, 1.5 condition drew significantly more responses than CS_2, but CS_2 was the dominant secondary reinforcer in the seq. treatment. No component differences were apparent for subjects who had received the OL, 2.5 compound CS. All 150 trial subgroups yielded more responses for CS_1 than for CS_2, but by insignificant amounts. However, Baker treated the $CS_1 > CS_2$ difference in the OL, 2.5 condition as significant on the basis of this comparison being highly similar in magnitude and direction to another which was significant in a twin within-subjects experiment (having a smaller error variance). Conversely, the 450 trial subgroups exhibited greater performance levels for CS_2, but again by insignificant degrees. As before, $CS_2 > CS_1$ differences in treatments OL, 1.5 and seq. were given the status of significance on the basis of within-subject results referred to earlier. Finally, for subjects who had received 900 training trials, CS_1 in conditions OL, 1.5 and seq. was the superior reinforcer, but only in the OL, 1.5 treatment were chance levels exceeded. In the OL, 2.5 condition, CS_2 responses surpassed those of CS_1 but by an insignificant extent. In opening his interpretation of these data, Baker

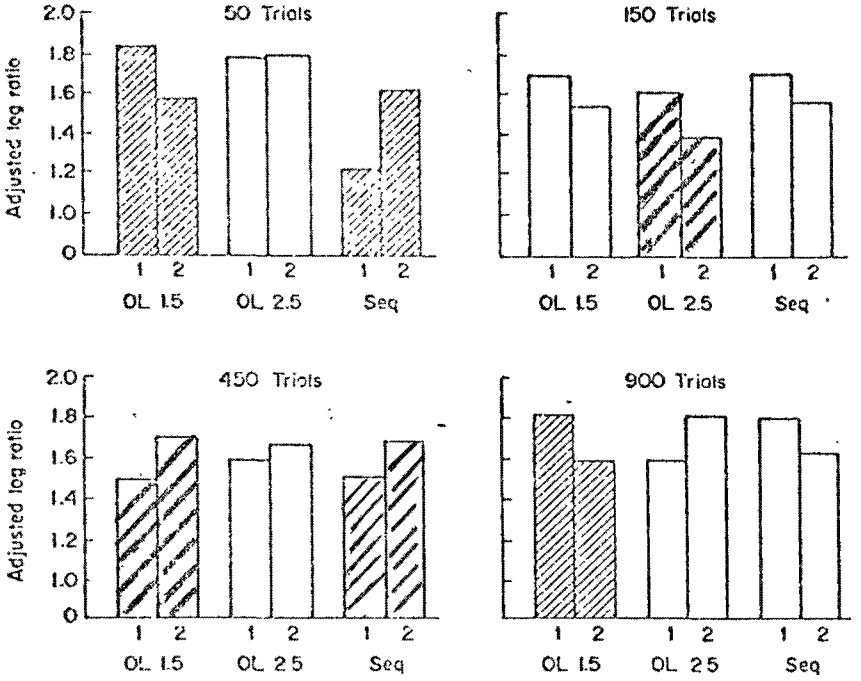

Fig. 3. Adjusted log ratio responses to the CS_1 and CS_2 in each of the three compound stimuli as a function of training in a between-Ss design. Diagonal lines indicate the significant differences.

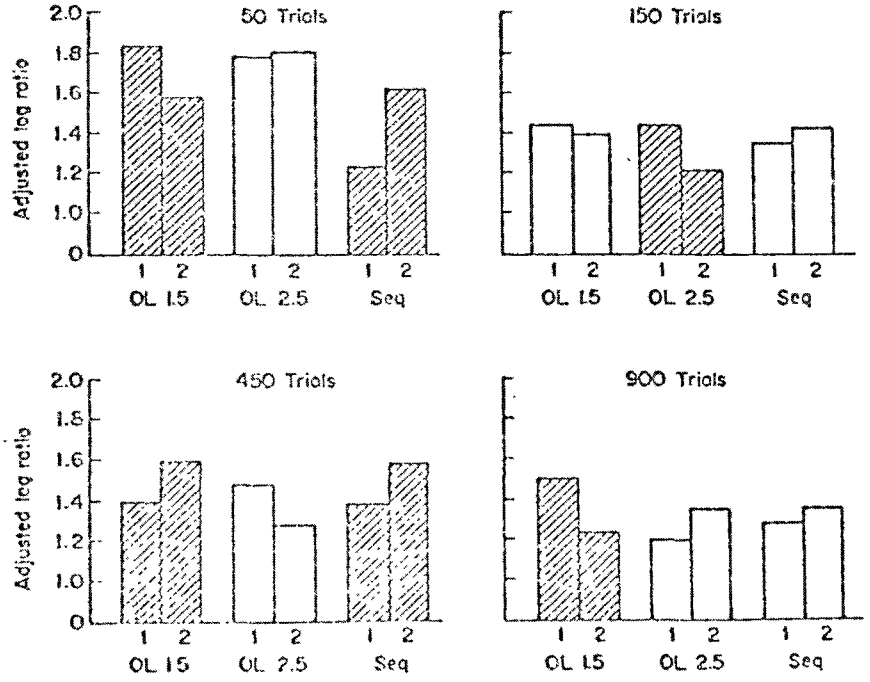

Fig. 2. Adjusted log ratio responses to the CS_1 and CS_2 in each of the three compound stimuli as a function of training in a within-Ss design. Diagonal lines indicate the significant differences.

Figure 1. Taken from T. W. Baker. Component dynamics within compound stimuli. In Topics in learning and performance. New York and London: Academic Press, 1972, p. 99. (Wide-spaced diagonals in Baker's Fig. 3 are added.)

pointed to a significant Training level X Component interaction which held an overall tendency for CS_2 responses to surpass those of CS_1 after 50 and 450 training trials and "recede" below CS_1 levels following 150 and 900 pairings. Component performances across the different levels of training did not show a consistent decline. Baker conceptualized this interaction in terms of a four-stage developmental process associated with each level of compound conditioning (50, 150, 450, and 900 pairings). During stage I (50 pairings) of development, CS_2 was assumed to acquire more CR strength than CS_1 due to its shorter CS-US interval. With increased training, it was supposed that CS_1 gained in response strength as the function of a backward component generalization of the sort inferred by Levis (1971, 1970) to occur during compound conditioning. Therefore, enhanced CS_1 test performances in stage II (150 pairings) were linked to the combined effects of the kind of component generalization mentioned above and another demonstrated by Borgealt, Donahoe and Weinstein (1968), between training and testing situations. With 450 conditioning trials (stage III), CS_2 was thought to "regain" superiority because of a severe steepening of both generalization gradients. Succeeding 900 training trials (stage IV), the predomination of CS_1 was related to the emergence of dynamic stereotype conditioning (sequence conditioning). From this formulation, Baker attempted to explain the Stimulus condition X Training level

X Component interaction described above. Accordingly, at the 50 trial training level, the seq. condition was judged to have obtained stage I CS_2 dominance, while the OL, 1.5 condition appeared to be well into the second stage of CS_1 dominance associated with component generalization. Because the OL, 2.5 treatment manifested component equality at this training level, it was concluded that this subgroup was at the midway point between stages I and II. The quasi significant increase of CS_1 responses over those of CS_2 (based on the twin within-subject experiment) in the OL, 2.5 condition after 150 conditioning trials was related to the belated acquisition of stage II CS_1 dominance. The insignificant CS_1 versus CS_2 performances for the other compound treatments within this training level were construed to indicate the onset of stage III CS_2 "predomination." Quasi significant CS_2 test domination for conditions OL, 1.5 and seq. within the 450 training level was conceived to reflect stage III of diminished component generalization. Once again, nonsignificant test results of the OL, 2.5 treatment at this training level placed this subgroup in an intermediate position between stages II and III. Significant CS_1 "predomination" for condition OL, 1.5 after 900 conditioning trials agreed with Baker's stage IV dynamic stereotype conditioning. Nonsignificant component test differences for the other compound treatments following 900 training trials were judged to represent a lag in their stage IV development.

The alternate shifting of response superiority between component stimuli (in the absence of progressive component CR diminution across independent training levels) could not be explained by the informational or (simultaneous) configural hypotheses. In the former, only CS_1 was predicted to gain supremacy, while in the latter it was anticipated that the responses supported by both component stimuli would decrease with increased quantities of prior compound conditioning. Baker's claim that stage IV CS_1 dominance was an instance of dynamic stereotype conditioning is vague. If this were so, both components should have maintained equal response levels, since in stereotype conditioning it is the ordinal position of a test stimulus that determines the nature of the CR and not the identity of the stimulus (Razran, 1971, p. 240; 1965a, p. 240; 1961, p. 125). Despite the absence of component CR diminution, the most likely construct to fit Baker's $CS_1CR > CS_2CR$ stage IV result would be the configurational concept of the Russian literature as it was related to the third stage of serial configuring (i.e., $CS_1CS_2CRs = CS_1CR > CS_2CR$).

STATEMENT OF THE PROBLEM

Component CR Diminution

Perhaps the most interesting aspect of the configural position was rooted in its contention that component CR

diminution occured with increased (simultaneous) compound conditioning. This alleged result seemed to go against everything written since Pavlov. However, Rescorla and Wagner (1972), after reformulating past interpretations of their researches (Rescorla, 1969; Wagner, 1969a, 1969b), put forth a novel theory of Pavlovian conditioning which allowed for component CR diminution in a manner consistent with the laws of classical conditioning. The portion of their theory that was related to conditioned excitation stated that if a simultaneous compound stimulus (AX) was followed by a US, the change in associative strength to each component (V_A and V_X) would be determined by the existing associative strength to the compound CS (V_{AX}). This idea was represented by $\Delta V_A = \alpha_{A \cdot \beta_1}(\lambda_1 - V_{AX})$ and $V_X = \alpha_{X \cdot \beta_1}(\lambda_1 - V_{AX})$, whereby alpha symbolized learning rate parameters related to component salience, and beta, the learning rate parameter associated with US_1. The value of λ_1, expressed the asymptotic level of component associative strength that US_1 could support. Therefore, the theoretical perspective embodied by these expressions predicted greater increments in associative strength for V_A and V_X on early AX conditioning trials when $\lambda_1 - V_{ax}$ would be at a maximum. Because it was also assumed that V_{AX} equalled the summation of excitation for V_A and V_X ($V_{AX} = V_A + V_X$), as similarly discussed by Grings and O'Donnel (1956), Grings, Uno and Fiebiger (1965),

Wolf (1963), Weiss (1964), the possibility would exist that V_{AX} could exceed λ_1 if V_A and V_X each equalled λ_1 prior to compound conditioning. In that event, $\lambda_1 - V_{AX}$ would have a negative value during compound training and, accordingly, V_A and V_X should lose degrees of associative strength as determined by their respective alpha values. Diminution of component associative strength should continue until $V_A + V_X$ (or V_{AX}) equalled λ_1. If V_A and V_X equalled 0 prior to compound (AX) conditioning (as in "configural conditioning"), V_{AX} should not exceed λ_1 and, therefore, the diminution of component associative strength would not result with compound training alone.

Although configural theory (Razran, 1965b) predicted component CR diminution, any role played by the excitatory summation of components in the diminution of component responses was denied. Instead, it was explained that the superiority of compound CRs over diminishing component CRs was the outcome of the simultaneous overtraining of two evolutionary levels of learning, configural and component conditioning. In the early stages of compound conditioning, interpolated extinction trials with each component stimulus lessened the degree of conditioning to the compound, but in the later stages of conditioning, the component extinction trials had no effect (Razran, 1971, p. 208; Woodbury, 1943). On this basis, it was the consensus of the Russian researchers that the compound responses, in the initial stage of

conditioning, were the result of the summation of component excitation, but the ineffectiveness of the component extinction trials in the later stages of conditioning was construed to be the result of configurational learning. Therefore, configuring was thought not to pre-exist prior to compound overtraining, but to develop through the simultaneous (simple) conditioning of the component stimuli. Razran (1971, 1965b), supported by ontogenetic and phylogenetic comparisons together with lesion studies (summary points 2 & 3, p. 8), suggested that each kind of learning had its own type of neural action with respect to locus, pattern, or both; and that the neural system which underlied the effects of configural overtraining occurred later in phylogeny than that system which supported component conditioning effects. During configural testing, it was conjectured that higher levels of the nervous system necessary for "configural CRs" were associated with the suppression of lower levels related to component CR evocations (Razran, 1965a, 1965b, pp. 215, 216, 227; 1971, pp. 213, 221). This aspect of the evolutionary version of the configural hypothesis was predicated on observations which revealed a release of lower order learning effects from suppression (i.e., diminution) in subjects under the influence of drugs, fatigue, emotional, physical, and mental disturbances, etc. Consistent with this line of thinking, and in view of the component conditioning phase prior to component CR diminution, diminished

component CRs resembled the extinguished CRs of conventionally conditioned stimuli. However, because component CR diminution was thought to occur with or without component contrasting, this result went counter to the laws of Pavlovian conditioning. In answer to such criticism, Razran (1965b, p. 209), after having studied four main evolutionary levels of learning (classical conditioning, operant conditioning, configural conditioning, symbolization), was able to report that with each kind of learning evolved some functional laws specific to each (i.e., configural learning) and these were not derivable from the laws of antecedent levels (i.e., Pavlovian component conditioning).

In conceptualizing the crucial difference between summation and configural theories, only one behavioral question need be entertained for now. Were compound test CRs a result of the summation of component test responses, or were they independent? Configural theory stated they were separate and, for this reason, it was possible to observe compound test CRs along with relatively ineffective component responses. Summation theory held them as dependent and predicted that any diminution in component CRs would be reflected by weakened compound test performances.

Up to this point in time, the summation theory of component CR diminution has been fashioned from experiments which had presented either negative or positive conditioning trials with component A prior to, or concomitant with compound

AX training trials (Rescorla, 1969; Wagner, 1969a, 1969b; and the blocking research in Kamin, 1968, 1969a, 1969b). In this way, the λ_1-V_{AX} value was influenced before or during compound conditioning. The last stages of these experiments consisted of test trials to assess the extent of component conditioning to X. Since these studies did not employ treatment groups given varying amounts of compound conditioning with similarly treated components, it was not meaningful to include these works in the fabric of the simultaneous compound conditioning literature review. Even so, the summation theory derived from these experiments was directly applicable to the simultaneous compound conditioning results reviewed earlier. For independent treatments given different quantities of (simultaneous) compound conditioning trials prior to testing, it was anticipated that any increase or decrease in the component test responses would be reflected in the corresponding compound tests (i.e., VAX = VA + VX). Compound test CRs were expected to follow the direction of the component test responses. These expectations were confirmed by Baker (1969). As the component test responses increased in frequency, from his independent undertrained to overtrained levels, so did the compound test responses. This same result was evident between the underconditioned and asymptotically conditioned treatments of Booth and Hammond (1971). But the compound and component test measures for their overtrained group showed component CR

diminution apart from a reduction in compound measures. Test responses for the compound CS failed to differ between the asymptotic and overtrained groups. Hence, the weight of evidence supporting the summation and configural theories is split. Compound and component responses were observed to grow together prior to overtraining. Following overtraining, the component responses increased with the compound test responses (Baker, 1969), but they also decreased without compound CR reduction (Booth & Hammond, 1971).

Unfortunately, the between-subject experiments of Baker (1969) and Booth and Hammond (1971) were the only works that employed the strong criterion of component CR diminution to evaluate the existence of configurational learning in simultaneous compound conditioning. Given the meagre amount of data on this issue, and a methodological shortcoming in both of these studies, no strong support for or against configural theory can be put forth. In Baker's experiment, the ascending values for the component responses across his independent groups seemed to rule out configuring even though consistent compound-component differentiation was demonstrated. However, Baker's work did not provide a control level of responding against which component (conditioned) excitation could be assessed. It could well have been the case that his overtrained groups contained no component excitation, indicating that configurational learning had occurred. On the other hand, the component CR diminution

found for overtrained subjects in Booth and Hammond's work may have simply been due to the interfering effects of orienting reflexes evoked by the overtrained component stimuli in testing. In their second experiment, these authors attempted to assess the extent to which orienting reflexes, evoked by component stimuli, may have been responsible for the inferior bar press suppression ratios (increased ongoing bar pressing for water) associated with their overtrained components. It was assumed by these researchers and Baker (1968) that the overtrained components in testing, because of their longer training period in compound, would have acquired the properties of novel stimuli to a greater degree than those in the asymptotic treatment. To test this explanation of component CR diminution, a truly random compound conditioning control (reviewed by Rescorla, 1967), was matched in all respects to their overtrained treatment with the exception of the CS/US contingency. A comparison of compound and component test responses between these conditions was intended to reveal the extent of nonassociative effects on appetitive operant responding. According to the authors, the enhanced bar press suppression eventually found for all their overtrained stimuli in testing over the control presentations, ruled out nonassociative factors (i.e., orienting reflexes) in the explanation of component CR diminution. However, the commonly accepted view that an underlying mechanism mediates conditioned bar

press suppression clearly disagreed with Booth and Hammond's mode of analysis for nonassociative influences. This position emerged from the numerous experiments reviewed by Rescorla and Solomon (1967) which showed that conditioned suppression of appetitively supported bar pressing was mediated by a conditioned emotional response (CER), or a central motivational state evoked by a CS. Given the prominent role of the CER in determining the suppression of operant responding, it is apparent that Booth and Hammond's control procedure could not rule out the possibility that the reduced effectiveness of the overtrained components might be linked to the disruptive influence of orienting reflexes on the CER. These kind of responses have been shown by Pavlov (1927) to be antagonistic, and therefore one would expect orienting reflexes to reduce the magnitude of the CER, thereby reducing the degree of bar press suppression to the overtrained components. When it is also considered that Booth and Hammond only used their first two compound and component test trials for configurational testing, their claim to have demonstrated configurational learning must be held suspect in view of the orienting reflex interpretation of component CR diminution.

Although the use of the random control treatment by Booth and Hammond could not be used to evaluate nonassociative influences of the overtrained components on mediated operant performance, the presence of stimulus evoked CERs

in the overtrained treatment could be detected with this control comparison. When such an analysis was carried out, it was found that, for the overtrained treatment, the compound suppression ratios were significantly smaller (more suppression) than those of the components, and suppression measures for all stimuli in the overtrained treatment surpassed those for the control stimuli. In the light of these comparisons, the diminished responses (suppression ratios) to the overtrained components in testing, relative to asymptotic components, could reflect the residual conditioned excitation (CERs) that remained during the disruptive effects of OR elicitation. Therefore, when the interfering effects of orienting reflexes on CERs are taken into account, the total pattern of results in Booth and Hammond's experiment might not include authentic component CR diminution. In this instance, Booth and Hammond's compound conditioning results would not contradict Baker's work (where 50 to 150 test trials in extinction precluded OR effects), since their overtrained components still manifested conditioned properties.

Because the possibility of component CR diminution following the onset of compound overtraining goes against the laws of Pavlovian conditioning and demonstrates configurational learning through (simultaneous) compound conditioning, it would not be of trivial importance to clearly establish this result. Therefore, to avoid confounding

configurational learning with the disruptive effects of orienting reflexes and to provide a measure of component conditioning, the following experiment will use long delay electrodermal conditioning in a between-subject experiment that would include a truly random control comparison in addition to two other treatments given different amounts of compound conditioning. Following configural conditioning in this situation, the first GSRs after stimulus test presentations represent either conditioned reflexes or unconditioned orienting reflexes, which can be separated from two other conditionable responses occurring just before and immediately after the point of US exposure. According to Booth and Hammond (1971) and Baker (1969), the first GSR following the component test presentations ought to reflect an unconditioned orienting reflex evoked by the absence of one stimulus element. Second and third GSRs, which follow the component test presentations, should reveal pure conditioning effects. This method of conditioning, therefore, could provide a comparison of unconditioned orienting reflexes evoked by the component stimuli of an overtrained treatment and one trained only to asymptote, in addition to furnishing two conditional responses with which configurational learning could be tested. At the same time, the truly random control comparison would provide an opportunity to demonstrate that the component stimuli of a compound CS lack conditioned properties following overtraining. This

is probably the ultimate test for configurational conditioning and, as yet, has not been reported.

When the (simultaneous) configurational conditioning literature is taken into account, five criteria for the demonstration of configural learning, with the second or perhaps the third GSR test response, can be set down. The first requires that the compound CRs of the overtrained treatment be significantly greater than those of an appropriate control comparison to demonstrate successful conditioning with either the stimulus elements of the compound, or the patterned aspects of the CS. Second, the same responses for a group trained only to asymptote should not differ from the overtrained level of responding to ensure that any component CR diminution, that might occur in the overtrained group, could not be linked to massed training effects, apart from compound overtraining. Third, to show that the stimulus elements of the compound CS initially acquire conditioned properties which diminish with overtraining, the component responses of subjects trained only to asymptote must be significantly greater than those of the overtrained and control treatments. Fourth, in addition to the last requirement, another would demand complete compound-component differentiation for the overtrained treatment, and no differences between the component responses of the overtrained and control group, in order to conclude that only patterned properties eventually became the active aspect

of the compound CS. Fifth, the lack of a negative correlation between the first component responses (orienting reflexes) and later diminished reactions (second or third responses) associated with the same component presentations, must be shown to rule out the disruptive influences of orienting reflexes which may be responsible for the component CR diminution obtained.

Given the fulfillment of these criteria, it can be concluded that the stimulus elements of a compound CS do condition initially, but during overtraining lose their conditioned properties (component CR diminution), while patterned aspects of the compound CS acquire conditioned strength.

The CR in Long Delay Electrodermal Conditioning

In long delay electrodermal conditioning with human subjects, the CS-US interval is usually not less than 7.5 sec and three kinds of GSRs can be measured on a single trial, instead of only one as in short delay conditioning (ISI 0.5 sec). The first response immediately follows the onset of the CS, while the second GSR usually occurs just before the US, and the third just after. First period GSRs can represent a conditioned response or an unconditioned orienting reflex. Second period responses are described as the anticipatory response to the US, since these GSRs are not prominent after CS onset when a US is not contingent upon the CS. In the absence of the US on test trials, the

third response is considered to be either a CR offset response or a disparity reaction to the unexpected nonoccurrence of the US. In Table 1 are the various latency criteria for the three classes of GSRs recorded from human subjects who participated in the following studies to be reviewed. Because the physiological latency of the GSR is slightly over one second, the first response interval may begin one second following the CS onset. The second interval can overlap the US by 1.0 sec for the same reason.

Stewart, Stern, Winokur and Fredman (1961) were first to interpret the significance of the first and second interval GSRs. They found that the frequency of first interval responses declined steadily with conditioning trials and paralleled previous first interval adaptation responses having an overall lower frequency level. Second interval GSRs were hardly present during adaptation training, but these responses formed an inverted U-shaped frequency curve over trials when the US followed CS presentations. The similarity of the first interval frequency curves between adaptation training and conditioning trials was taken to represent unconditioned ORs, while the initial frequency increase of second interval responses in conditioning earned CR status. The authors cited massed reinforcements to explain the eventual decrease in the number of second interval GSRs during the later conditioning trials. Lockhart and Grings (1963) objected to the CR criterion used by

Table 1

GSR Intervals Employed by Studies To Be Reviewed

	I S I Sec	Sec from CS Onset		
		First Response	Second Response	Third Response
Dawson (1970)	8.0	1.0 - 3.9	4.0 - 8.9	9.0 - 12.0
Dawson and Biferno (1973)	8.0	1.0 - 3.5	3.6 - 8.9	- -
Epstein, Burstein and Smith (1971)	5.0	0.0 - 3.2	3.2 - 6.4	- -
Gale and Ax (1968)	9.5	1.0 - 4.5	4.5 -10.5	10.5 - 14.5
Gale and Stern (1967)	7.8	1.5 - 4.5	4.5 - 8.5	- -
Leonard and Winokur (1963)	7.5	1.5 - 3.5	3.5 - 8.5	8.5 - 11.0
McDonald and Johnson (1965)	10.0	1.3 - 2.9	3.0 -11.0	- -
Ohman (1971)	10.0	1.0 - 4.0	4.0 -11.0	11.0 - 14.0
Prokasy and Ebel (1967)	8.0	1.35- 4.95	4.95- 9.53	9.53- 14.55
Schiffman and Furedy (1972)	7.5	1.0 - 5.0	5.0 - 9.0	9.0 - 13.0
Stewart, Stern, Winokur and Fredman (1961)	7.5	1.5 - 4.5	4.5 - 8.5	- -

Stewart et al. (1961) and have appealed for the use of sensitization and pseudo-conditioning controls. Such control comparisons would more accurately relate experimental group responses to the CS-US contingency. Response comparisons between adaptation and conditioning periods in Stewart et al. (1961) did not accomplish this degree of control.

In accord with Lockhart and Grings (1963), McDonald and Johnson (1964) trained human subjects assigned to an experimental or a pseudo-conditioning control treatment. Frequency and magnitude measures for first interval responses were compared between the adaptation and conditioning periods. These responses fell short of the significance in both treatment groups. The same was true for between-group comparisons within the adaptation and conditioning segments. With response frequency as the only measure, no between-group differences were noted for the second interval responses in habituation, but in the experimental treatment these responses increased in frequency from habituation to the end of the conditioning period. The responses of the control group decreased in frequency. This treatment variation emerged once again with a between-group analysis of the conditioned responses.

Prokasy and Ebel (1967) examined first, second, and third interval GSRs in the fashion of McDonald and Johnson (1965), but their experimental and sensitization control treatments each contained a high and low intensity CS subgroup (four treatment conditions). In conditioning, first

interval responses for the experimental groups did surpass control reactions in terms of magnitude, amplitude, and frequency. High CS intensity increased the magnitude and frequency of all first interval GSRs. The same measures showed that experimental and control GSRs decreased with conditioning trials but control responses fell more sharply. Analysis of second interval GSRs of the conditioning period uncovered greater magnitude and frequency scores for the experimental treatment relative to control subjects. CS intensity had no effect and in no cases were the amplitudes of GSRs affected by the experimental operations. In contrast to the first interval responses, second interval reactions increased slightly across conditioning trials, while control reactions decreased slightly. Unreinforced test trials during conditioning provided for the quantification of third interval responses. As with the second interval response, magnitude and frequency measures demonstrated third interval conditioning, but amplitude scores did not differ between groups. A stimulus intensity effect was found, but only with a separate frequency measure.

Other researchers have taken advantage of the discrimination conditioning paradigm to control for sensitization factors in conditioning, Leonard and Winokur (1963) presented two distinct tones to subjects as positive and negative cues in a long delay (differential) GSR conditioning situation. The number of first interval responses in

conditioning was the same for the positively and negatively reinforced stimuli, but second interval responses between stimulus conditions did differ in favor of the positive cue. No differences in a prior adaptation period secured these results. Gale and Stern (1967) enlarged the differential conditioning technique of sensitization control by including several discrimination conditioning controls in addition to the discrimination treatment. In one discrimination control, the positive cue was partially reinforced and, in another, both cues were presented in an unpaired random manner with a US. A third received backward conditioning to the positive stimulus. Amplitude measures for first interval responses of the conditioning period showed that the positive cue in the experimental condition evoked larger responses than any of the control stimuli, and did not yield a waning response pattern across trials. All control stimuli for groups exhibited GSR adaptation with the exception of the positive cue in the backward conditioning treatment. Consistent with these outcomes were the greater degrees of differentiation between discriminative stimuli for the experimental subjects over controls. The positive and negative cue difference scores for trial blocks were measures of conditioning with orienting influences excluded. Such a curve for first interval GSRs of the experimental condition (Gale et al., 1967) did express the negatively accelerated growth function associated with the development of "the CR."

Frequency quantifications agreed with amplitude measures. The positive second interval GSRs in the experimental condition were also greater and more frequent than all other control stimuli. Gale and Ax (1968) gave their human subjects 300 trials (60 per day) with the same experimental discrimination training as in Gale and Stern (1967). First and second interval response amplitude disclosed successful differential conditioning, and even with the extended training, only insignificant degrees of adaptation were detected for the first interval reaction. Both kinds of conditioned reactions reached a maximum on the first day of training and held steady to the last.

Another more elaborate GSR discrimination experiment (Dawson & Biferno, 1973), using long delay conditioning, presented five tones on a single trial in order to mask the CS+, US contingency with two tasks related to the identification of certain aspects of the stimulus sequence. The last two tones of each sequence served as the CS- and CS+, with the last tone as the CS+. For two conditioning treatments, half of the subjects in each group received the highest frequency as the CS+, while the other halves received the lowest frequency for the positive cue. One of these conditions, the instructed CS+, US contingent treatment, was told that the shock was predictable, while the other treatment which also received CS+, US contingent pairings, was not so instructed. In this way, the noninstructed group

had a lower probability of becoming aware of the CS+, US contingency. A third group also received the US following the last tone, but within this treatment there was no CS+, US contingency as both the highest and lowest sounding tones served as the last stimulus on half of the trials. This treatment, like the second, was noninstructed and served as a control condition for the US terminated stimulus sequences.

During conditioning, the successful performance of the masking tasks for all subjects indicated that the masking procedure did not prevent the subjects from perceiving all stimuli presented on each trial. Following the experiment, it was verified that almost all of the noninstructed subjects failed to perceive the CS+, US contingency, while almost all of the instructed subjects were aware of the CS+, US contingency. One of the indicators of awareness consisted of a postconditioning questionnaire, while another was based on the push-button responses of each subject during conditioning, indicating the degree of certainty regarding the point in time that the US would occur on a trial. Almost all the instructed subjects indicated their degree of awareness by learning to press their "certainty shock" button during the first or second GSR interval (or both) associated with the CS+. Practically all noninstructed subjects failed to learn to consistently press their "certain shock" button during the CS+ periods.

When first and second response magnitudes in the last half of acquisition were analyzed for discrimination learning, only the contingent CS+, US instructed group yielded significant differential responding for both GSR periods. The contingent CS+, US noninstructed condition, and the noncontingent-noninstructed control treatment did not differentially respond in either GSR interval.

On the basis of concurrent "shock certainty" button presses, the authors determined the point (trial) at which awareness emerged for subjects who eventually became aware of the CS+, US contingency. When the GSR magnitudes for the discriminative stimuli were compared for the first interval GSRs within the last three trials prior to the point of awareness, no differentiation was evident. The same measures within the first three trials after the point of awareness showed clear differentiation. A similar pattern of results was observed for second interval responses, with the exception of the last two post-awareness trials which did not reveal clear discrimination conditioning. These results supported the view that both the first and second interval GSRs could yield discrimination conditioning, and that awareness of the CS+, US contingency was an important factor in the determination of autonomic conditioning.

In an experiment which employed the same basic discrimination procedure as Dawson and Biferno (1973) to mask the CS+, US contingency, Dawson (1970) demonstrated first interval GSR

discrimination conditioning with pairs (CS+, CS-) of test trial magnitudes only when subjects were instructed that the US would usually follow the CS+. The conditioning treatment that was not instructed about the CS+, US contingency did not yield first interval GSR conditioning. Second interval GSR magnitudes for test trial pairs failed to furnish discrimination learning in either the instructed or noninstructed conditioning groups. However, when the frequency of second interval peaked responses for successive pairs of test trials (each trial could contain more than one second interval response) were analyzed, second interval discrimination conditioning was revealed for the instructed group, but not for the noninstructed condition. These results agreed with Dawson and Biferno (1973), and suggested, along with Prokasy and Ebel (1967), that the second interval response was probabilistic. It should be pointed out, however, that although this experiment used an 8.0 sec CS-US interval as in Dawson and Biferno (1973), the tones employed were not extended to the onset of the US and, therefore, this experiment was of the trace conditioning type.

Schiffman and Furedy (1972) recorded first, second, and third GSRs from subjects given one of two kinds of discrimination training that involved differently treated CS- stimuli. One treatment received a CS+ and an explicitly unpaired CS-. This CS- was of the conventional type in that shocks were never allowed to come within 29.0 sec after the CS-. The

second treatment utilized a noncontingent CS- with the CS+. Here, the CS- was uncorrelated with the US presentations, as some of the reinforcements might have occurred just after the presentation of this CS-. Both treatment groups employed 15 US alone presentations for 15 CS- or RS presentations which accompanied 15 CS-US trials. After the training period, subjects from both groups were assessed as to the accuracy of their subjective estimates regarding the degree of correlation between the CS and US occurrences.

Given the procedural differences between groups, it was possible to test whether or not the uncorrelated CS- would yield more or less discrimination learning than the explicitly unpaired CS-. The contingency position of Rescorla (1967) held that the uncorrelated CS- would reach a level of excitation above that for a negatively US correlated CS-. Therefore, contingency theory predicted that the explicity unpaired CS- condition would yield a greater degree of discrimination than the uncorrelated condition because of lower GSR levels to the CS- following training.

Contrary to the contingency prediction, it was found that the degree of GSR discrimination for the first interval response in the uncorrelated CS- condition was greater than the extent of first interval conditioning in the unpaired CS- group, but this difference in discrimination learning was not significant. Post-training tests for the subjective estimates of the degree of CS, US correlation showed a clear cognitive discrimination, as the CS+ in both groups was associated with the US to a greater degree than the CS-. However, cognitive

discrimination for the unpair CS- group was greater than that in the uncorrelated CS- treatment. Therefore, the GSR data went against Rescorla,s contingency theory, while the magnitudes of cognitive discrimination agreed with it. When the scores for subjective estimation of the CS+, Us contingency were correlated with the algebraic difference between CS+ and CS- GSR scores, no relationship was evident.

Second and third interval GSRs for both kinds of conditioning treatments failed to condition and, therefore, subjective contingency estimations were not scored.

In all, it was concluded that the second and third interval GSRs do not reliably reveal discrimination conditioning as is perhaps commonly believed. Apart from these response intervals, first interval GSRs showed clear conditioning in both conditioning groups, but the greater degree of discrimination learning in the uncorrelated CS- condition, which was not significant, went against the contingency theory of conditioning. The lack of agreement between the degree of first interval discrimination learning and the subjective contingency estimates ruled out cognitive prediction as a crucial factor in the acquisition of the discrimination learning obtained.

Ohman (1971) emphasized that the CS-US stimulus condition was of a greater stimulus complexity than the unpaired CS control situation, and that all GSR differences between experimental and control treatments may have simply been OR differences related to stimulus complexity. To control for this possibility, Ohman tested human subjects for stimulus

generalization after either long delay conditioning or unpaired CS/US presentations with a tone CS (3000 hz). If CS-related responses (first, second, or third interval GSRs) were conditionable, he believed that stimulus generalization tests (three tones as generalization stimuli) would reveal decreased GSR measures with increased stimulus change, but if they represented ORs these GSR measures should increase. In acquisition, first interval GSRs for the experimental and control treatments decreased in magnitude with continued conditioning trials, and no treatment differences were noted. Second and third interval GSRs for the experimental subjects increased in magnitude early in conditioning, but then declined. Control magnitudes were significantly less than the experimental GSRs and gradually decreased with continued training. The stimulus generalization test consisted of single presentations of the CS and three generalization stimuli (200, 500, 1200 hz) all arranged in counterbalanced orders within groups. First interval test gradients for the experimental and control treatments were of the OR type, increasing in magnitude with the degree of stimulus novelty. No treatment differences were apparent. The second and third interval response sets held a conditioning effect, but both kinds of GSRs yielded flat generalization gradients along with their controls. Additional control GSRs taken from another group of subjects prior to the actual experiment revealed no differences in initial reactivity to the CS

and generalization stimuli. In a second experiment, tests for temporal generalization with a 10.0 sec tone CS and three generalization stimuli (1.0, 4.0, 7.0 sec) produced evidence for OR involvement early in the conditioning of the third interval GSR, and CR involvement toward the end of training. For subjects given nine conditioning trials prior to temporal generalization testing, the resulting gradients for both the experimental and sensitization treatments were of the OR type. The experimental and control treatments with 24 conditioning trials yielded divergent generalization gradients. The experimental subjects provided a falling gradient from the trained CS, and controls a rising gradient.[1]

In view of experiments I and II, Ohman classified the first interval GSRs as ORs, but the third interval GSRs were said to have characterized ORs early in conditioning and CRs sometime thereafter. The flat generalization gradients for the second interval responses of experimental and control subjects in experiment I, were related to OR involvement, but the significant conditioning effect for these gradients left the nature of the second interval response in doubt.

Epstein, Burstein and Smith (1971) corroborated the first interval GSR results of Ohman (1971), and also

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1 The rationale relating these offset responses to third interval GSRs was based on a complement of control data too lengthy to review here.

reproduced the second interval GSR conditioning effect, but with a decremental generalization gradient. The second interval control gradient tended toward the ascending OR type. Basically, three experimental and four control treatments were trained with either variations of classical GSR conditioning or control conditioning procedures, all interpolated between adaptation and generalization test phases. Both phases consisted of similar test presentations of a CS and three generalization stimuli (tones: CS 1967, 1000, 468, 153 hz). The potential for a pre- and post-training comparison of gradients ensured that any (decremental) stimulus generalization gradient obtained could be linked either to the conditioning operations, or to unlearned stimulus reactions. Initially, adaptation tests showed a U-shaped GSR magnitude curve across the CS and generalization stimuli, when the first interval responses for all groups were analyzed. Second interval reactions were not differentially affected by stimulus quality. All experimental and control treatments were comparable prior to interpolated training with the exception of one experimental group which only differed from the rest in terms of enhanced second interval GSR magnitudes. This difference was attributable to the requirement of these subjects to verbally label the stimulus qualities during their presentation, while the other experimental groups either did not label, or labelled the stimulus presentations after their offset.

In conditioning acquisition, the three experimental conditions which verbally labelled the CS during or after its presentation, or not at all, yielded first interval acquisition curves that portrayed an inverted U-shaped magnitude pattern. Experimental subjects who labelled the CS during its presentation had manifested greater magnitudes relative to the other experimental treatments, but in the last stages of conditioning between-group differences could not be seen. No treatment variations prevailed between three control groups (backward conditioning, US alone, CS alone; random CS-UCS control training data deleted) during the training period. Throughout the experiment, all control subjects had verbally labelled the CS after its presentations, and their acquisition curves for GSR magnitude were almost flat. A comparison of CS responses for combined experimental and control treatments did reveal a first interval conditioning effect. Second interval GSRs during training were not affected by variations within or between the experimental and control conditions. Because first and second interval CRs at the end of training were equal to, or less than, CS magnitude levels at the termination of adaptation testing, analysis of the generalization test magnitudes could not be performed until conditioning of the trained CS could be demonstrated. For this purpose, the first and second interval GSRs for each group were examined across the last adaptation and first generalization CS test trials. Separate

experimental and control group analysis revealed no first interval GSR differences, but second interval reaction magnitudes of the experimental subjects in (generalization) testing were highly significant over the CS adaptation responses. No significant pre-post training CS differences were reported for the second interval control treatments. To test for the existence of a decremental generalization gradient in each conditioning treatment, the first and second interval GSRs were compared separately across the adaptation and generalization test phases. A phase X tone interaction here would indicate a change in curve form for the generalization stimuli, from adaptation to generalization testing. As it turned out, a significant phase X tone interaction was only obtained when the second interval measures for the experimental groups were pooled for analysis. This interaction did in fact indicate a decremental generalization gradient for second interval GSRs. The experimental first interval responses and all control measures did not express the phase X tone interaction. Therefore, interpolated conditioning did not change either the first interval CS magnitudes from their pretraining levels, nor their generalization gradient from the adaptation test pattern. Second interval responses were conditioned and a decremental stimulus generalization gradient was obtained.

In summary, it can be said that the between-group experiment (McDonald & Johnson, 1965; Prokasy & Ebel, 1967)

has uncovered evidence for and against first interval conditioning while reliably demonstrating associative learning with the second response. Conversely, with the discrimination learning paradigm for sensitization control, it was only the first response that conditioned consistently (Leonard & Winokur, 1963; Gale & Stern, 1967; Gale & Ax, 1968; Dawson, 1970; Schiffman & Furedy, 1972; Dawson & Biferno, 1973). The stimulus generalization experiments (Ohman, 1971; Epstein, Bernstein & Smith, 1971) furnished evidence for second response conditioning, but unanimously denied first interval GSR conditioning, and provided data which supported an orienting reflex interpretation of the first interval response. Third interval GSR conditioning was also unreliable, in that simple conditioning techniques (Ohman, 1971; Prokasy & Ebel, 1967) produced positive findings, but the discrimination method (Schiffman & Furedy, 1972) failed to reveal differences. Given the complex variations between simple and discrimination conditioning techniques reviewed, in addition to differences in CS+, US awareness factors between some of these experiments, no simple explanation can account for the diversity of results reported.

However, because the long delay conditioning procedure of Prokasy and Ebel (1967) successfully effected the conditioning of the first, second, and third interval GSRs, it was adopted for the present experiment with one change. Instead of an explicitly unpaired control, a truly random

control comparison was employed partly to test first interval conditioning with an uncorrelated CS/US control treatment. With this technique, it was anticipated that, in testing, significant compound conditioning could be demonstrated for the second (or third) interval GSRs recorded from overtrained subjects and those trained only to asymptote. Moreover, this procedure could reveal unconditioned orienting reflexes (first interval responses) elicited by the component test presentations in the overtrained treatment. This information should be helpful in interpreting the potential result of component CR diminution for second or third interval GSRs of the compound overtrained condition relative to one trained to asymptote. If it can be shown that there exists no treatment differences for first responses evoked by the component stimuli in testing or, more importantly, the absence of a negative correlation between the first and second (or perhaps third) interval test responses for the overtrained components, the configurational interpretation for the potential finding of component CR diminution would be secure.

Measurement of the Galvanic Skin Response

The GSR refers to a relatively rapid electrodermal response evoked either by stimuli presented to subjects during experimentation, or by uncontrolled events within the subject. These reactions are superimposed upon gradual

background variations of electrodermal activity linked to the experimental situation, called the basal level. Therefore, GSRs elicited by stimuli are usually measured from the basal level, just before stimulus onset, to the peak of the response.

One method of measurement employed to record the GSR involves changes in the electrical resistance of the skin. For this procedure, a constant current is generated by applying a voltage source to the subject's resistance in series with a large known resistance, greater than the subject's skin resistance by a factor of 50 times or more. With this arrangement, the voltage changes across the subject's electrodes, due to skin resistance changes, would have little effect on the current level and be linearly related to variations in the subject's skin resistance (Ohm's Law, $R = E/I$). Shifts in voltage, therefore, are recorded in order to indicate variations in the subject's skin resistance.

A second procedure for measuring the GSR, records skin conductance by placing the subject in series with a voltage source and a "signal resistor." Both the resistor and the impedance of the voltage source (battery) are small relative to the subject's skin resistance (by at least 10 times), to ensure a constant voltage across the electrodes in spite of changes in current. Since, in this case, the voltage across the "signal resistor" is linearly proportional to the current flow, which in turn is a linear function of the

subject's skin conductance (SC = I/E), this voltage is amplified and recorded to reflect the changes in the subject's conductance. It should also be mentioned that, if resistance changes are measured with this system, voltage values across the signal resistor will be inversely proportional to the resistance of the subject.

For both circuits to perform adequately, it is necessary that the parameters for voltage and current (applied or developed) should be within a certain range in order to prevent affecting the skin resistance to be measured. Initially, it was pointed out (Edelberg, 1960) that, with current densities of 10UA/cm^2 and over, skin resistance decreased and, therefore, constant-current circuitry was recommended with a current density less than 10UA/cm^2. However, Edelberg (1967) demonstrated that, with subjects having low basal levels, very high current densities (75UA/cm^2) were needed to affect their apparent resistances, while other subjects with high basal levels were affected by current densities as minute as 4UA/cm^2. Since the ranges of current-voltage linearity (ranges for no change in apparent resistance) for all subjects were associated with 0.8V or less (across a single site), Edelberg concluded that voltage developed, and not current applied, was the limiting factor. In addition, Edelberg (1967) reported that when longer durations for current flow were allowed at a fixed voltage, the current eventually increased, thereby

introducing a time variant factor. On this basis, Edelberg (1967) recommended a value of 0.5V to avoid time-variant and time-invariant effects. Lykken and Venables (1971) reported that they also obtained similar results to those of Edelberg and concur that 0.5V is the appropriate limit for experimentation. Therefore, constant voltage circuitry was advocated by these authors as one can control voltage and current density most easily by this method.

Apart from the circuitry to be used, the literature on the physiological mechanism underlying electrodermal responding would advise the use of skin conductance as an index for this kind of response. The grounds for such a position was provided by research that established a linear relationship between skin conductance and sudomotor activity, shown to be the main factor responsible for electrodermal responding. One such study performed by Lader (1970) used atropine applied to the skin to inhibit sweat gland activity. While the drug was taking effect, changes in skin conductance gradually decreased to zero when expressed as the proportion of conductance changes taken from a control site. Comparable resistance measures revealed dramatic irregularities for the same time period. Similarly, Darrow (1964), who related the degree of sweat gland activity to concomitant skin conductance and resistance measures, found that only skin conductance bore a linear relationship. In addition to these studies, another by Thomas and Korr (1957) demonstrated

linearity between the actual number of active sweat glands per unit area and the skin conductance measure. Therefore, since sweat glands seem to react independently to experimental stimuli which evoke the GSR, and potentially provide low resistance pathways through the skin, it is probably correct to conclude that sweat glands of the skin act as resistances in parallel that diminish in value upon excitation (Montagu & Coles, 1966; Tregear, 1966). If it is accepted that sweat glands serve as resistances in parallel which act as independent current pathways, skin conductance measures would not only provide the most direct index of electrodermal responding, but reduce the complexity concerning the possible dependency of the GSR on basal levels. This follows from the knowledge that conductances in parallel are additive, while a change in resistance must be related to basal levels, since a change in one parallel resistance affects the total resistance which accumulates across the other resistors.

In conclusion, it can be said that skin conductance is the most appropriate index of electrodermal responding, and since constant voltage circuitry can record such values directly without affecting apparent basal measures, this system is probably the best alternative for experimentation. In the experiment to follow, therefore, GSRs were measured in conductance units with the constant voltage procedure.

The apparatus used employed an operational amplifier feedback system for the signal resistor, which gave a theoretical resistance of zero, and thus had no effect on the measurement of subjects' conductance changes.

METHOD

Subjects

Forty-one subjects attending introductory courses in psychology at the University of Ottawa took part in the experiment as paid volunteers, after having been informed that harmless electrodermal shocks would be administered. No subjects on medication of any sort were allowed to participate in the experiment and, for those admitted, no psychometric data were collected. Five subjects had their data spoiled by experimenter error, equipment failure, or adaptation to the US. Out of the 36 subjects who actually contributed data for analysis, half were males and the other half were females. These subjects ranged in age from 18 to 30 years.

Apparatus

Subjects were seated in a 210 cm x 210 cm x 240 cm isolation chamber dimly lit by a dome lamp with an intensity of 9 ft. candles measured by a Basic Science Industries light meter at a distance of 12.0 mm. An electric fan masked

any ambient sounds and provided subjects with proper ventilation.

Galvanic skin responses were recorded with Beckman Silver-Silver Chloride electrodes which were filled with 50% physiological saline in unibase paste and placed upon Beckman adhesive collars having a diameter of 1.0 cm. Basal skin conductance and conductance changes were transduced into voltage changes by applying a constant voltage of a half volt to the subject and detecting current changes with an operational amplifier. The resulting voltages were recorded on two channels of a Watanabi Multicorder Servo Recorder with six type WA 611-1 amplifiers. This system was calibrated by replacing the subject with 1% precision resistors to give 10.0 micromhos basal conductance and 1.0 micromhos conductance change. After testing this system with regard to changes in conductance, it was found to be linear.

A tone of 2800 hz was delivered for 8.0 sec by a Sonalert SC628 behind the subject at a distance of 1.0 meter from the subject's head. Sound intensity level was measured using a Bruel and Kjoer type 220 precision sound level meter and was found to be 64 dbs at a distance of 12.0 mm from the source. Visual stimulation produced by two incandescent bulbs of 60 watts each was presented for 8.0 sec from behind and above the subject at a distance of 1.5 meters from the subject's head. The light intensity at 69 ft. candles

was measured by a Basic Science Industries light meter at a distance of 12.0 mm from the source.

The US, with a duration of 0.2 sec, was a constant current arm shock of 2.0 ma. It was delivered through two Grass Silver-Silver Chloride electrodes with a diameter of 1.0 cm and a current density of .32 ma/sq cm. This was achieved by a transistorized constant current regulated power supply. The constant current output was controlled by the appropriate fixed resistors and a fixed voltage on the input. The current selected was in error by less than 1.0% for the range of subjects' skin resistance values. This had been verified by using a General Radio, No. 1432L decade resistance in place of the subjects and by measuring the resulting current by a Sencone, FE149 meter.

The light, tone, and shock contingency was selected by a manual switch box, and the stimulus durations were controlled by a Lafayette four bank timer No. 52010. Pseudo randomized intertrial intervals with an average value of 44.4 sec were programmed with a Lafayette eight bank timer, model No. 5431. When random shocks were required, a Mousseau (No. 003) random generator was activated through the switch box. All stimulus events were recorded on additional channels of the Watanabi recorder.

All apparatus with the exception of the light and sound sources were placed outside the experimental chamber, but a

Mini Scan No. 5050 Audiovisual unit allowed the experimenter to hear and observe the subjects and the stimulation.

Experimental Design

To demonstrate configurational learning through long delay electrodermal conditioning, the present experiment employed a three phased design (table 2). Three groups of 12 human subjects each, received one of three compound conditioning treatments, with a simultaneous light plus tone CS and an arm shock US interpolated between phase 1, adaptation training and phase 3, configurational testing. In phase 1, adaptation training, and phase 3, configurational testing, all subjects received identical compound CS, light alone, and tone alone exposures in order to evaluate the differential effects of the compound conditioning treatments. Throughout the experiment, the compound and component stimuli had durations of 8.0 sec, while the US presentations in conditioning lasted for 0.2 sec after a delay interval of 8.0 sec. Each phase of experimentation had a $\overline{X}$ intertrial interval of 44.4 sec.

In phase 1, adaptation training, and phase 3, configurational testing, all subjects received 18 trials composed of six triads which each contained different orders of a simultaneous light plus tone, light alone, tone alone. The six triads were pseudo randomized for each subject in such a way as to ensure complete counterbalancing of stimuli

Table 2

Three Phased Experimental Design

		Phase I Adaptation Training Triads																		Phase II Interpolated Conditioning Treatments	Phase III Configurational Testing Triads																	
		1			2			3			4			5			6				1			2			3			4			5			6		
		C	L	T	C	L	T	C	L	T	C	L	T	C	L	T	C	L	T		C	L	T	C	L	T	C	L	T	C	L	T	C	L	T	C	L	T
Random Control	S 1 = = 12																			30 CS-US																	
Asymptotic training	S 1 = = 12																			10 CS-US																	
Over-training	S 1 = = 12																			30 RS/US																	

across subjects (i.e., counterbalancing across subjects and within each subject's set of triads). Therefore, phase 1, adaptation training, in addition to reducing the possible influences of orienting reflexes in the test phase of experimentation, allowed the experimenter to demonstrate the comparability of unconditioned GSRs (for first, second, and third intervals) evoked by stimuli presented to three groups of subjects that would eventually receive one of the three kinds of conditioning treatments in the second phase of experimentation.

Phase 2, acquisition training, consisted of two separate classical conditioning treatments and one control condition. Thus, three groups of 12 human subjects each received one of the three conditioning procedures. One treatment intended to achieve overtraining was presented with 30 compound CS (light + tone)-US (arm shock) pairings, while a second, designed only to achieve the point of asymptotic learning, received 10 such pairings. Upon the completion of the experiment, analyses of the acquisition training data would provide a measure of how close the asymptotic and overtraining treatments actually came to obtaining their intended purpose. The third conditioning treatment was a truly random control in which 30 compound stimuli were presented in a similar manner to the other conditioning treatments (same $\overline{X}$ intertrial interval), but 30 US exposures were randomly interspersed throughout the entire conditioning period. The

minimum interstimulus interval for the shocks was 22.0 sec. In this situation, uncorrelated arm shocks occurred anytime before, during, or after the CS, thereby providing an unbiased baseline in testing aginst which the degree of conditioning for the overtrained stimuli could be compared. Other control treatments such as the explicitly unpaired control employ a negatively correlated CS, US relation (Rescorla, 1967) that might reduce the extent of unconditioned GSR activity to the control stimuli in testing (Prokasy, Williams, Kempfer, Lee, Jenson, 1973) and, therefore, exaggerate differences between the GSRs evoked by the overtrained and control stimuli. Such a distortion in the present work would preclude a fair test for the absence of conditioned excitation associated with the overtrained component stimuli in testing. Moreover, since the truly random control in testing would furnish greater sensitized responses than the same method of control with only 10 uncorrelated CS, US presentations, all asymptotic responses (first, second, and third interval GSRs) in testing could also be compared with those of the truly random control comparison. Successful conditioning demonstrated with these comparisons would occur in spite of a bias against the asymptotic treatment.

Phase 3, configurational testing, was identical to phase 1, adaptation training, and as previously suggested, would provide independent treatment comparisons that could demonstrate configurational learning according to the

previously stated criteria. Further, a comparison of experimental versus control treatment differences, from adaptation training to configurational testing, would ensure that differences in testing were in fact due to interpolated compound conditioning.

Procedure

Two subjects were run each day after being randomly assigned to one of the three experimental conditions, with the restriction that all treatments be balanced for gender toward the end of each week. Immediately following a subject's treatment allocation, the GSR electrodes were fixed to the ventral surface of the first and second fingers of the right hand. Shock electrodes, 10 mm apart, were also attached to the forearm muscle of the left arm for the administration of the US. The subjects were then taken from an anti-room and seated in the opened experimental chamber where the leads for GSR recording and shock stimulation were fitted to proper receptacles. Immediately thereafter, each subject received a series of 10 arm shocks in ascending steps of 0.2 ma in order to raise the subjects to the level of US intensity (2.0 ma) to be used in interpolated conditioning. The following instructions were then given:

> The purpose of this experiment is to evaluate the effects of subvocalization on a person's skin reactions to a tone, a light, a simultaneous light plus tone, and a harmless arm shock. For this purpose, it is crucial that you pronounce the name of

> each stimulus under your breath, without moving your lips during each stimulus presentation. This will be very difficult for the short duration shock stimulus but you must do your best for all stimulus presentations. The failure to subvocalize will show up on the skin reaction records as in lie detector tests and will invalidate your results. Therefore, your sincere participation is required until you are informed that the session is over. The electrodes attached to the fingers of your right hand will record the effects of subvocalization on your skin reactions to the above-mentioned stimuli. You will feel nothing through these recording electrodes. Just relax your right hand and keep it as still as possible throughout this experiment. However, you will receive shock through the electrodes attached to the left arm.

Upon completion of the post-shock instructions, the door to the experimental chamber was closed and a 2-minute rest period preceded the commencement of phase 1, adaptation training. Phase 2 (interpolated) compound conditioning treatments were initiated upon the presentation of the last adaptation stimulus. The end of phase 2 training marked the beginning of a 1-hour rest period designed to dissipate any massed training effect that might influence phase 3 test responses. The subjects had their electrode leads detached for this period and were accompanied about the laboratory by the experimenter for a duration of five minutes prior to being seated in the anti-room. In the last stages of the break, the stimulation and recording leads were reattached and the subjects were then returned to the experimental chamber. Two minutes after the closure of the chamber door, phase 3 testing began with two CS-US pairings which served as warm-up trials for the conditioning treatments. The

comparable control warm-up consisted of two random presentations with the CS and US. Following an experimental session, a fee of $5.00 was paid to the subject.

Dependent Variables

The three GSR intervals defined by Prokasy and Ebel (1967) were chosen for data analysis. The first interval was from 1.35 to 4.95 sec after CS onset, while the second interval was contained by the 4.95 and 9.53 sec points. The third interval fell between 9.53 and 14.55 sec from the CS onset. The first GSR within the first interval was accepted as the first interval response, but the response amplitude for the second and third response was defined as the peak resistance change reached within the second and third interval, independent of the number of humps occurring within an interval. Thus, the first interval response was always the first GSR chronologically, while what are called second and third responses were not always second and third humps in the GSR tracing. Out of all the trials given in the entire experiment, only 1.4% of them contained a second response within the first GSR interval. As in Prokasy and Ebel (1967), these second responses (chronologically) were counted as responses occurring within the second interval. In Appendix 1 are representative samples of first, second, and third interval GSRs which had occurred throughout the experiment.

All GSRs were scored from the point of stimulus onset in units of micromhos conductance change (Δc) which were later expressed as square root transformations in order to normalize response distributions. Micromhos conductance changes were calculated by counting lined divisions on the recording paper and, thereafter, locating the appropriate micromhos conductance change value on a plot of previously known values fed into the system for the purpose of calibration. The $\sqrt{\Delta c_s}$ for each kind of stimulation were analyzed in terms of GSR magnitude, which was the average response value for sets of trials with zero entries included. This scoring unit was one of several used by Prokasy and Ebel (1967), and reflected both probability and amplitude measures for a single set of trials.

RESULTS

Of the six presentations for each stimulus type (compound and component stimuli) in the adaptation and configurational test phases of experimentation, square root conductance changes (μ mhos) for the first and last three exposures were averaged and expressed as magnitude scores. In this way, all six trials for each stimulus condition in either adaptation training or configural testing (36 trials in all) contributed two magnitude scores which were eventually treated as trial block values. With this arrangement, it was possible to perform separate split plot analyses of variance for first,

second, and third responses in adaptation training and configural testing, having conditioning treatments (CT) as the between-subject source of variance, and trial blocks (TB) with stimulus conditions (SC) as the within-subject source of variance (3 X 2 X 3 split plot analysis of variance, Kirk, 1968). In Tables 3 through 8, are mean magnitudes and standard deviations for the split plot analysis of variance cell values which express the degree of electrodermal responding for first, second, and third responses in adaptation training and configural testing. F_{max} tests for all split plot matrices verified the homogeneity of variance assumption with the exception of the matrix for the second interval response in adaptation training. Heterogeneity of variance for second responses in adaptation training was the result of infrequent responding. However, because between-treatment comparisons with equal ns are little affected by heterogeneity of variance (Box, 1953), these data were analyzed.

Table 3

Mean Magnitude Scores and Standard Deviations for the First Response Analysis of Variance Cell Values in Adaptation Training

Groups	N		Trial Blocks 1 Cp	1 L	1 T	2 Cp	2 L	2 T
Random Control	12	$\bar{X}$	.609	.579	.472	.470	.435	.372
		SD	.308	.266	.272	.306	.292	.278
Asymptotic	12	$\bar{X}$	.573	.545	.498	.531	.441	.406
		SD	.232	.234	.243	.309	.258	.201
Overtrained	12	$\bar{X}$	.489	.434	.457	.544	.415	.386
		SD	.246	.227	.218	.303	.221	.302

Table 4

Mean Magnitude Scores and Standard Deviations for the Second Response Analysis of Variance Cell Values in Adaptation Training

Groups	N		Trial Blocks 1 Cp	1 L	1 T	2 Cp	2 L	2 T
Random Control	12	$\overline{X}$	.157	.142	.130	.069	.099	.046
		SD	.132	.118	.156	.100	.127	.079
Asymptotic	12	$\overline{X}$	.053	.022	.095	.080	.072	.052
		SD	.102	.058	.174	.123	.105	.111
Overtrained	12	$\overline{X}$	.173	.134	.111	.164	.096	.179
		SD	.161	.170	.174	.224	.176	.204

Table 5

Mean Magnitude Scores and Standard Deviations for the Third Response Analysis of Variance Cell Values in Adaptation Training

Groups	N		Trial Blocks 1 Cp	1 L	1 T	2 Cp	2 L	2 T
Random Control	12	$\bar{X}$	.132	.121	.103	.116	.082	.070
		SD	.117	.120	.078	.157	.150	.100
Asymptotic	12	$\bar{X}$	.206	.175	.191	.169	.096	.111
		SD	.202	.191	.235	.169	.093	.140
Overtrained	12	$\bar{X}$	.149	.207	.152	.178	.159	.121
		SD	.120	.186	.135	.150	.159	.114

Table 6

Mean Magnitude Scores and Standard Deviations for the First Response Analysis of Variance Cell Values in Configurational Testing

Groups	N		Trial Blocks 1 Cp	1 L	1 T	2 Cp	2 L	2 T
Random Control	12	$\bar{X}$	.726	.657	.538	.691	.763	.572
		SD	.194	.218	.182	.180	.260	.252
Asymptotic	12	$\bar{X}$	.622	.565	.511	.588	.501	.455
		SD	.359	.326	.294	.308	.324	.264
Overtrained	12	$\bar{X}$	.618	.487	.484	.635	.490	.473
		SD	.268	.187	.199	.245	.166	.251

Table 7

Mean Magnitude Scores and Standard Deviations for the Second Response Analysis of Variance Cell Values in Configurational Testing

Groups	N		Trial Blocks 1 Cp	1 L	1 T	2 Cp	2 L	2 T
Random Control	12	$\bar{X}$	.121	.074	.174	.128	.135	.126
		SD	.152	.100	.178	.175	.167	.175
Asymptotic	12	$\bar{X}$	.421	.248	.227	.339	.212	.164
		SD	.266	.237	.165	.263	.240	.188
Overtrained	12	$\bar{X}$	.357	.114	.247	.369	.088	.117
		SD	.247	.131	.178	.212	.108	.145

Table 8

Mean Magnitude Scores and Standard Deviations for the Third Response Analysis of Variance Cell Values in Configurational Testing

Groups	N		Trial Blocks 1 Cp	1 L	1 T	2 Cp	2 L	2 T
Random Control	12	$\bar{X}$	.200	.204	.099	.270	.197	.164
		SD	.182	.137	.118	.238	.163	.125
Asymptotic	12	$\bar{X}$	.490	.287	.282	.425	.172	.221
		SD	.249	.216	.172	.282	.179	.205
Overtrained	12	$\bar{X}$	.467	.236	.297	.357	.192	.130
		SD	.212	.140	.171	.174	.160	.115

Adaptation Phase

The split plot analysis of variance for first responses in adaptation training (Table 9 and Figure 2) yielded a significant trial block ($F(1,33)=6.379$, $p < .05$); and stimulus condition effect ($F(2,66)=19.443$, $p < .01$). The means for the first ($\bar{X}=.518$) and second ($\bar{X}=.445$) trial blocks indicated OR adaptation, while the means for stimulus conditions (Figure 3) showed that the compound stimulus ($\bar{X}=.535$) evoked the largest first interval magnitude. Tukey's test for pairwise comparisons (α .05, HSD=.040) of means held the compound response significantly greater than the light ($\bar{X}=.474$) reaction which, in turn, surpassed the response level for the tone ($\bar{X}=.431$).

Identical analyses of variance for the second and third interval GSRs in adaptation training revealed no significant differences, save a trial block effect ($F(1,33)=4.642$, p .05) for third period reactions (Table 10 and Figure 4). These GSRs showed a decrease in magnitude from the first trial block ($\bar{X}=.159$) to the second ($\bar{X}=.123$).

Test Phase

The split plot analysis of variance for the first interval test results (Table 11 and Figure 5) was similar to that in adaptation, as only a stimulus condition effect arose ($F(2,66)=16.461$, p .01). When the means for the

Table 9

Significant F Ratios for CT X TB X SC Analysis of Variance - First Interval GSR Magnitude in Adaptation

Source	df	MS	F	F .05/.01*
TB	1	.286	6.379**	4.17/7.56
TB,R(CT)[a]	33	(.045)		
SC	2	.196	19.443***	4.17/7.56
SC,R(CT)[a]	66	(.010)		

*Conservative F, Test Levels (Geisser-Greenhouse).
**p < .05
***p < .01
[a]Error MSs in parentheses

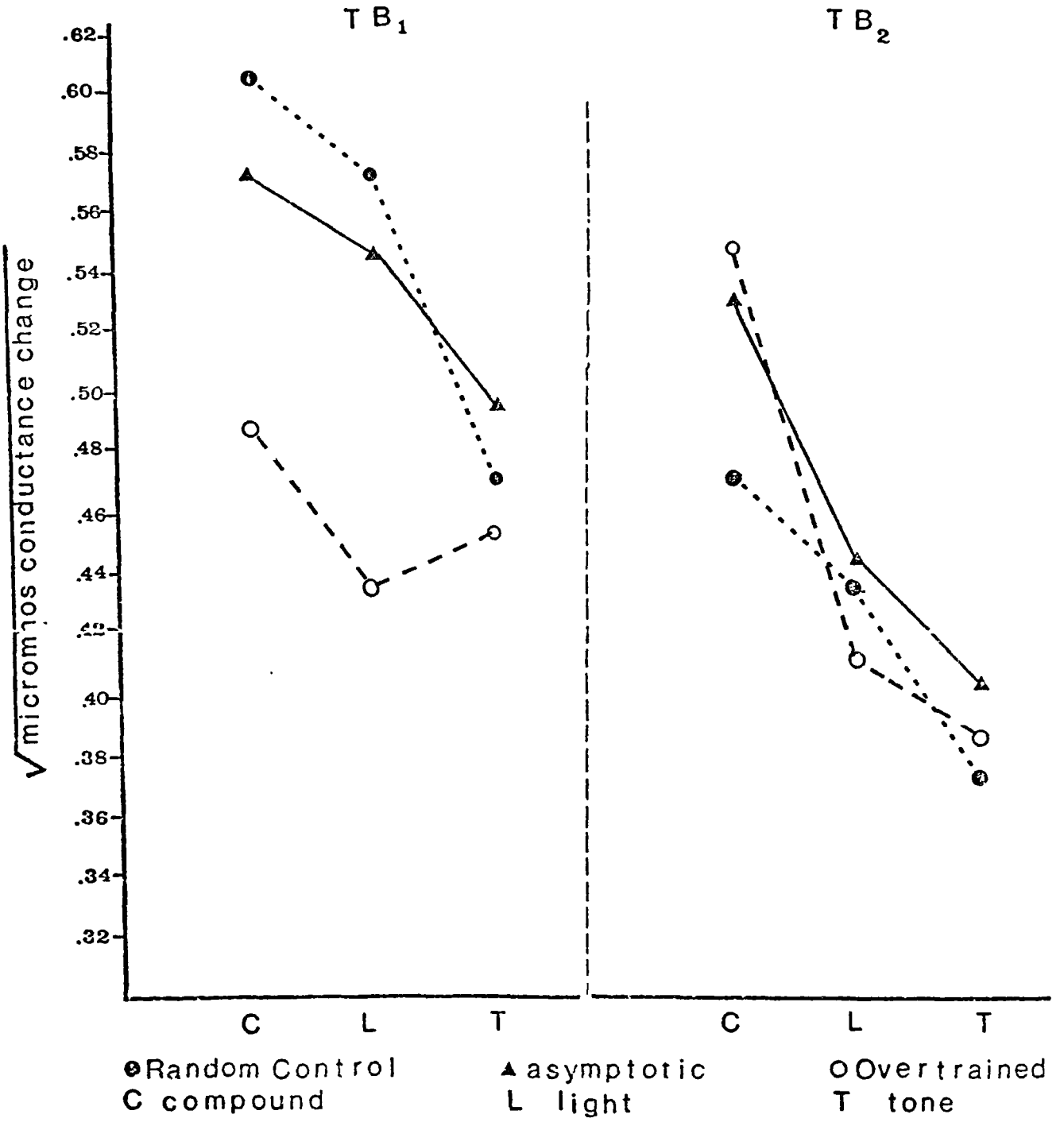

Figure 2. Mean Magnitudes for First Interval GSRs Evoked by Compound and Component Stimuli Presented to Three Treatment Groups during the Two Trial Blocks of Adaptation Training.

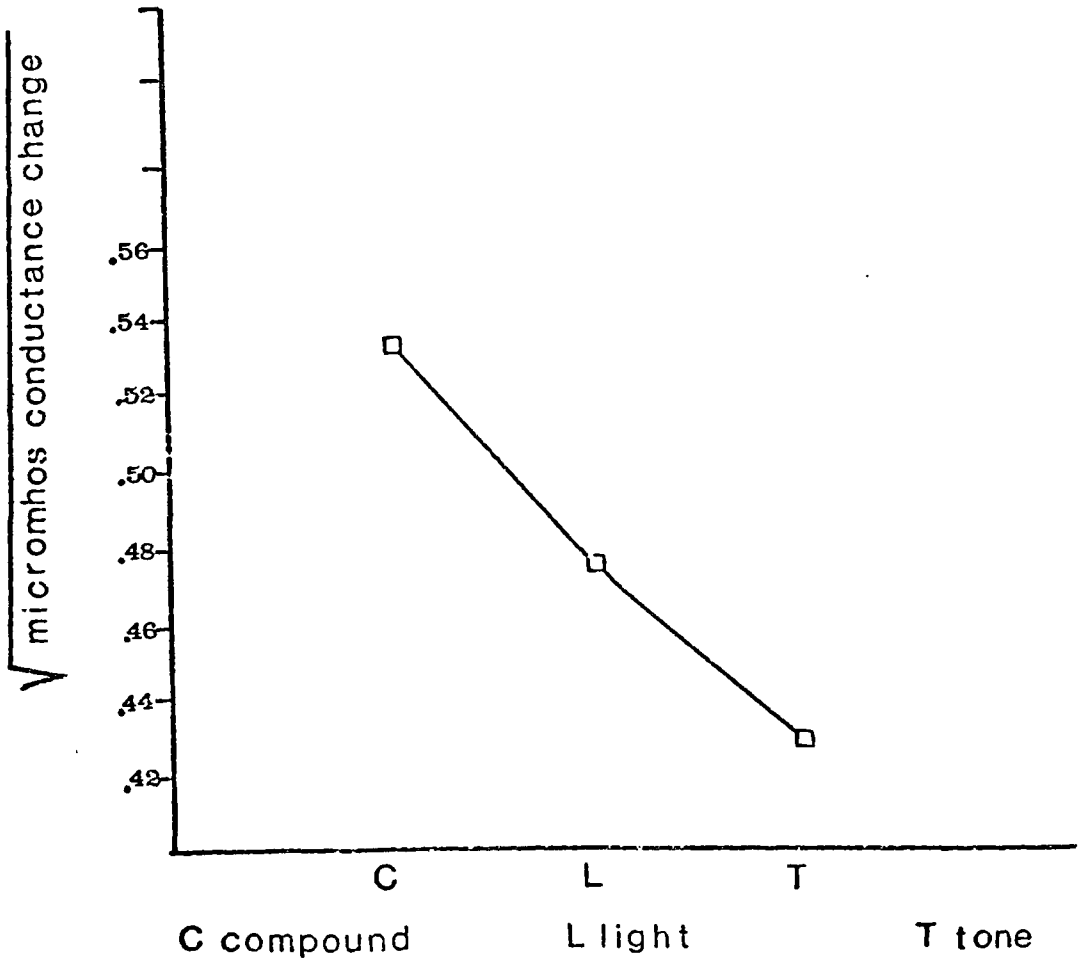

Figure 3. Overall Mean Magnitudes for First Interval GSRs Evoked by the Compound and Component Stimuli in Adaptation Training.

Table 10

Significant F Ratio for CT X TB X SC Analysis of Variance - Third Interval GSR Magnitude in Adaptation

Source	df	MS	F	F .05/.01*
TB	1	.074	4.642**	4.17/7.56
TB,R(CT)[a]	33	(.016)		

*Conservative F, Test α Levels (Geisser-Greenhouse)
**p <.05
***p <.01
[a]Error MS in parentheses

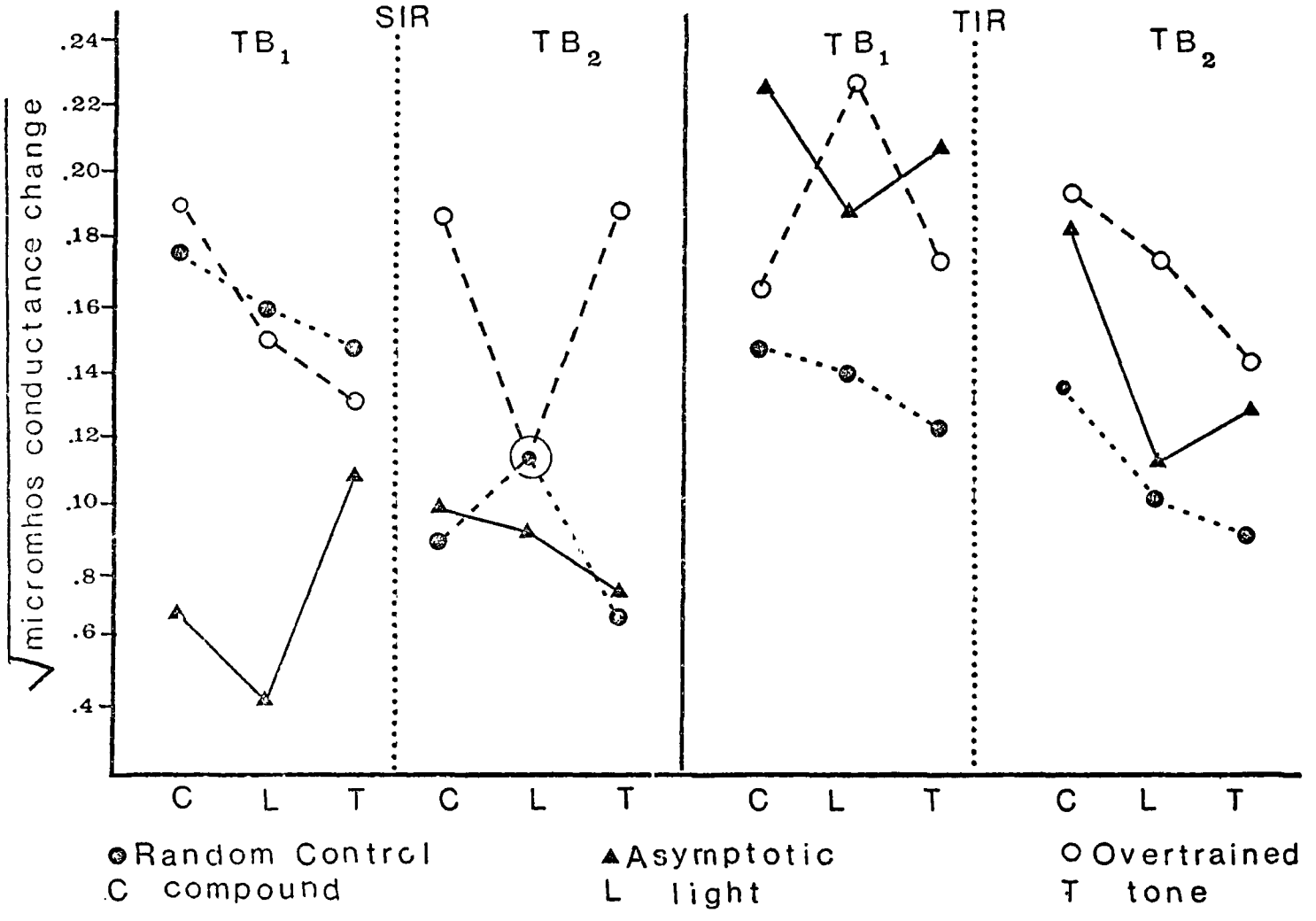

Figure 4. Mean Magnitudes for Second and Third Interval GSRs Evoked by the Compound and Component Stimuli Presented to Three Treatment Groups during the Two Trial Blocks of Adaptation Training.

Table 11

Significant F Ratio for CT X TB X SC Analysis of Variance - First Interval GSR Magnitude in Testing

Source	df	MS	F	F .05/.01*
SC	2	.359	16.461**	4.17/7.56
SC,R(CT)[a]	66	(.022)		

*Conservative F, Test α Levels (Geisser-Greenhouse)
**$p < .05$
***$p < .01$
[a]Error MS in parentheses

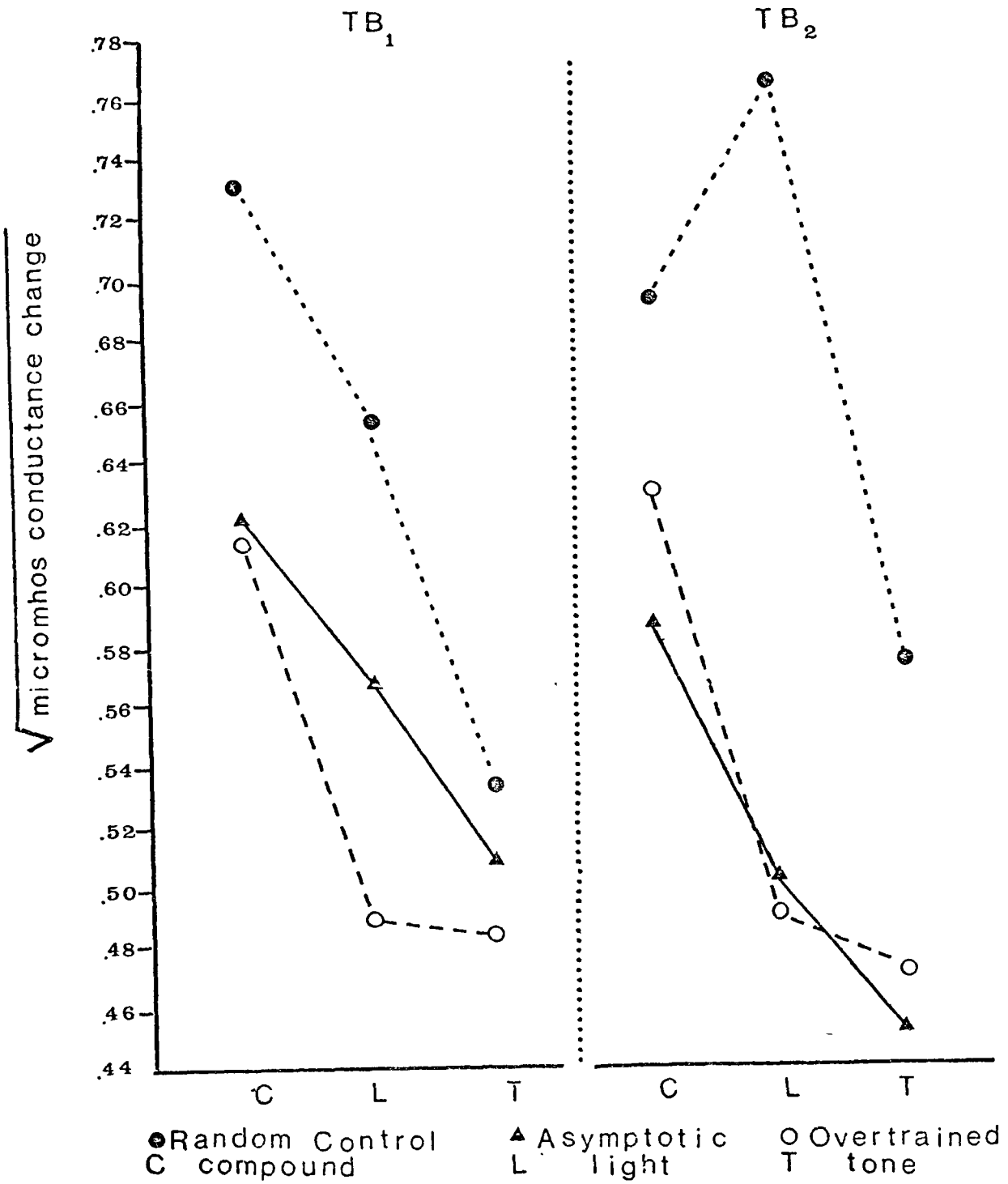

Figure 5. Mean Magnitudes for First Interval GSRs Evoked by Compound and Component Stimuli Presented to Three Treatment groups during the Two Blocks of Configurational Testing.

compound ($\bar{X}$=.646) and component reactions (light $\bar{X}$=.576, tone $\bar{X}$=.506) were compared through Tukey's pairwise method (α .05, HSD=.058), the compound performance was found superior to the reaction level of the light component, which in turn exceeded the tone response (Figure 6).

The same analysis of variance for the second interval GSR test data (Table 12 and Figure 7) produced a highly significant stimulus condition result ($F(2,66)=15.821$, $p < .01$); and conditioning treatment X stimulus condition interaction ($F(4,66)=4.998$, $p < .01$). Analysis for simple effects (Table 12 and Figure 8) showed that the second responses evoked by compound stimuli differed between the conditioning treatments ($F(2,99)=8.00$, $p < .01$), while the same electrodermal responses for either component stimulus did not differ between groups [light ($F(2,99)=2.18$, $p > .05$), tone ($F(2,99)=.196$, $p > .05$)]. The latter result seemed to rule out the possibility of component CR diminution in the overtrained conditioning treatment. Pairwise contrasts of treatment means (Tukey α .05, HSD=.170) for the compound second responses emphasized the similarity of conditioned performances between the asymptotic ($\bar{X}$=.379) and overtrained ($\bar{X}$=.362) subjects, as both treatment groups were clearly superior to the control comparison ($\bar{X}$=.124).

Degree of compound response dominance over component levels within each treatment provided a second important set of simple effects for second interval GSRs (Table 12 and

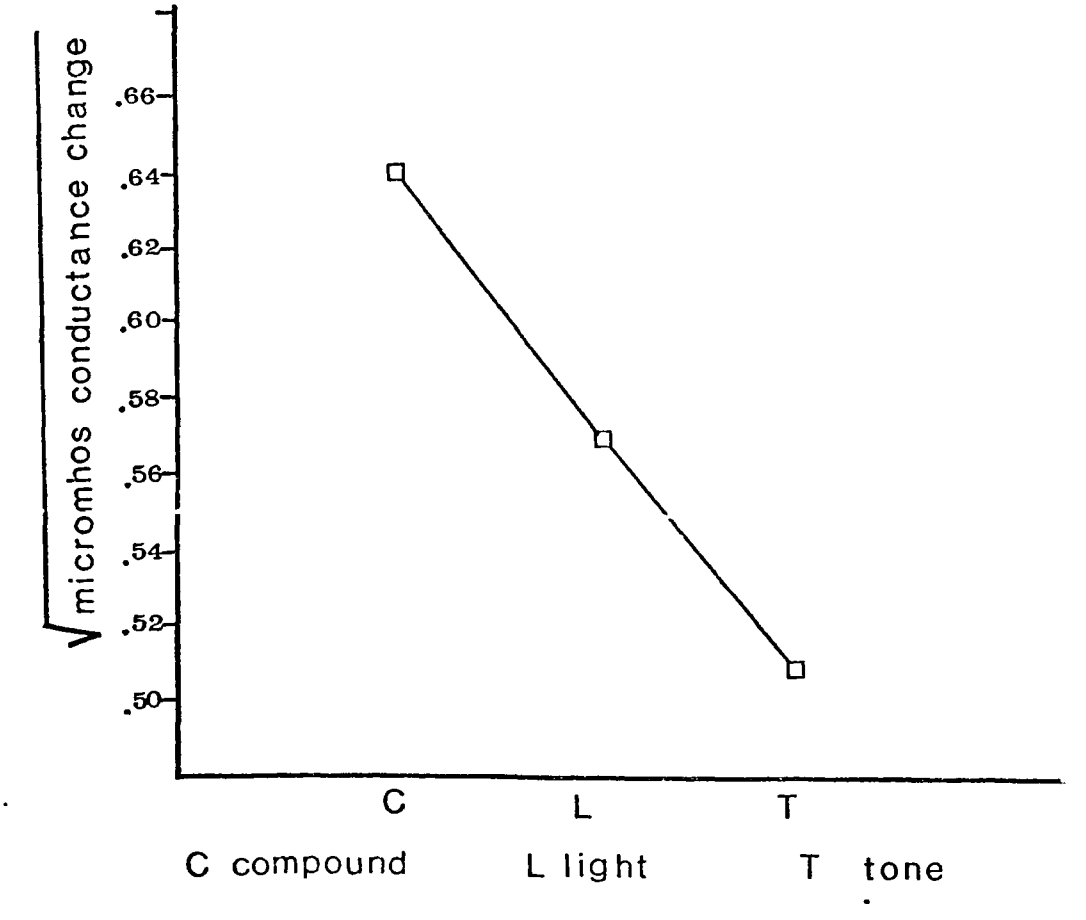

Figure 6. Overall Mean Magnitudes for First Interval GSRs Evoked by the Compound and Component Stimuli in Configurational Testing.

Table 12

Significant F Ratios for CT X TB X SC Analysis of Variance - Second Interval GSR Magnitude in Testing

Source	df	MS	F	F .05/.01*
SC	2	.413	15.821***	4.17/7.56
CT X SC	4	.130	4.998**	3.32/5.39
SC,R(CT)[a]	66	(.025)		
Simple Effects				
CT_i at Compound CS	2	.488	8.00***	
CT_i at Light Component	2	.133	2.18	
CT_i at Tone Component	2	.012	0.196	
R(CT)+SC,R(CT)[a]	99	(.061)		
SC_i in Asymptotic,CT	2	.227	9.08***	
SC_i in Overtrained,CT	2	.430	17.20***	
SC_i in Control,CT	2	.013	0.52	
SC,R(CT)	66	(.025)		

*Conservative F, Test α Levels (Geisser-Greenhouse). Simple effects from R. E. Kirk, Experimental design: Procedures for the behavioral sciences. Belmont, California: Wadsworth, 1968, p. 305.

**$p < .05$

***$p < .01$

[a]Error MS in parentheses

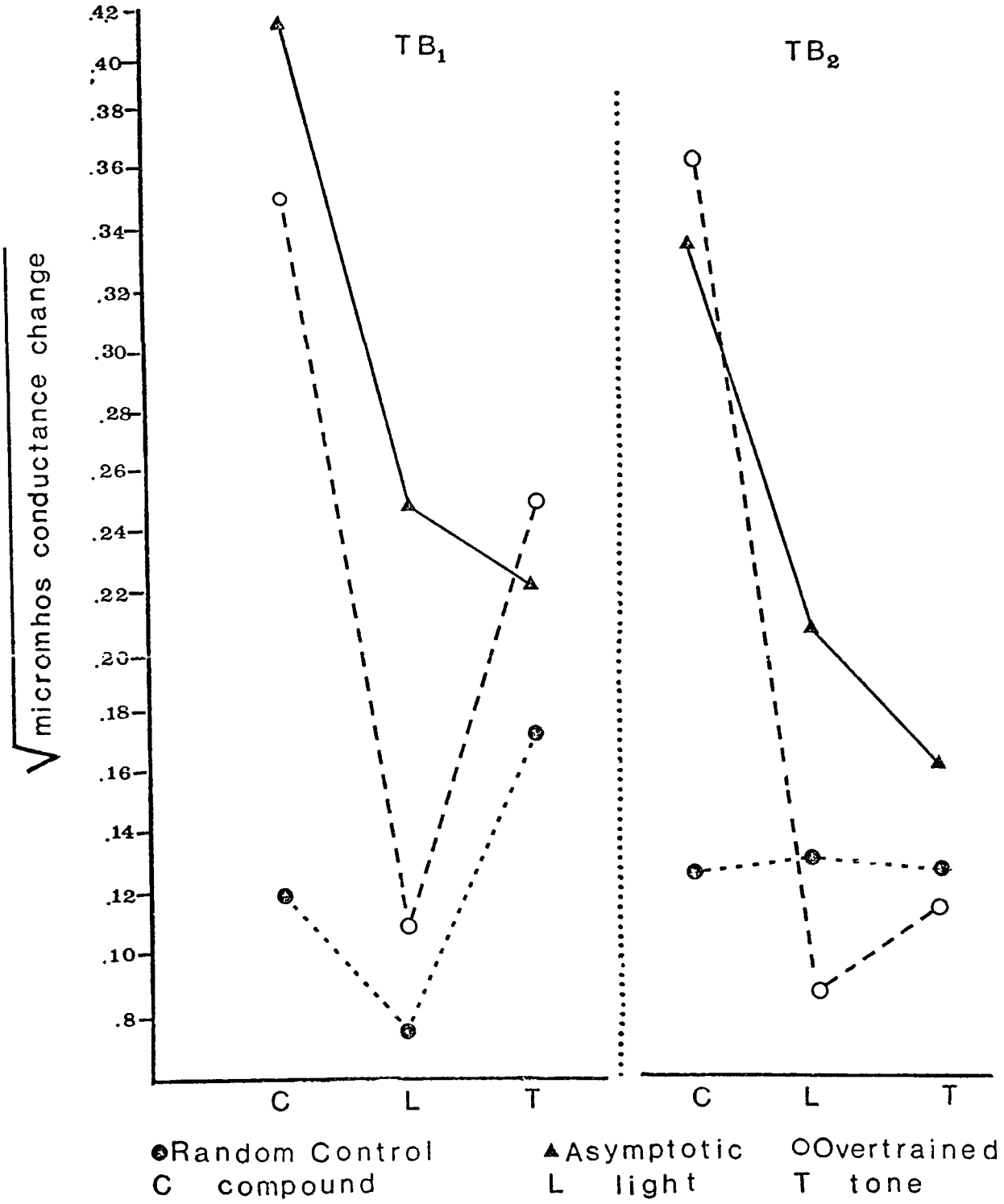

Figure 7. Mean Magnitudes for Second Interval GSRs Evoked by Compound and Component Stimuli Presented to Three Treatment Groups during the Two Trial Blocks of Configurational Testing.

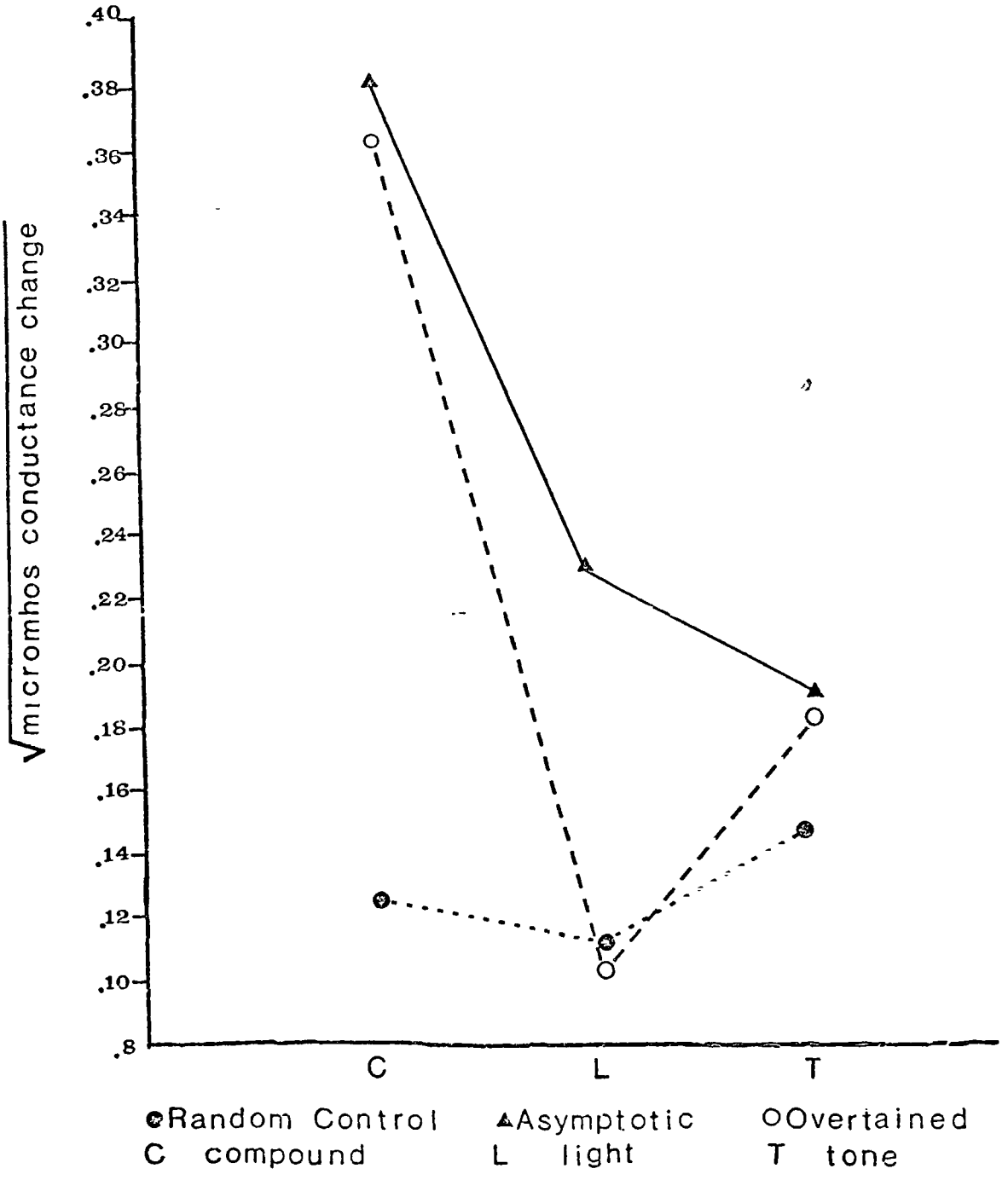

Figure 8. Mean Magnitude, with Trial Blocks Combined, for Second Interval GSRs Evoked by Compound and Component Stimuli Presented to Three Treatment Groups during Configurational Testing.

Figure 8). Here, both the asymptotic and overtrained conditioning treatments possessed a significant difference for stimulus conditions ($\underline{F}(2,66)=9.08$, $p < .01$); ($\underline{F}(2,66)=17.20$, $p < .01$). As anticipated, control subjects did not differentially respond to the compound and component stimuli ($\underline{F}(2,66)=0.52$, $p > .05$). Pairwise contrasts of means (Tukey α .05, HSD=.110) underscored a clear compound conditioning outcome, with the compound response level for the asymptotically conditioned group ($\overline{X}=.379$) and overtrained subjects ($\overline{X}=.362$) exceeding their respective component reactions (light $\overline{X}=.231$, tone $\overline{X}=.195$; light $\overline{X}=.100$, tone $\overline{X}=.183$).

Significant third interval GSR variances for the split plot analysis (Table 13 and Figure 9) were associated with trial blocks ($\underline{F}(1,33)=4.234$, $p < .05$); stimulus conditions ($\underline{F}(2,66)=24.626$, $p < .01$); the treatment X trial block interaction ($\underline{F}(2,33)=3.877$), $p < .05$); and the treatment X stimulus condition interaction ($\underline{F}(4,66)=2.581$, $p < .05$). Significant simple effects for these interactions (Table 13) consisted of two kinds of conditioning treatment differences. One, with combined stimulus conditions (Figure 10), occurred within trial block 1 ($\underline{F}(2,66)=5.857$, $p < .01$); and the other (Figure 11) was for compound responses alone ($\underline{F}(2,99)=6.600$, $p < .01$). Pairwise contrasts (Tukey α.05, HSD=.142) for conditioning treatment means in trial block 1 established response dominance for the asymptotic ($\overline{X}=.354$) and overtrained ($\overline{X}=.332$) subjects over controls ($\overline{X}=.167$). The same

Table 13

Significant F Ratios for CT X TB X SC Analysis of Variance - Third Interval GSR Magnitude in Testing

Source	df	MS	F	F .05/.01*
TB	1	.123	4.234**	4.17/7.56
CT X TB	2	.113	3.877**	3.32/5.39
TB,R(CT)[a]	33	(.029)		
SC	2	.628	24.626***	4.17/7.56
CT X SC	4	.066	2.581	3.32/5.39
SC,R(CT)[a]	66	(.026)		
Simple Effects				
CT_i at TB_1	2	.376	5.875***	
CT_i at TB_2	2	.034	0.531	
R(CT)+TB,R(CT)[a]	66	(.064)		
CT_i at Compound CS	2	.330	6.600***	
CT_i at Light Component	2	.006	0.120	
CT_i at Tone Component	2	.090	1.800	
R(CT)+SC,R(CT)[a]	99	(.050)		
TB_i in Asymptotic,CT	1	.118	4.214**	
TB_i in Overtrained,CT	1	.196	7.00**	
TB_i in Control,CT	1	.035	1.25	
TB,R(CT)[a]	33	(.028)		
SC_i in Asymptotic,CT	2	.381	14.64***	
SC_i in Overtrained,CT	2	.310	11.91***	
SC_i in Control,CT	2	.066	2.54	
SC,R(CT)[a]	66	(.026)		

*Conservative F, test α Levels (Geisser-Greenhouse). Simple effects from R. E. Kirk, Experimental design: Procedures for the behavioral sciences. Belmont, California: Wadsworth, 1968, p. 305.

**p <.05

***p <.01

[a]Error MS in parentheses

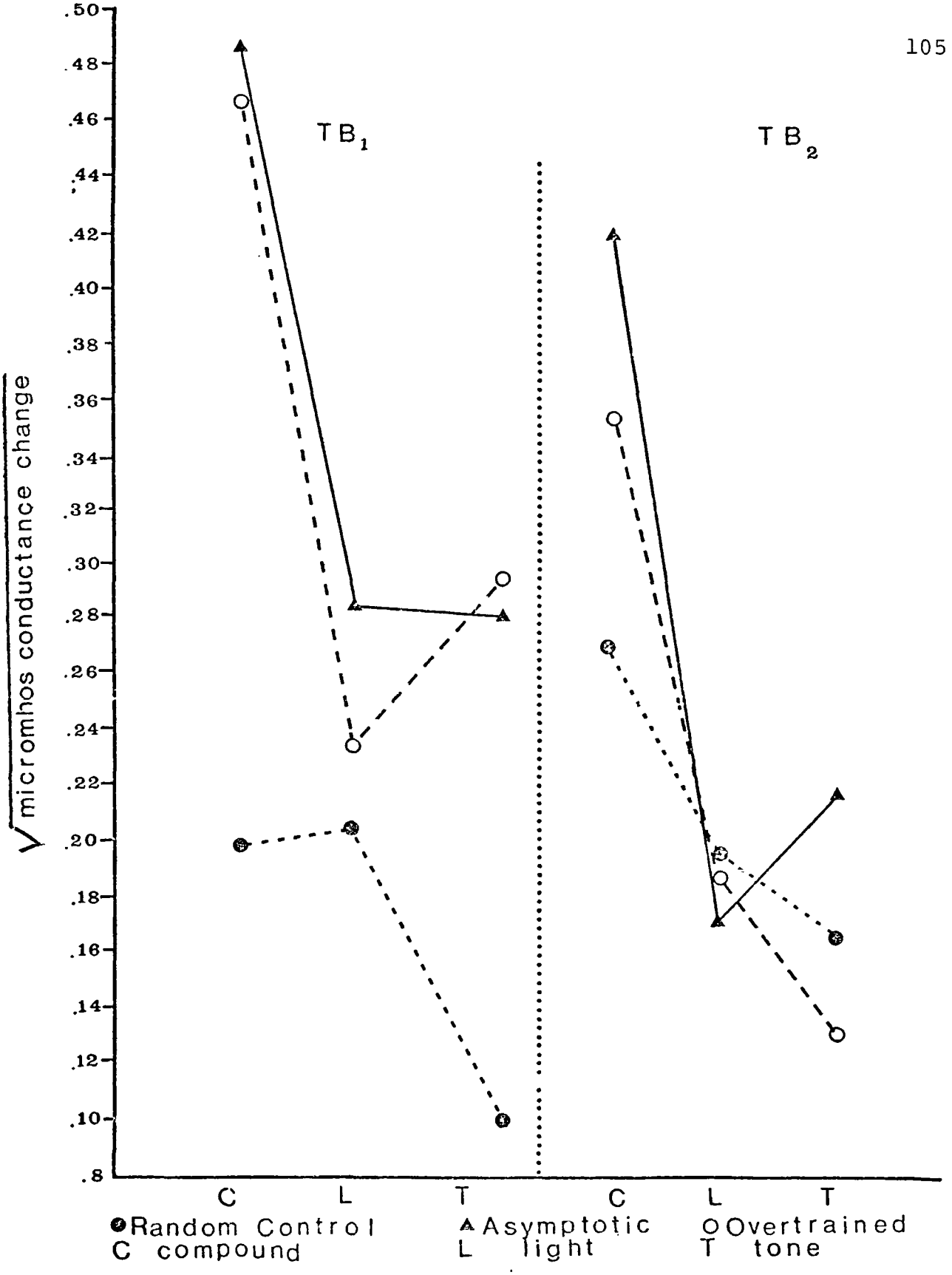

Figure 9. Mean Magnitudes for Third Interval GSRs Evoked by Compound and Component Stimuli Presented to Three Treatment Groups during the Two Trial Blocks of Configurational Testing.

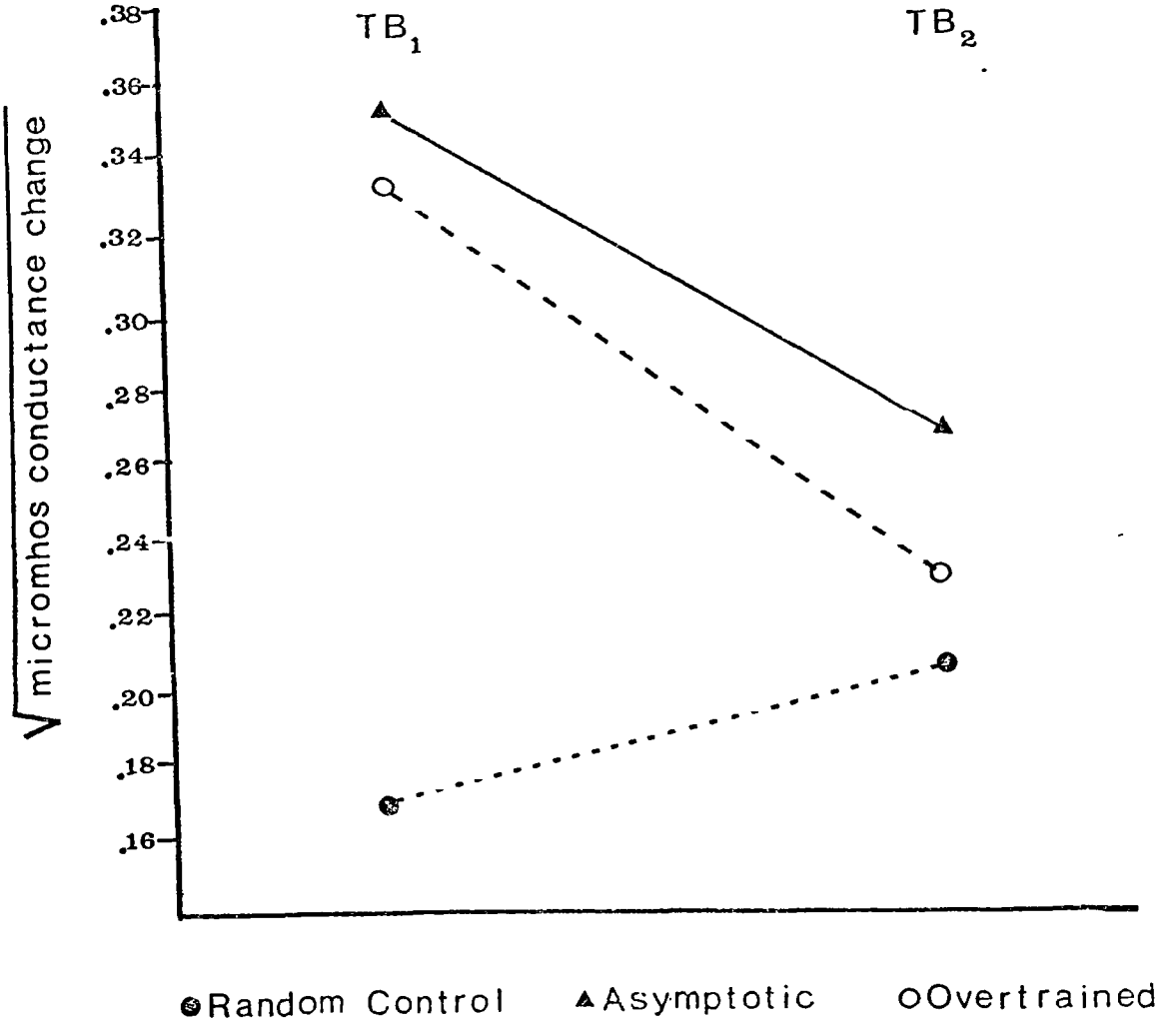

Figure 10. Mean Magnitudes, with Stimulus Conditions Combined, for Third Interval GSRs of Each Treatment Group from the First Trial Block to the Second in Configurational Testing.

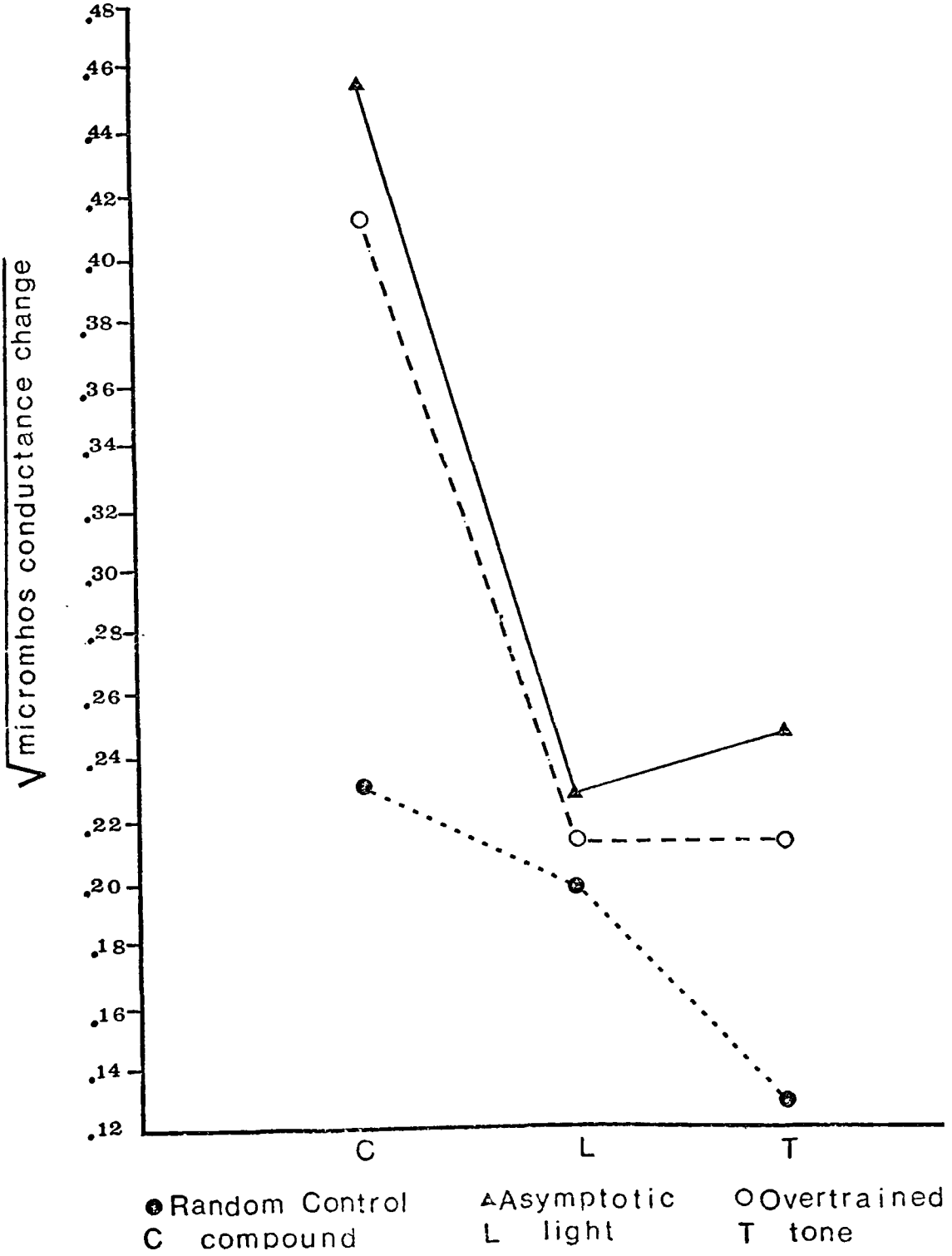

Figure 11. Mean Magnitudes, with Trial Blocks Combined, for Third Interval GSRs Evoked by the Compound and Component Stimuli Presented to Three Treatment Groups during Configurational Testing.

method of comparison (Tukey α .05, HSD=.154) for compound responses between conditioning treatments manifested the same result (asymptotic $\bar{X}$=.458, overtrained $\bar{X}$=.411, control $\bar{X}$=.234), thereby substantiating compound conditioning for the third interval response. The overall trial block effect showed a decrease in responding from the first trial block ($\bar{X}$=.284) to the second ($\bar{X}$=.237), but related tests for simple effects (Table 13 and Figure 10) revealed that only the overtrained and asymptotic treatments showed an overall third response reduction across trial blocks ($F(1,33)=7.00$, $p < .05$); ($F(1,33)=4.214$, $p < .05$). Out of the remaining simple effects for the treatment X stimulus condition interaction (Table 13 and Figure 11), differences between stimulus conditions were found within the asymptotic ($F(2,66)=14.64$, $p < .01$), and overtrained ($F(2,66)=11.91$, $p < .01$) conditioning treatments, but not for the control measures ($F(2,66)=2.54$, $p < .05$). Comparisons (Tukey α .05, HSD=.112) of stimulus condition means for the asymptotic conditioning treatment (compound $\bar{X}$=.458, light $\bar{X}$=.229, tone $\bar{X}$=.251) and overtrained subjects (compound $\bar{X}$=.411, light $\bar{X}$=.213, tone $\bar{X}$=.214) agreed with the third interval GSR conditioning result above, when the nonsignificant control finding was considered.

Combined Adaptation and Test Phase Analysis

To demonstrate configurational learning according to all the previously stated criteria, and at the same time compare stimulus conditions within and between treatments ,

from adaptation training to configurational testing, analyses of variance with conditioning treatments as the between-subject source of variance and experimental phases with stimulus conditions as the within-subject source of variance were carried out separately for first, second, and third interval GSRs. For these analyses, it was decided to employ magnitude scores based on all six trials for each stimulus condition, so that each subject contributed a single magnitude score per stimulus condition in adaptation training and configurational testing, yielding a 3 X 2 X 3 analysis of variance. In addition, the single magnitude scores for each stimulus condition in testing would allow the computation of the degree of correlation between first and second (or third) component responses in testing to assess the orienting reflex interpretation of component CR diminution. Because adaptation (with first and third responses) and extinction effects (on third responses) in previous split plot analyses did not interact with stimulus condition responding, there seemed no serious reason prohibiting the analysis of magnitude scores based on all six trials for each stimulus condition.

In this split plot analysis, compound conditioning would be detected by a CT X EP X SC triple interaction produced by a significant increase in compound magnitude scores for the asymptotic and overtrained treatments over the control value, from adaptation training to configurational

testing. Simple-simple effects for an anticipated CT X SC interaction in testing would reveal the degree of compound conditioning and indicate whether or not component CR diminution had occurred in the overtrained treatment relative to the asymptotic values. In addition, the extent of conditioned excitation associated with the overtrained components could be evaluated with comparable control measures. As it happened, this triple interaction was only significant in the analysis of variance for the second interval response ($F(4,66)=4.162$, $p < .01$) (Table 14 and Figure 12). In Table 15 are the mean magnitudes and standard deviations for the second response analysis of variance cell values. Simple effect analyses (Table 14 and Figure 12) did show a significant CT X SC interaction in the test phase (EP_2) alone ($F(4,132)=6.88$, $p < .01$). The simple-simple effects between conditioning treatments (Table 14 and Figure 12) were only significant for the compound responses ($F(2,198)=11.67$, $p < .01$ and component reactions to the light ($F(2,198)=3.047$), $p < .05$). Tukey pairwise comparisons (α .05, HSD=.137) for these means showed that the compound second responses for the asymptotic ($\bar{X}=.379$) and overtrained ($\bar{X}=.364$) conditioning treatments did exceed the control value ($\bar{X}=.124$), while it was the component response level of the asymptotic group (light $\bar{X}=.229$) that marginally surpassed both the overtrained (light $\bar{X}=.101$) and control (light $\bar{X}=.104$) component values. These results

Table 14

Significant F Ratios for CT X EP X SC Analysis of Variance - Second Interval GSR Magnitude in Adaptation and Testing

Source	df	MS	F	F .05/.01*
CT X EP X SC	4	.037	4.162***	3.32/5.39
EP,SC,R(CT)[a]	66	(.009)		
Simple Effects				
CT X SC in EP_1	4	.003	.333	
CT X SC in EP_2	4	.062	6.88***	
SC,R(CT)+EP,SC,R(CT)[a]	132	(.009)		
Simple-Simple Effects				
CT_i at Compound CS	2	.245	11.67***	
CT_i at Light Component	2	.064	3.047**	
CT_i at Tone Component	2	.004	0.190	
MS w. Cell[a]	198	(.021)		
SC_i in Asymptotic,CT	2	.114	12.66***	
SC_i in Overtrained,CT	2	.216	24.00***	
SC_i in Control,CT	2	.006	0.666	
SC,R(CT)+EP,SC,R(CT)[a]	132	(.009)		

*Conservative F, Test α Levels (Geisser-Greenhouse). Simple effects from R. E. Kirk, Experimental design: Procedures for the Behavioral Sciences. Belmont, California: Wadsworth, 1968, p. 305.

**p <.05

***p <.01

[a]Error MS in parentheses

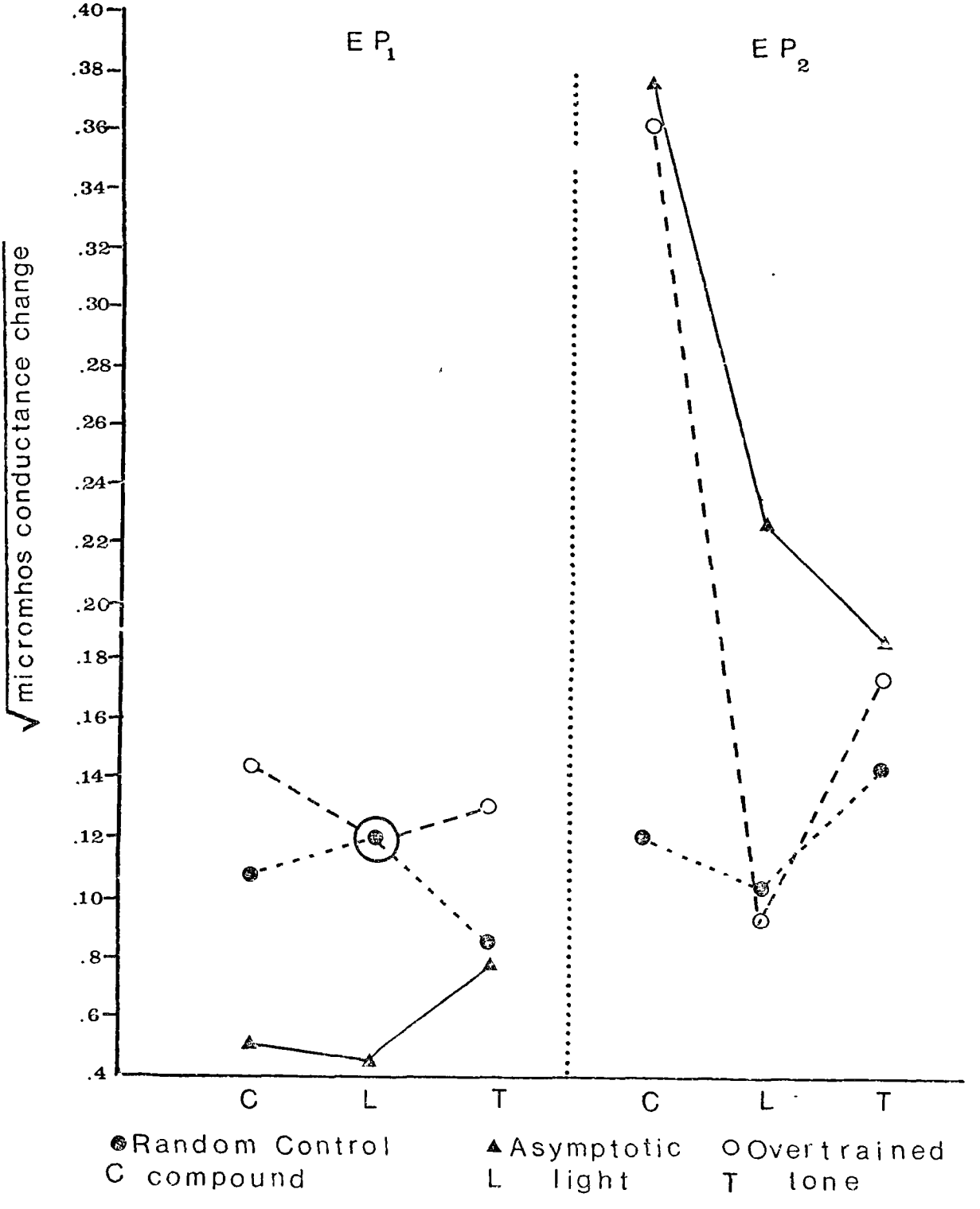

Figure 12. Mean Magnitudes for Second Interval GSRs Evoked by Compound and Component Stimuli Presented to Three Treatment Groups during Adaptation Training and Configurational Testing.

Table 15

Mean Magnitude Scores and Standard Deviations for the Second Response Analysis of Variance Cell Values in Adaptation Training and Configurational Testing

Groups	N		Trial Blocks: Adaptation Training Cp	L	T	Configurational Testing Cp	L	T
Random Control	12	$\bar{X}$	.113	.120	.087	.125	.104	.150
		SD	.104	.095	.102	.144	.111	.155
Asymptotic	12	$\bar{X}$	.058	.047	.081	.380	.230	.196
		SD	.091	.071	.136	.257	.220	.164
Overtrained		$\bar{X}$	.146	.120	.140	.363	.111	.182
		SD	.134	.134	.137	.206	.110	.143

did reflect compound conditioning with partial component CR diminution. The compound conditioning result was consistent with significant simple-simple stimulus condition effects found within the asymptotic ($F(2,132)=12.66$, $p < .01$), and overtrained ($F(2,132)=24.00$, $p < .01$) groups (Table 14 and Figure 12). Comparison of means for stimulus conditions (Tukey α .05, HSD=.091) within these treatments showed that the compound second responses in the asymptotic ($\bar{X}=.379$) and ovetrained ($\bar{X}=.364$) treatments surpassed their respective component response magnitudes (light $\bar{X}=.229$, tone $\bar{X}=.195$; light $\bar{X}=.101$, tone $\bar{X}=.182$). The control values for stimulus conditions did not vary between each other ($F(2,132)=.666$, $p < .05$). The same tests for simple-simple effects were carried out with comparable within and between-subject comparisons for the second interval GSRs in adaptation training, even though no conditioning treatment and stimulus condition interaction occurred. As expected, no differences were uncovered.

It has been previously stated that the truly random control treatment with 30 US and 30 compound CS trials should produce a greater amount of sensitization than a comparable control comparison for the asymptotic treatment. The potential difference that might emerge between these control comparisons, with regard to the compound test reactions, could be ruled out if it can be shown that no sensitization occurs for the three electrodermal responses

of the truly random control treatment (30 CS, 30 US trials), from adaptation training to configurational testing. For this purpose, three separate one-way analyses of variance were performed on first, second, and third interval GSRs in order to compare compound magnitude scores which expressed the average response value for the last three compound control trials in adaptation training and the first three compound control trials in configurational testing. No differences between the control compound reactions in adaptation training and configurational testing for first, second, and third response magnitudes would demonstrate the lack of sensitization in the present experiment and establish the truly random control comparison (30 CS, 30 US trials) as an accurate contrast condition to assess conditioning in the asymptotic treatment. As it turned out, the first and third responses of the compound stimulus in testing were significantly greater than those in adaptation training ($F(1,11)=5.8378$, $p < .01$; $F(1,11)=4.893$, $p < .05$). Compound magnitude scores for the second electrodermal response failed to differ between adaptation training and testing ($F(1,11)=1.844$, $p < .05$). Thus, at least for the second response, the truly random control was an unbiased comparison with which to evaluate conditioning in the asymptotic treatment.

Only the second interval response associated with the light component revealed a trend toward component CR

diminution in the previous analysis with a clearer manifestation of this result in the last. Therefore, only the magnitude scores for first and second component responses within the asymptotic and overtrained treatment were correlated. Correlations obtained between the first and second responses in the overtrained treatment were $\rho=-.188$ for the light, and $\rho=+.226$ for the tone. The same correlation coefficients for the asymptotic treatment were $\rho=-.477$ for the light component, and $\rho=+.471$ for the tone component. Students' t-test failed to indicate significance at the .05 alpha level for all correlation coefficients.

Acquisition Training

In the present experiment, it was of interest to determine whether or not the test phase reflected compound conditioning that could be demonstrated during acquisition training. However, in training, uncorrelated US presentations for the truly random control treatment sometimes occurred within five seconds prior to the CS, or within 14.95 sec after CS onset and, therefore, these trials could not be used to verify conditioning. Thus, the first and second responses ($\sqrt{\Delta CS}$) for all conditioning treatments were averaged over blocks of five conditioning trials, but control responses occurring on CS trials that were immediately preceded by a random shock, or those in which such shocks occurred, were deleted from the successive trial block

averages. In this manner, successful compound conditioning during training could be tested.

The first trial block analysis compared the first response between the overtrained and control treatment over six successive trial blocks (30 compound conditioning trials). A treatment X trial block analysis of variance (Table 16), which compared trial block means, did produce a trial block effect ($\underline{F}(5,110)=4.450$, $p < .01$), but no other. Trial block means for individual and combined treatments are plotted in Figure 13, and show that the trial block effect was the result of a decrease in trial block responding from the beginning of training to the end. The same treatment X trial block analysis of variance for compound second responses (Table 17) yielded both a treatment ($\underline{F}(1,22)=11.860$, $p < .01$) and trial block effect ($\underline{F}(5,110)=3.580$, $p < .01$) in the absence of an interaction. Figure 14, which contains individual and combined treatment trial block means, shows a decrease in second interval responding for combined treatments which was similar to that with the first response.

When post hoc tests were used to compare individual trial block means between treatments, it was revealed that all six trial blocks for first interval responding failed to differ between the overtrained and control treatments, with the exception of the first trial block (Tukey $\alpha.05$,

Table 16

F Ratios in the Treatment X Trial Block Analysis of Variance for First Interval Responses During Compound Conditioning

Source	*df*	*MS*	*F*
CT	1	.707	1.870
R(CT)	22	(.378)[a]	
TB	5	.089	4.450**
CT X TB	5	.015	.750
TB,R(CT)	110	(.020)[a]	

**p <.01

[a]Error *MS* in parentheses

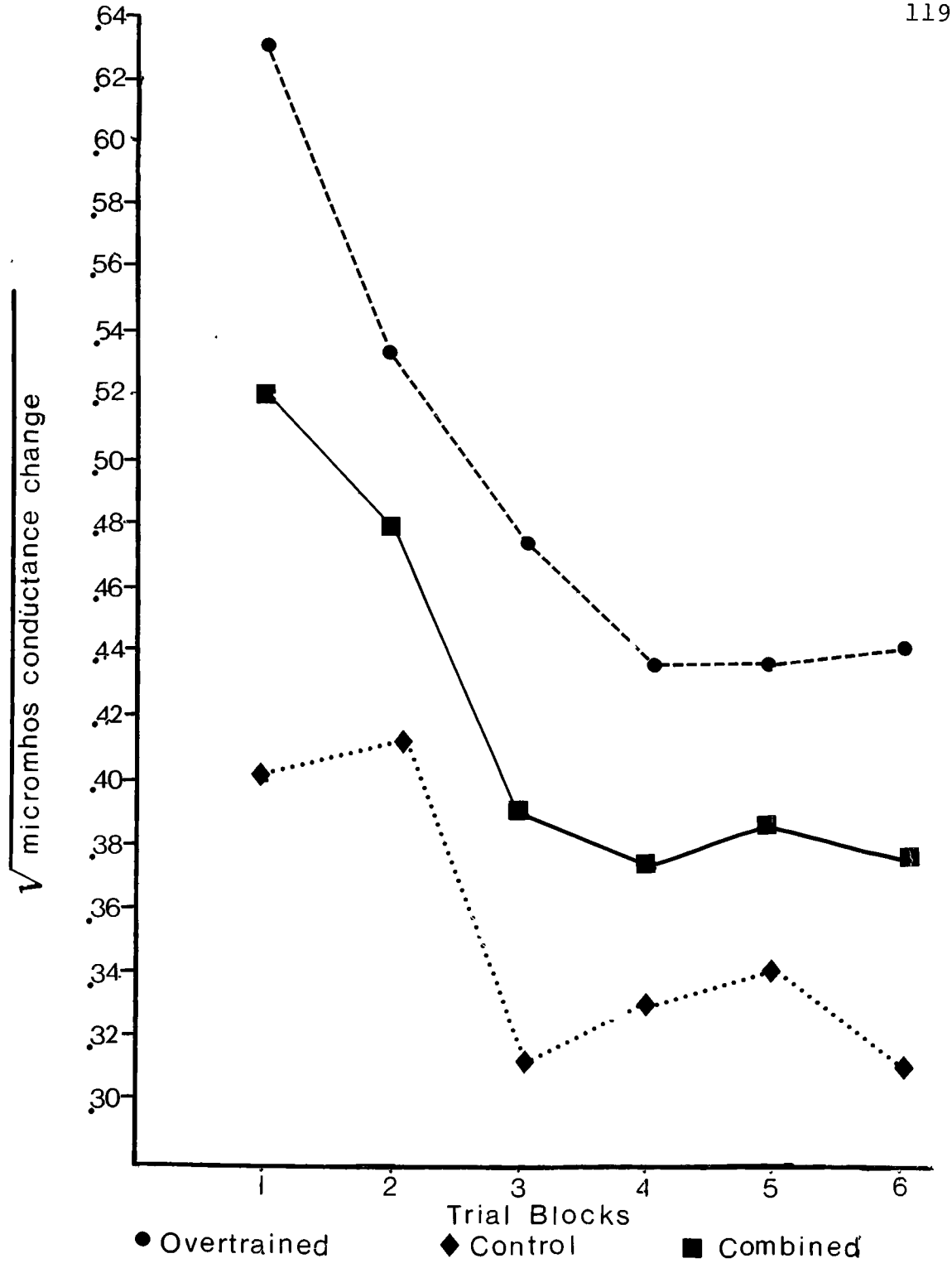

Figure 13. Trial Block Means for the First Response Recorded from Subjects during Acquisition Training.

Table 17

F Ratios in the Treatment X Trial Block Analysis of Variance for Second Interval Responses During Compound Conditioning

Source	*df*	*MS*	*F*
CT	1	1.317	11.860***
R(CT)	22	(.111)[a]	
TB	5	.068	3.580***
CT X TB	5	.031	1.630
TB,R(CT)	110	(.019)	

***p <.01

[a]Error *MS* in parentheses

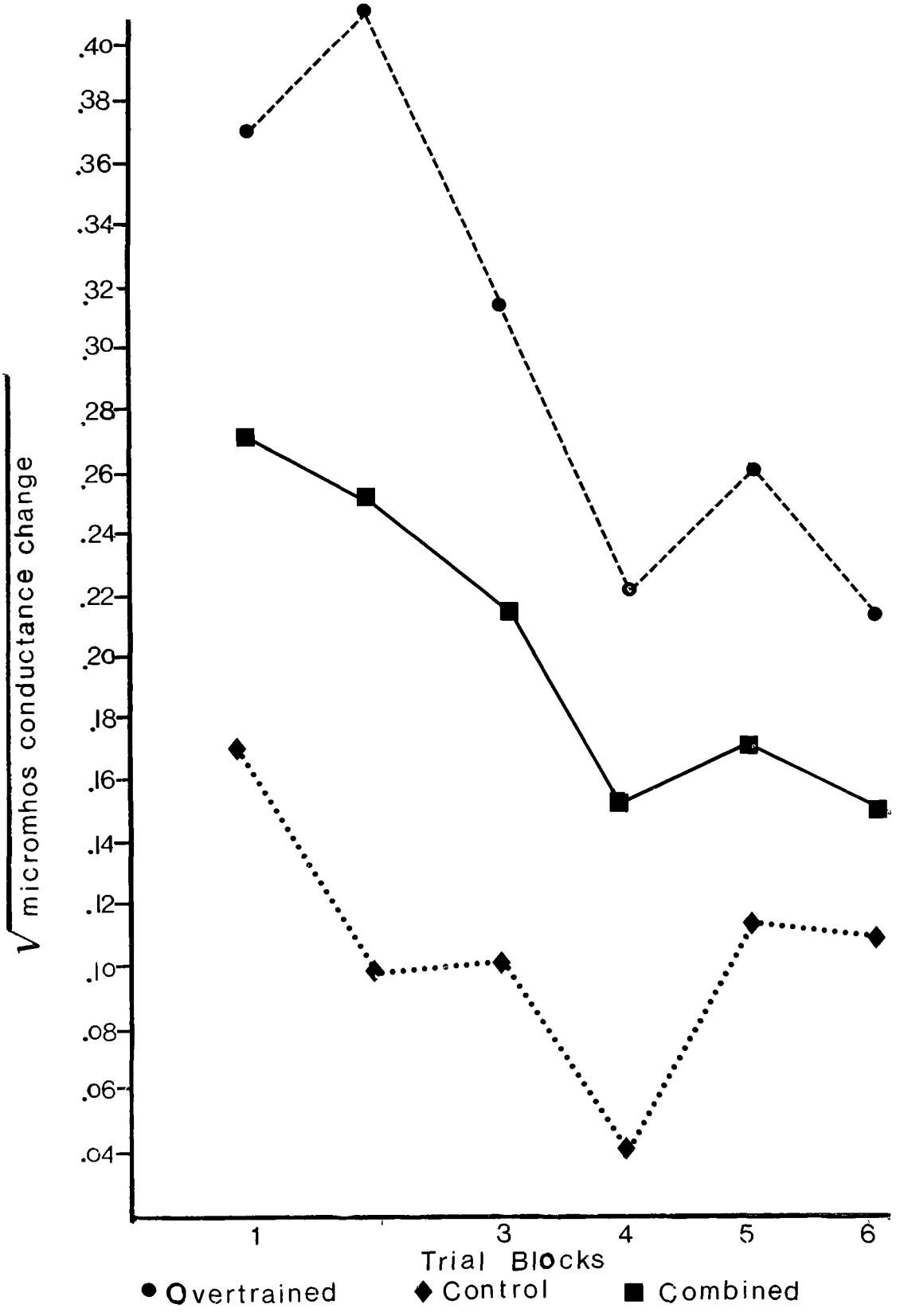

Figure 14. Trial Block Means for the Second Response Recorded from Subjects during Acquisition Training.

HSD=.226; Figure 13). The same post hoc tests for the second response (Tukey α.05, HSD=.147; Figure 14) showed that only the last trial block failed to differ between treatments.

An ancillary treatment X trial block analysis of variance which compared the first two trial blocks between the asymptotic and overtrained groups yielded only a trial block effect ($F(1,22)=26.400$, $p < .01$) for the first response (Table 18; Figure 15), and no differences related to second interval responding (Table 19; Figure 16). Therefore, the asymptotic and overtrained treatments were pooled ($n=24$) and compared with the control group ($n=12$) using the same analysis of variance as before, but with a provision for unequal treatment ns. This treatment X trial block (2 X 2) analysis of variance also uncovered a trial block effect ($F(1,34)=5.710$, $p < .05$) for the first response and no other (Table 20; Figure 17). The same analysis for the second response (Table 21; Figure 18) failed to furnish a trial block effect, but the difference between treatments was significant in favor of the pooled conditioning group ($F(1,34)=6.900$, $p < .05$).

The preceding analyses, with two levels for trial blocks, were consistent with the previous tests when treatment effects are considered, but the lack of a trial block effect for the second response suggested that these responses might exhibit ascending values for the first 10 conditioning trials. Such a finding would demonstrate second interval

Table 18

F Ratios in the Treatment X Trial Block Analysis of Variance for First Interval Responses During Compound Conditioning

Source	*df*	*MS*	*F*
CT	1	.079	.490
R(CT)	22	(.161)[a]	
TB	1	.132	26.400***
CT X TB	1	.001	.200
TB,R(CT)	22	(.005)[a]	

***p <.01

[a]Error *MS* in parentheses

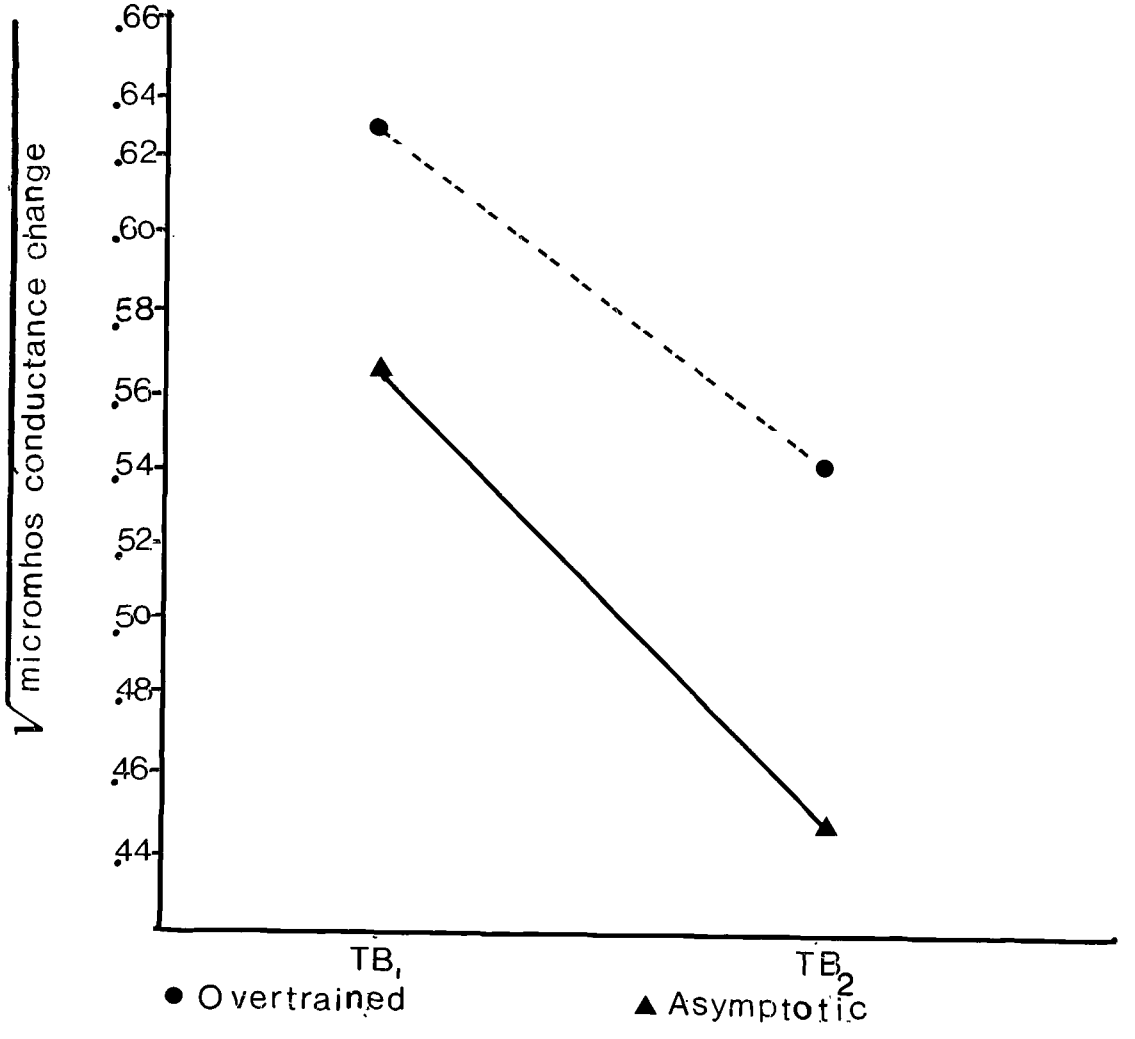

Figure 15. Trial Block Means between the Asymptotic and Overtrained Treatments for the First Response in Acquisition Training.

Table 19

F Ratios in the Treatment X Trial Block Analysis of Variance for Second Interval Responses During Compound Conditioning

Source	*df*	*MS*	*F*
CT	1	.148	1.780
R(CT)	22	(.083)[a]	
TB	1	.004	.360
CT X TB	1	.004	.360
TB,R(CT)	22	(.011)[a]	

[a]Error *MS* in parentheses

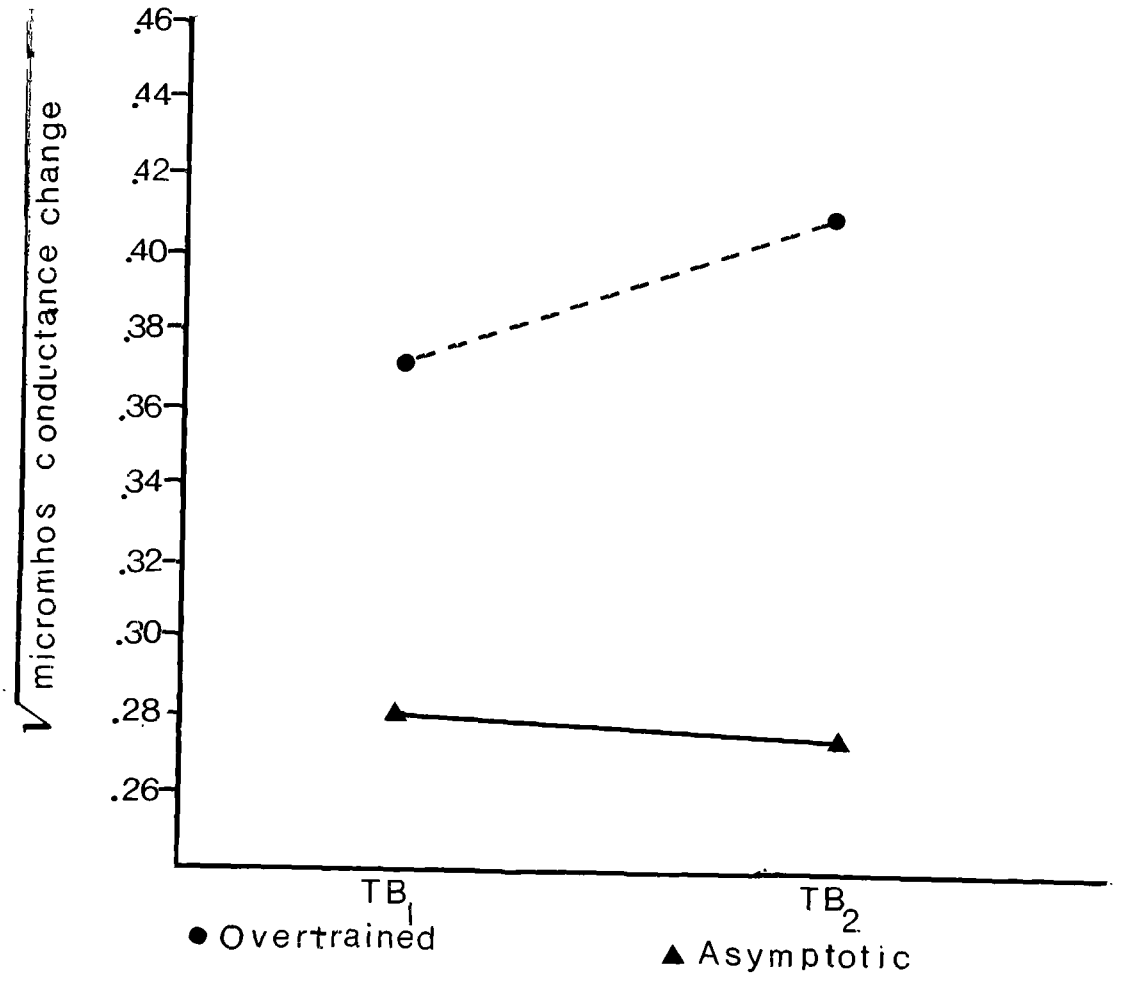

Figure 16. Trial Block Means between the Asymptotic and Overtrained Treatments for the Second Response in Acquisition Training.

Table 20

F Ratios in the Treatment X Trial Block Analysis of Variance for First Interval Responses During Compound Conditioning

Source	*df*	*MS*	*F*
CT	1	.316	1.860
R(CT)	34	(.170)[a]	
TR	1	.080	5.710**
CT X TR	1	.052	3.70
TR,R(CT)	34	(.014)[a]	

**p <.05

[a]Error *MS* in parentheses

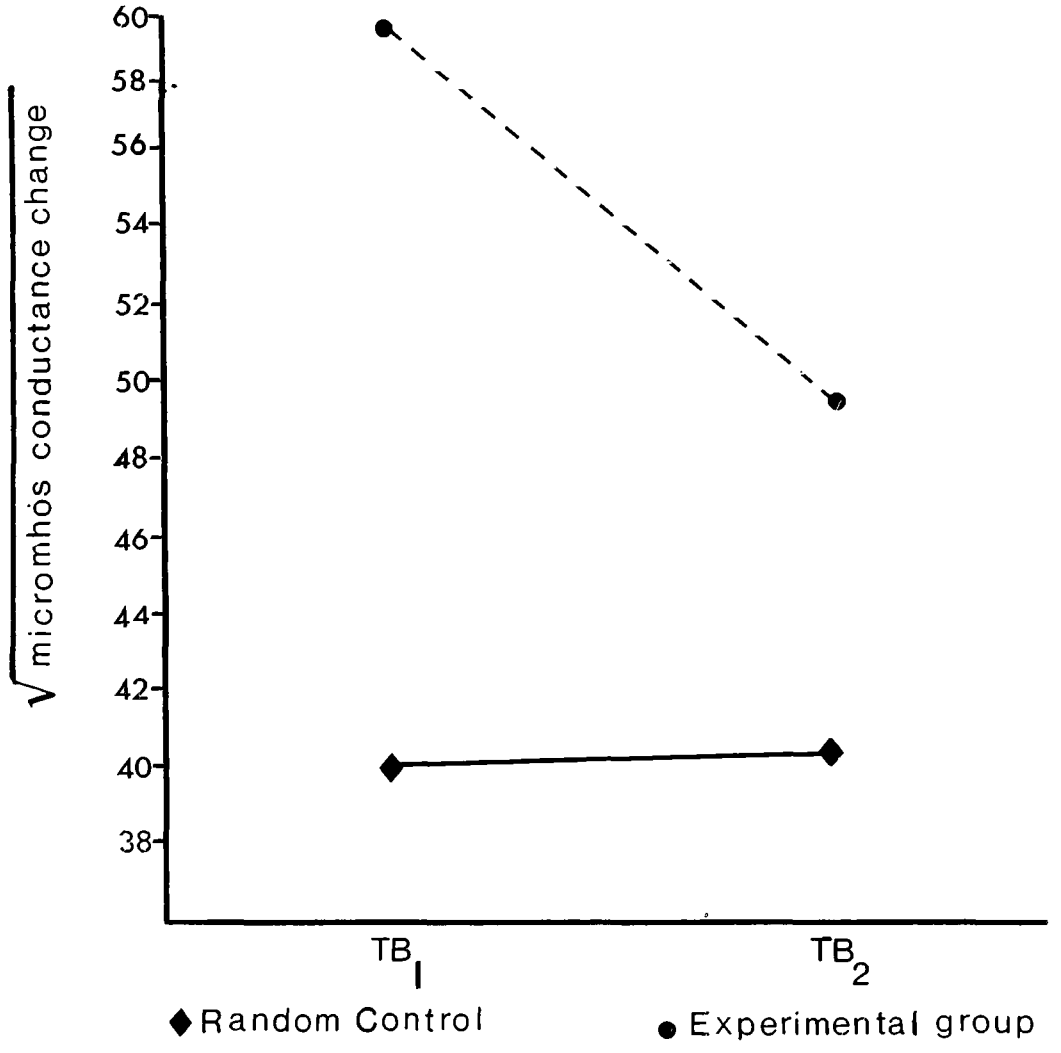

Figure 17. Trial Block Means between the Experimental and Control Treatments for the First Response in Acquisition Training.

Table 21

F Ratios in the Treatment X Trial Block Analysis of Variance for Second Interval Responses During Compound Conditioning

Source	*df*	*MS*	*F*
CT	1	.662	6.900**
R(CT)	34	(.096)[a]	
TR	1	.001	
CT X TR	1	.027	.070
TR,R(CT)	34	(.015)[a]	1.800

**p <.05

[a]Error *MS* in parentheses

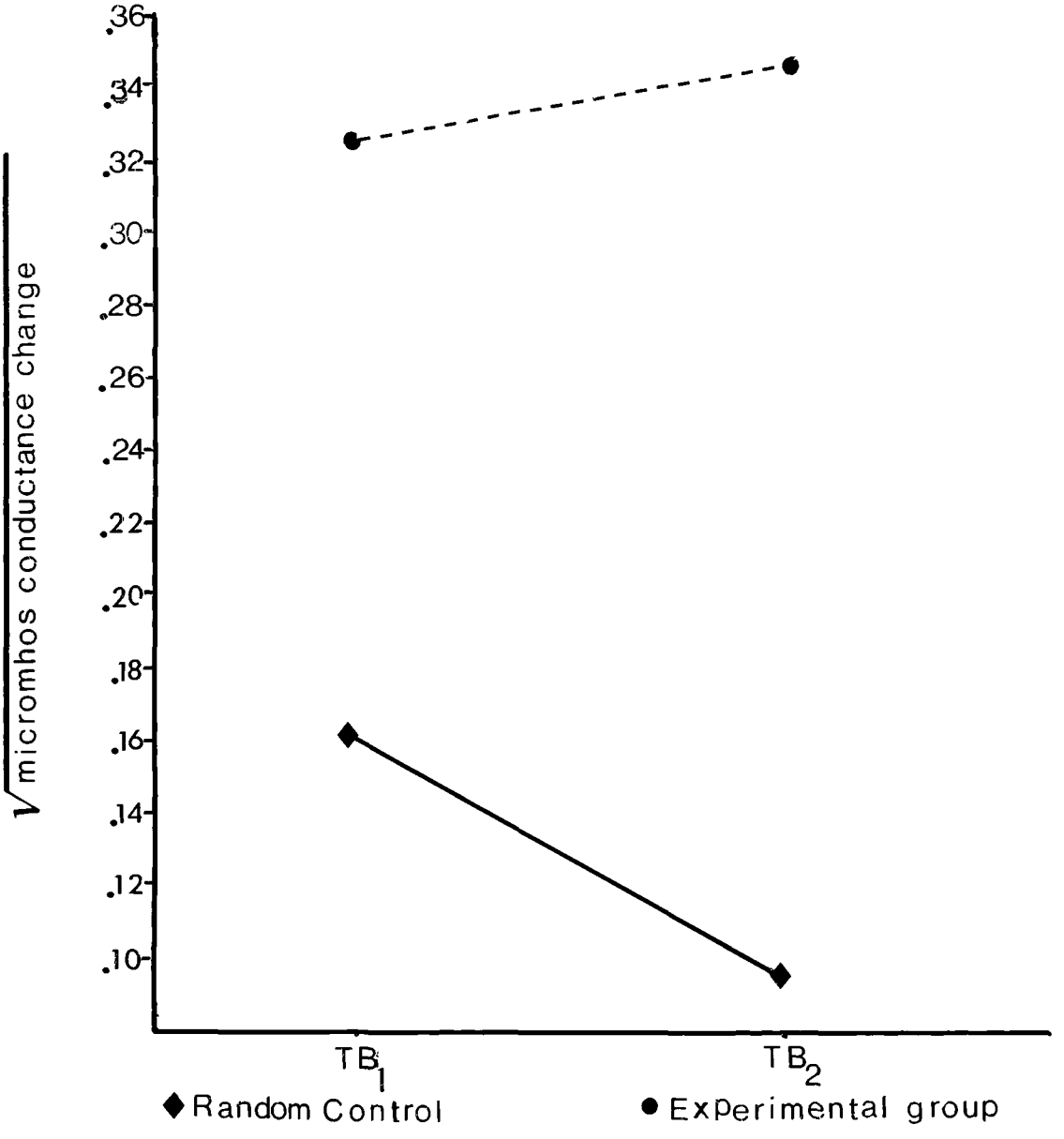

Figure 18. Trial Block Means between the Experimental and Control Treatments for the Second Response in Acquisition Training.

conditioning and provide an opportunity to establish the point of asymptotic performance for the second response. Although it would not be necessary for this experiment to demonstrate that the "asymptotic treatment" was actually trained exactly to asymptote, it would be helpful to know how soon the tenth conditioning trial occurred after the onset of asymptotic performance, since it is the extent of compound overtraining that was postulated to affect the degree of configurational learning (component CR diminution). Therefore, the first and second responses across the first 10 conditioning trials were compared separately between the asymptotic and overtrained treatments by two treatment X trial analyses of variance (2 X 10).

First responses for individual and combined treatments are plotted in Figure 19 for conditioning trials 1 through 10. Included in this figure is the graph of first responses for the last 10 conditioning trials of the overtrained treatment. A treatment X trial analysis of variance (Table 22) for the first responses occurring within the first 10 conditioning trials did produce a significant trial effect ($F(9,198)=4.2944$, $p < .01$), but no other. Pairwise comparisons of trial means for combined treatments (Tukey α .05, HSD=.138) did reveal that only the third trial differed from the first, and by the eighth trial, response levels were well below the first. Together with the foregoing trial block

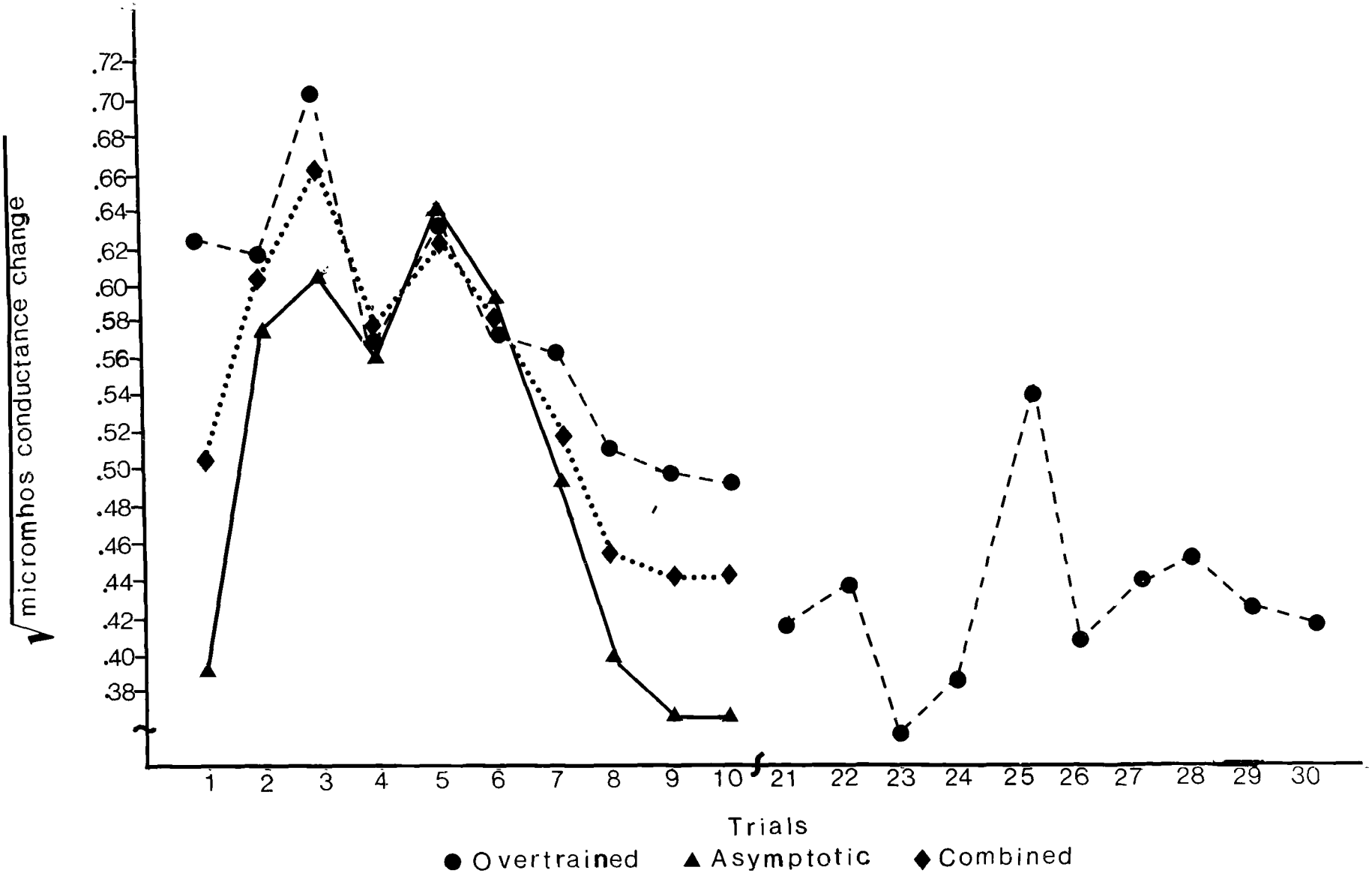

Figure 19. Trial Means for the First Response Recorded from Subjects in the Asymptotic and Overtrained Treatments during the First and Last Ten Trials of Acquisition Training.

Table 22

Significant F Ratio in the Treatment X Trial Analysis of Variance for First Interval Responses in Acquisition Training

Source	df	MS	F
TR	9	.153	4.2944***
TR,R(CT)[a]	198	(.035)	

[a]Error term in parentheses
***p <.01

results, these findings did expose sensitization effects and adaptation influences thereafter.

Second responses for individual and combined treatments are plotted in Figure 20 for conditioning trials 1 through 10, in addition to a graph of second responses for the last 10 conditioning trials of the overtrained treatment. The treatment X trial analysis of variance (Table 23) for these responses yielded a significant trial effect ($F(9,198) = 6.0198$, $p < .01$) in the absence of others. Tukey's pairwise contrasts (α .05, HSD=.196) for trial means showed that trials 2 through 10 for combined treatments did differ from the first conditioning exposure, with the exception of the eighth trial, but all trials from the second to the tenth failed to vary. When these results are taken with the significant treatment effects for trial blocks, it may be concluded that second interval conditioning did occur, and that the onset of asymptotic performance for the second response occurs by the second trial (one trial learning). The decrease in second interval responding in overtraining, given the significant difference between the conditioning and control treatments, suggests that either habituation or the refractoriness of peripheral GSR mechanisms might be responsible for this eventual decrease in second interval responding.

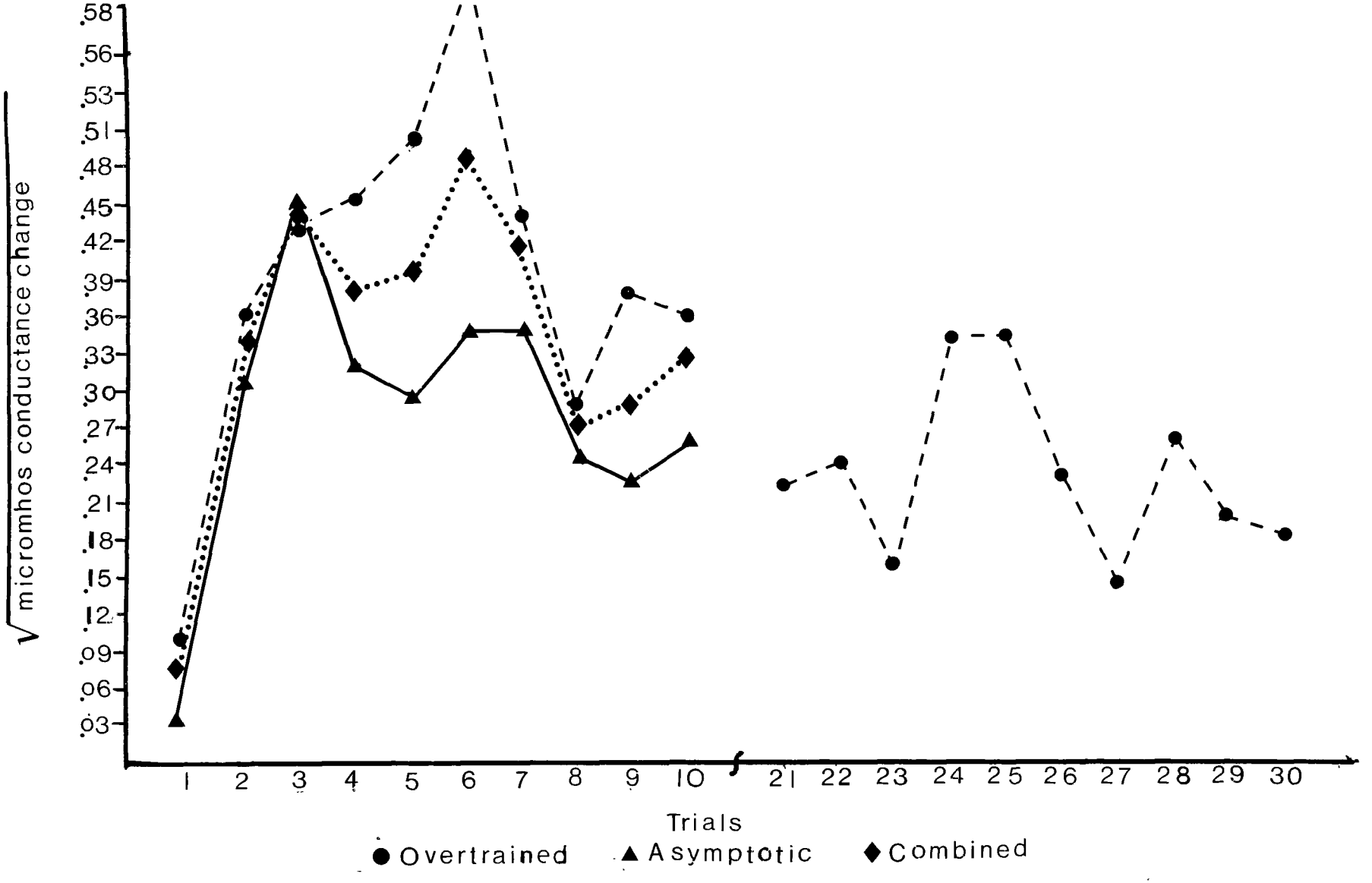

Figure 20. Trial Means for the Second Response Recorded from Subjects in the Asymptotic and Overtrained Treatments during the First and Last Ten Trials of Acquisition Training.

Table 23

Significant F Ratio in the Treatment X Trial Analysis of Variance for Second Interval Responses in Acquisition Training

Source	df	MS	F
T	9	.290	6.0198***
T,R(CT)	198	(.048)[a]	

[a]Error term in parentheses
***p <.01

DISCUSSION

In the past, perceptual integration learning had been inferred from the increased differentiation that occurred between the test responses of the compound CS and component stimuli with greater amounts of prior compound conditioning (Wickens et al., 1970; Razran, 1971). However, when it was demonstrated in a between-subject experiment (Baker, 1969) that the component responses also increased with compound training, component CR diminution across independent comparisons became the sole criterion from which configuring could be inferred. Unfortunately, the only compound conditioning experiment which had demonstrated component CR diminution according to this standard (Booth & Hammond, 1971), contained a methodological shortcoming. In that work, the component CR diminution obtained may have been determined by the disruptive affects of orienting reflexes evoked by the component test stimuli in the overtrained treatment, which were assumed to have an enhanced novelty value relative to those in the asymptotic group. Therefore, the present experiment used long delay electrodermal conditioning which could provide an opportunity to observe any systematic relationship between orienting reflexes and anticipatory or conditioned responses evoked by the component stimuli in testing. With this technique, five criteria were stated through which it could be shown that the stimulus elements of a compound CS

do condition initially, but during overtraining lose their conditioned properties (component CR diminution), while patterned aspects of the compound CS acquire conditioned strength. To this end it was required, in part, that: (1) the compound CRs of the overtrained group be significantly greater than those of the truly random control comparison to demonstrate successful conditioning either to the stimulus elements of the compound, or to patterned aspects of the CS. For the same responses in the asymptotic treatment, it was necessary for them not to be greater than the overtrained level of responding (2) to ensure that any component CR diminution in the overtrained group could not be linked to massed training effects, apart from compound overtraining. Further, to show that the stimulus elements of the compound CS initially acquire conditioned properties which diminish completely with overtraining (3) the component responses of the asymptotic treatment had to be significantly greater than those in the overtrained and control treatments. In addition to the last requirement, another (4) demanded no differences between the component responses of the overtrained and control groups in order to conclude that patterned properties eventually became the only active aspect of the compound CS. Finally, (5) the lack of a negative correlation between the first component responses and the diminished later reactions, associated with the same component presentations, had to be shown to rule out the disruptive influences of orienting reflexes that could be responsible for the component CR diminution obtained in configural conditioning.

Considering the foregoing criteria for the present experiment, the lack of conditioning for the first response would assure that the component first responses were, in fact, unconditioned orienting reactions, unconfounded by conditioned properties; and that the successful compound conditioning for either the second or third interval responses would permit the possibility of fulfilling the requirements for demonstrating configurational learning.

First interval Response

In adaptation training and configural testing, no first response differences between the experimental and control treatments were noted for any single stimulus condition. The ineffectiveness of the conditioning operations on the first response was highlighted further by the unchanged stimulus condition relationships within and between the treatment groups, from adaptation training to configurational testing. However, in adaptation training, the second trial block yielded inferior magnitudes relative to the first, but during configurational testing, both trial blocks were equivalent. In addition to these results, it was found that the first responses of the control compound CS in the last half of adaptation training were significantly less than those occurring in the first half of configurational testing, indicating a sensitization effect. This finding was corroborated in the CT X EP X SC split plot analysis which revealed that the overall level of first interval responding in configurational testing was superior to that in adaptation training.[1] Given the lack of conditioning for the first

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1 CT X EP X SC, see Appendix 2.

response and stimulus adaptation prior to conditioning with sensitization thereafter, it can be concluded that the first response, at least in the present experiment, had the characteristics of the orienting reflex.

Taken as a whole, this pattern of results went well with the stimulus generalization results of Epstein et al. (1971) and Ohman (1971). In those works, first interval test reactions for the CS failed to depart from appropriate control standards, and relationships between stimulus conditions within groups remained constant across the adaptation and test phases of experimentation (Epstein et al., 1971), or between experimental and control treatments in stimulus generalization testing (Ohman, 1971).

In compound conditioning, the first reponse acquisition data were found to be consistent with the configurational test results. A treatment X trial block analysis failed to uncover a difference between the overtrained and control treatments, while a trial block effect showed that there was a significant reduction in first interval responding, from the first trial block to the sixth. Ancillary treatment X trial block analyses, involving the first two trial blocks, indicated that the asymptotic and overtrained treatments were equivalent, and when these groups were pooled and compared with the control treatment, only the previous trial block effect emerged. Similarly, a treatment X trial analysis indicated that the first responses for the overtrained subjects did not differ

from those of the asymptotic treatment over the first 10 conditioning trials. However, post hoc tests to investigate a trial effect that was forthcoming showed that only the third conditioning trial was significantly greater than the first, and thereafter, first interval responding decreased below the performance level of the first trial.

Given the lack of first interval conditioning with adaptation effects found during compound conditioning, and the sensitization result present in the analysis of discrete conditioning trials, it would seem appropriate to put forth an orienting reflex interpretation for first interval responding during compound conditioning. Further, the equivalence of compound first responses between the initial conditioning trials and those throughout the configural test phase would suggest that the 1-hour rest period following conditioning serves to reinstate ORs and prolong sensitization effects to a level greater than that observed during the conditioning period.

Out of all the studies which employed simple component conditioning, the present results for the first response only disagreed with those of Prokasy and Ebel (1967). In their experiment, first interval conditioning was the result of the more rapid decrease in the performance of their explicitly unpaired control treatments beneath the waning responses of the conditioning groups. In this instance, the

successful conditioning result may have come about through inhibitory properites that can be acquired by stimuli presented in an explicitly unpaired control treatment (Rescorla, 1967). The truly random control used in the present experiment was initially designed to prevent the acquisition of inhibitory properties by control stimuli. Even so, although no simple conditioning experiments employing the electrodermal response have compared the explicitly unpaired control with the truly random treatment, the majority of discrimination experiments which had (Schiffman & Furedy, 1972; Furedy, 1971), demonstrated no differences.

The reliable discrimination conditioning of the first response in Gale and Ax (1968) and Gale and Stern (1967) might be explained by negative contrast effects of the type associated with CS+ and CS- trials in the behavioral contrast experiment (Reynolds, 1961), or Pavlovian induction research (Pavlov, 1927). Accordingly, during successful long delay discrimination conditioning, the CS- onset responses would be expected to diminish to a greater extent than those of an explicitly unpaired CS- treatment in a simple conditioning experiment. This would yield a greater separation between CS+ and CS- onset responses in the discrimination experiment, as compared to an experiment with independent comparisons, and give the appearance of first response conditioning for the discrimination experiment. Nonetheless, the weak evidence for discrimination conditioning with delayed responses renders

the disparate results between single and multiple cue designs problematic.

Second Interval Response

In adaptation training, no differences between the conditioning treatments were observed for all stimulus conditions, nor was stimulus quality a factor in the determination of the second response as it was for the first response. In the test phase, compound second responses for both experimental groups were comparable and significantly larger than those of the random control, but the component responses to the light in the overtrained group were indistinguishable from the control level, well below the responses to the light in the asymptotic treatment. The second responses to the tone did not differ between treatments. When stimulus conditions in testing were compared within either the asymptotic or overtrained treatments, the compound second responses were always greater than the component responses, which failed to differ statistically. Stimulus conditions did not differ within the control treatment.

All the above changes in stimulus condition relationships, within and between treatments from adaptation training to configural testing, became evident once again when the simple-simple effects for the CT X EP X SC interaction were examined. The infrequent and low amplitude second responses, together with the nonexistent stimulus condition effect in

adaptation training, offered little encouragement for an orienting reflex interpretation of the second interval response; while evidence for conditioning in testing from the within and between treatment comparisons could not be disputed.

Second interval results for both conditioning treatments paralleled the stimulus generalization findings of Epstein et al. (1971). Their second responses for the CS were significantly larger than those of an independent control comparison and the within treatment generalization responses, which did not differ. Ohman (1971), who used novel generalization stimuli in testing (no adaptation presentations prior to generalization testing), did achieve a second response conditioning effect, but no differences were seen between the stimulus conditions within his experimental and control groups. This failure may have been the result of stimulus novelty in testing.

As with the first response, the compound conditioning data for second interval responding agreed with the configural test results. A treatment X trial block analysis of second interval responding during conditioning disclosed that the responses of the overtrained treatment were significantly greater than those for the control subjects. In addition to the treatment effect, there was a significant reduction in trial block responding for the combined treatments, from the beginning of conditioning to the end. Post hoc

tests between treatments for trial block means revealed significant differences for each block of trials with the exception of the last, thereby divulging a sustained conditioning effect in spite of the waning trial block values which resembled first interval responding. Ancillary treatment X trial block analyses, with only the first two trial blocks, showed that second interval responding between the asymptotic and overtrained treatments did not differ, and when these groups were pooled and compared to the control comparison, only a treatment effect became evident in favor of the pooled group. The lack of a trial block effect warranted a treatment X trial analysis involving the first 10 conditioning trials for the asymptotic and overtrained subjects in order to detect the possibility of ascending trial values. This result would constitute evidence for second interval conditioning when taken with the previous trial block analyses. Such an analysis indicated no differences between treatments, but the trial effect was significant. Post hoc comparison of trial means for combined treatments disclosed that trials 2 through 10 did surpass the first conditioning exposure, with the exception of the eighth trial, but all trials from the second to the tenth did not differ. Taken together, all previous results would signify that the second response was successfully conditioned, and that associative learning occurred on the very first conditioning trial. The decrement of second interval responding

during overtraining suggests that either habituation to the CS, or refractoriness of the peripheral GSR mechanism might have been responsible for this outcome. However, compound CRs throughout configural testing were of the same magnitude as those occurring during the first 10 trials of conditioning, while control responses did not change substantially across the different phases of experimentation. Thus, for the second response, the 1-hour rest period reinstated the conditioned responses through the dissipation of temporary influences, and the sensitization effects observed for the first response were not reflected by the second response.

In addition to the stimulus generalization studies, all the evidence from simple component conditioning experiments agreed with the second interval conditioning result obtained in the present effort. Similarly, all conventional discrimination experiments (Gale et al., 1967, 1968) which yielded first response conditioning, also revealed second interval conditioning with the exception of Schiffman and Furedy (1972). Their results were difficult to understand because it was felt that discrimination conditioning was the most likely technique to reveal second interval learning in view of possible behavioral contrast effects (Reynolds, 1961; Pavlov, 1927). However, given this negative finding, it should be mentioned that in two unconventional discrimination conditioning experiments (with a sequence of stimulus presentations on a single trial), second interval conditioning

was only demonstrated either in the first of three test trials (Dawson & Biferno, 1973), or only with an ancillary analysis (Dawson, 1970). Withal, second interval conditioning seemed to be more explicit in simple conditioning than with discrimination conditioning procedures. In any case, the comparable degree of second interval compound conditioning for the asymptotic and overtrained treatments in the present work fulfilled an important condition for observing component CR diminution and relating it to either configurational learning, or the influence of orienting reflexes.

Third Interval Response

The third interval GSRs in adaptation training portrayed an absence of variations related to stimulus quality and conditioning group effects, but the overall response level did decrease from the first trial block to the second, indicating adaptation. This pattern of third interval responding continued into the test phase as the same trial block effect was forthcoming, but with a difference. Now, the overall response levels for the asymptotic and overtrained treatments in trial block 1 were greater than the combined control performance, but in the second trial block, no such differences were maintained. A between group comparison of third responses, for the compound stimuli with trial blocks combined, showed that both conditioning treatments were similar and excelled the control responses by significant margins. Within

conditioning treatments, the compound responses for the third interval surpassed those of their respective sets of components, while the GSRs of the control compound did not. Component third responses within and between conditioning treatments did not exceed the levels of chance variation, thereby indicating no component CR diminution. However, the compound conditioning result for the third response in testing was weakened by the nonsignificant triple interaction in the CT X EP X SC analysis of variance (see Appendix 2), which showed that the differences between treatments for the compound stimulus in testing did not exceed the same differences in adaptation training.

The negated conditioning result for the third response in the CT X TB X SC analysis, together with the trial block effects in adaptation training and configural testing, suggested that the third response could have initially reflected stimulus offset responses in adaptation training and the disparity reaction concept of Grings (1960) during configurational testing. In these phases of experimentation, it was conceivable that the overall reduction of third responses evident for all treatment groups in phase 1 was the result of adaptation to the offset of all stimuli, but in phase 3, the extinction result associated with the asymptotic and overtrained treatment may have been linked to the subjects' disconfirmation of their (unfulfilled) expectations of shock associated with the compound CS. Ohman's (1971)

claim, that the third responses were underlied by orienting reflexes early in conditioning and conditioned responses later, was not supported by these data as the component responses in testing did not approach the compound reaction level in the asymptotic treatment, nor did the within treatment conditioning results for the asymptotic and overtrained subjects differ in any. However, because this experiment utilized adaptation training prior to conditioning, it was not a fair test of Ohman's view.

Configural Conditioning

The successful compound conditioning of the second response allowed the evaluation of configurational learning according to the previously stated criteria, and the absence of such conditioning for the first response simplified an OR interpretation of the first component responses.

Second interval compound conditioning (1) for the overtrained subjects and the absence of conditioned excitation to their component stimuli (4) indicated that eventually, patterned aspects of the compound stimulus became the CS, apart from the stimulus elements. The (marginally) superior second responses evoked by the light component in the asymptotic group, relative to the overtrained and control levels (3) was evidence that the light component acquired conditioned properties early in conditioning but became ineffective on succeeding compound conditioning trials. The second responses

for the tone component in the asymptotic treatment did not differ from the overtrained and control levels of responding. However, the point of onset for asymptotic performance was observed to occur by the second compound conditioning trial and, therefore, the asymptotic treatment was actually another overtrained group which could have yielded component diminution for the tone, before the tenth conditioning trial. Since the first responses(ORs) to the tone, during adaptation training, were significantly less than the equivalent responses to the light, it might be argued that the light stimulus was the stronger component. Given the correctness of this assumption, the present result agreed with those of the Russians', in that the response strength of the weaker tone component took less overtraining to drop out than the stronger light. An alternative explanation for the present component results could deny component conditioning to the tone and attribute response diminution for the light to massed training trials (apart from what they contained), and not compound overtraining (i.e., configuring). However, these objections to the configurational interpretation were not supported by the complete set of results. In testing, the second responses for the light and tone in either conditioning group differed significantly from the compound performance suggesting that no overshadowing had taken place, and that each component was an essential part in the fabric of the overtrained compound stimulus (or configure). Therefore, since extinction trials

with the component stimuli only affect the compound responses early in conditioning (Razran, 1971, p. 208), the involvement of the embedded tone component would most likely involve prior conditioning. Insofar as massed training can account for component CR diminution related to the light, perfect equality between the overtrained and asymptotic compound performances ruled out that influence (2). The demonstration of component CR diminution as a function of the degree of compound overtraining, and the absence of a negative correlation between the first and second component responses in both experimentl treatments, linked the diminution of component CRs to configuring and not to the disruptive influences of the orienting reflex (5).

With this profile of results, it can be concluded that configurational learning does occur with compound overtraining alone, and that, unlike Booth and Hammond's (1971) second experiment, it was further demonstrated that the diminished component responses of the overtrained treatment revealed no conditioned properties. It is important to reiterate here that Baker's (1969) study did not furnish a control condition for each of his independent treatment comparisons, which showed an increase in compound-component differentiation and component CR strength, with increasing amounts of compound training trials. With such control levels, it might very well have been found that in the early stages of compound training, component conditioning was demonstrable, but in

the later test periods no component conditioning might have resulted. In this instance, configuring would have been demonstrated and, therefore, in addition to the present findings, Baker's results need not be considered inconsistent with those of Booth and Hammond.

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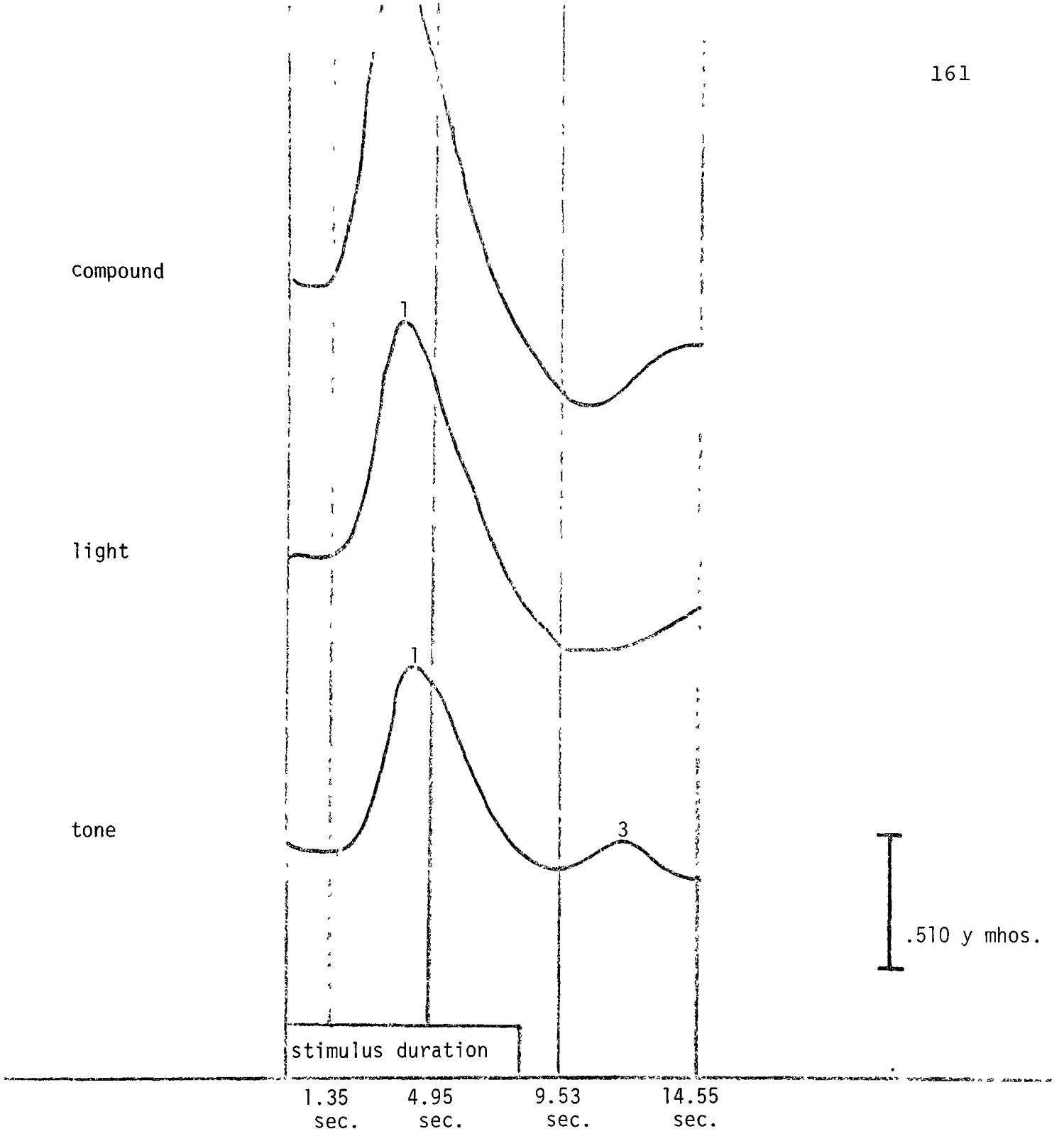
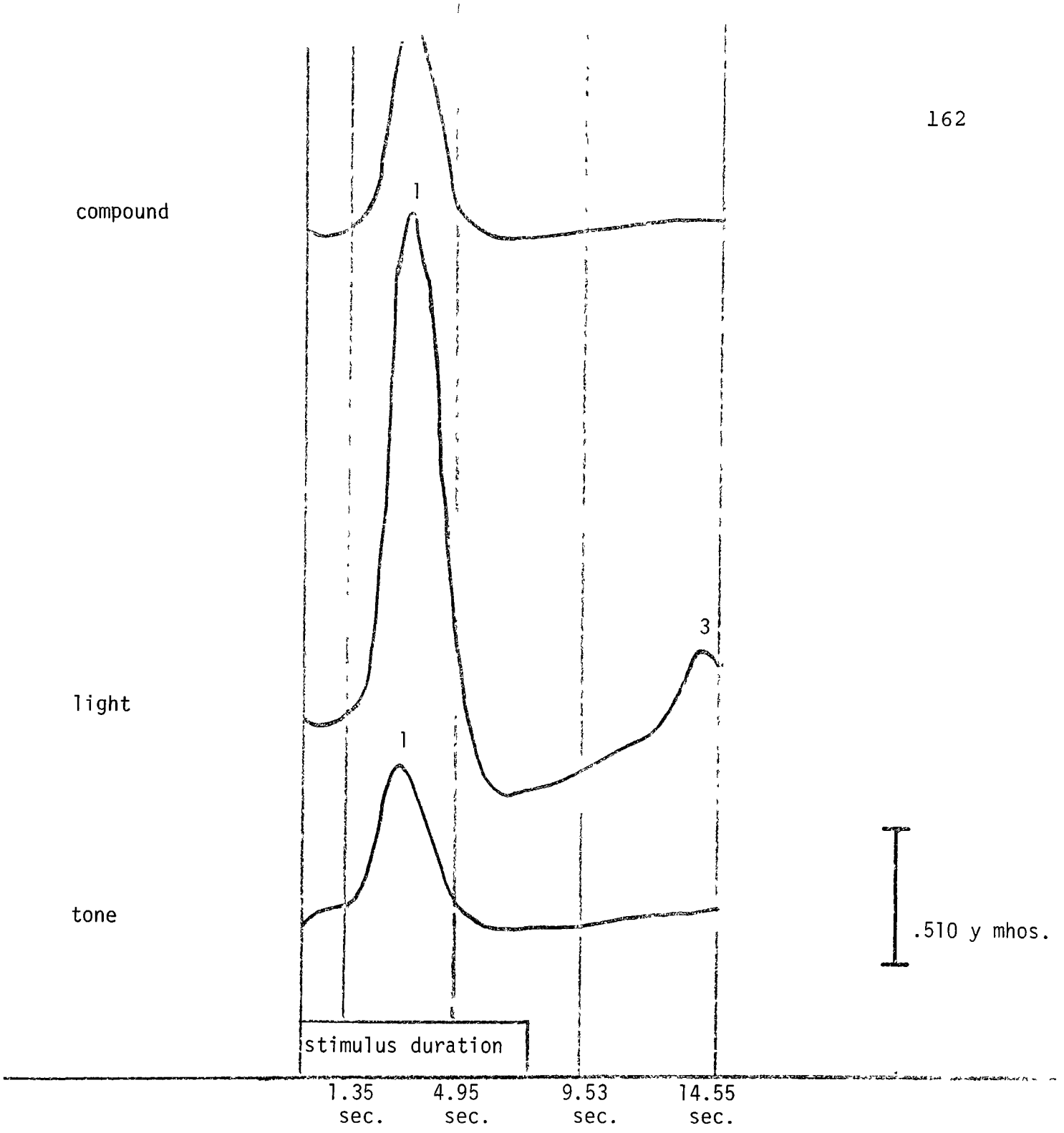
APPENDIX 1

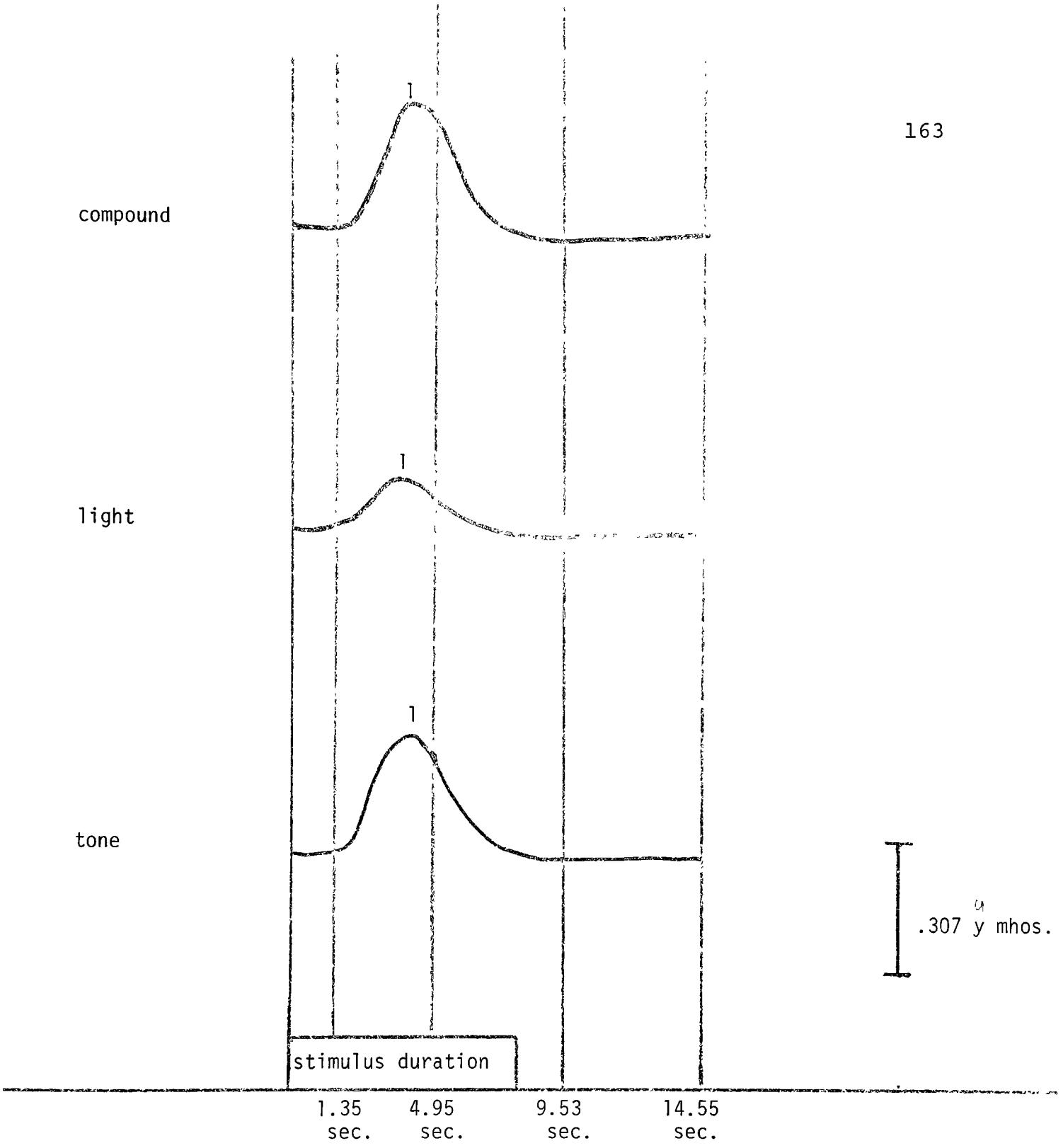
REPRESENTATIVE SAMPLES OF FIRST, SECOND, AND THIRD INTERVAL GSRs DURING THE THREE PHASES OF EXPERIMENTATION



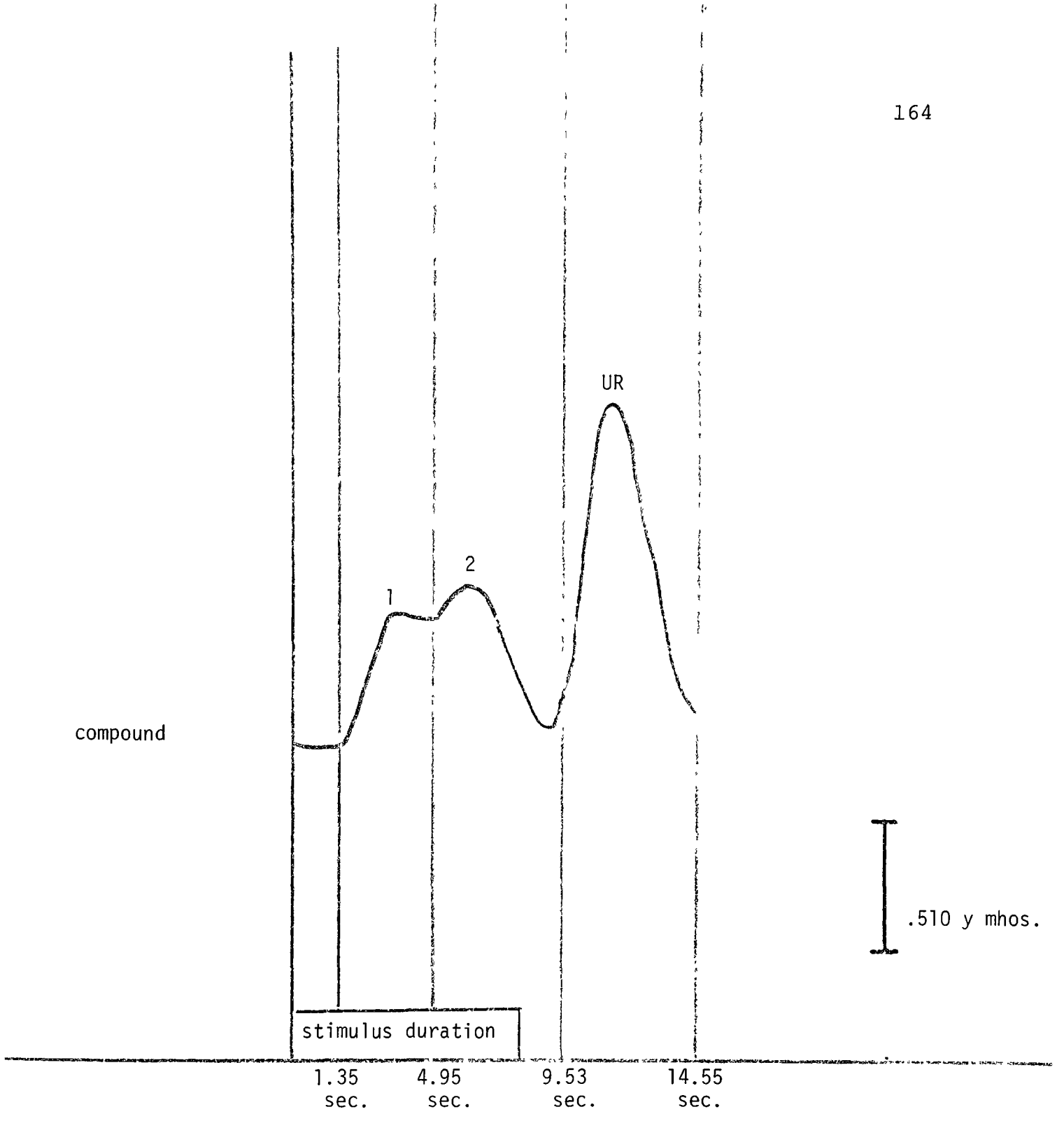
Example of first, second and third interval responding from subject no. 4, during adaptation training.



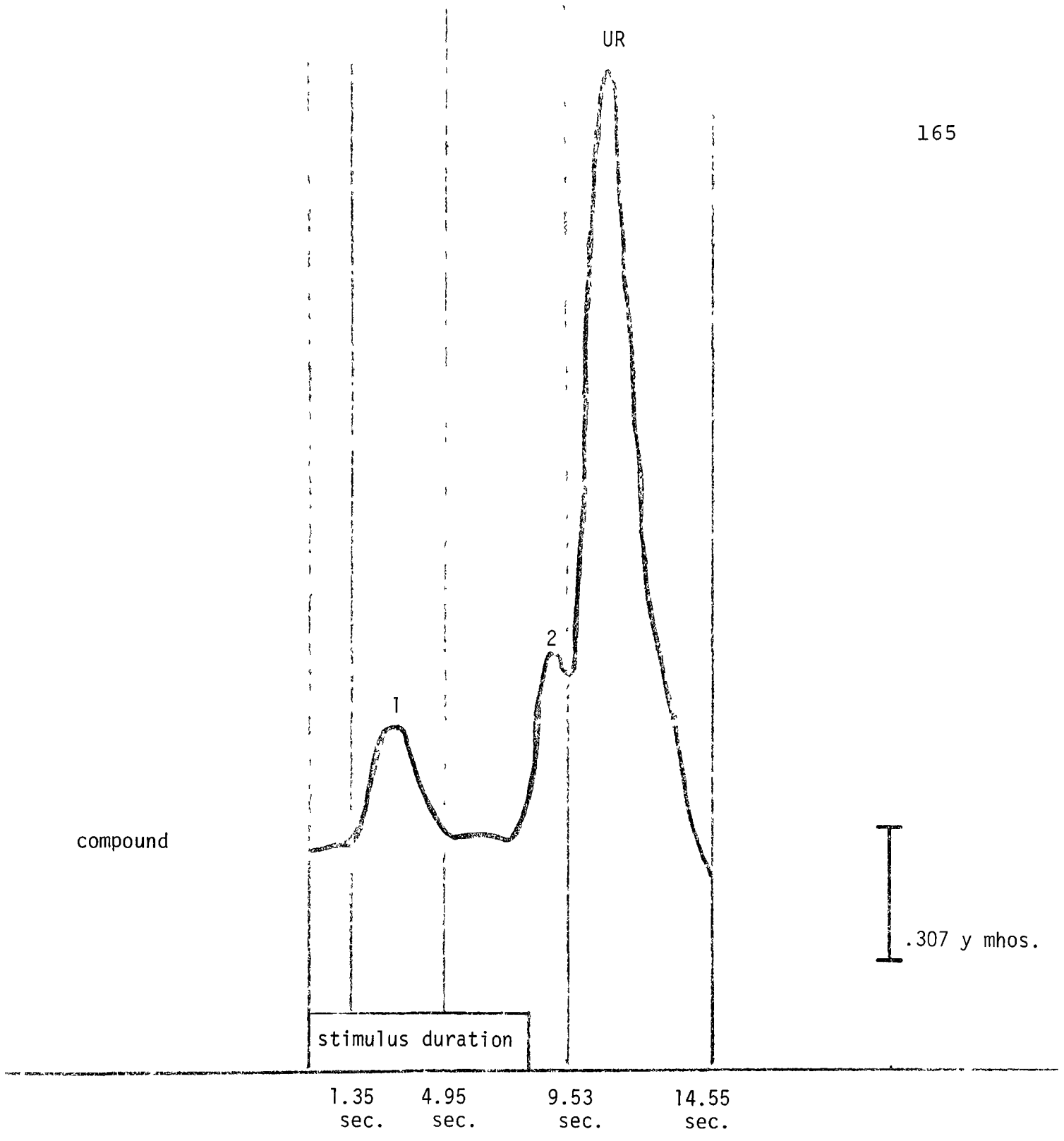
Example of first, second and third interval responding by subject no. 12, during adaptation training.



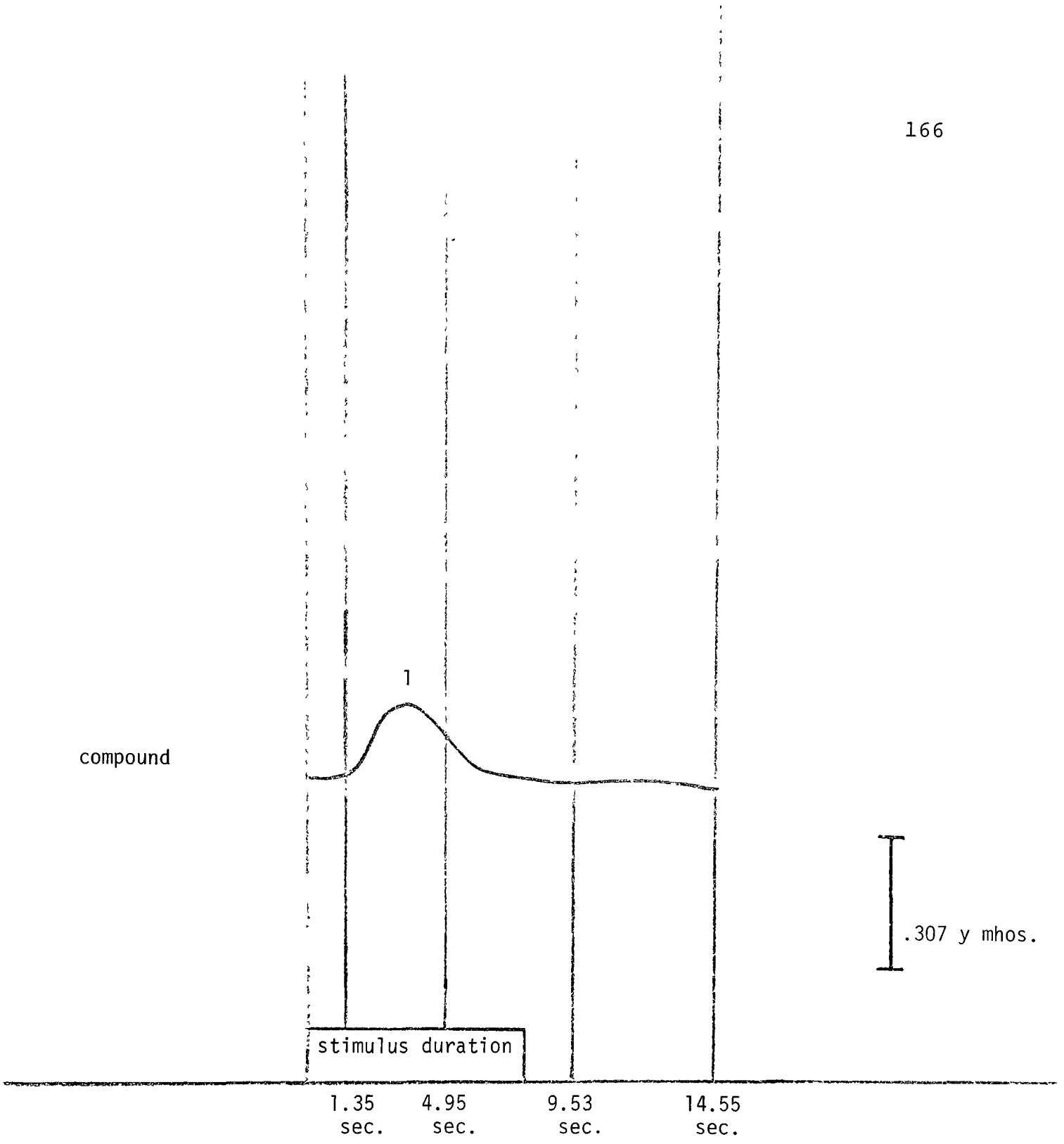
Example of first, second and third interval responding from subject no. 10, during adaptation training.



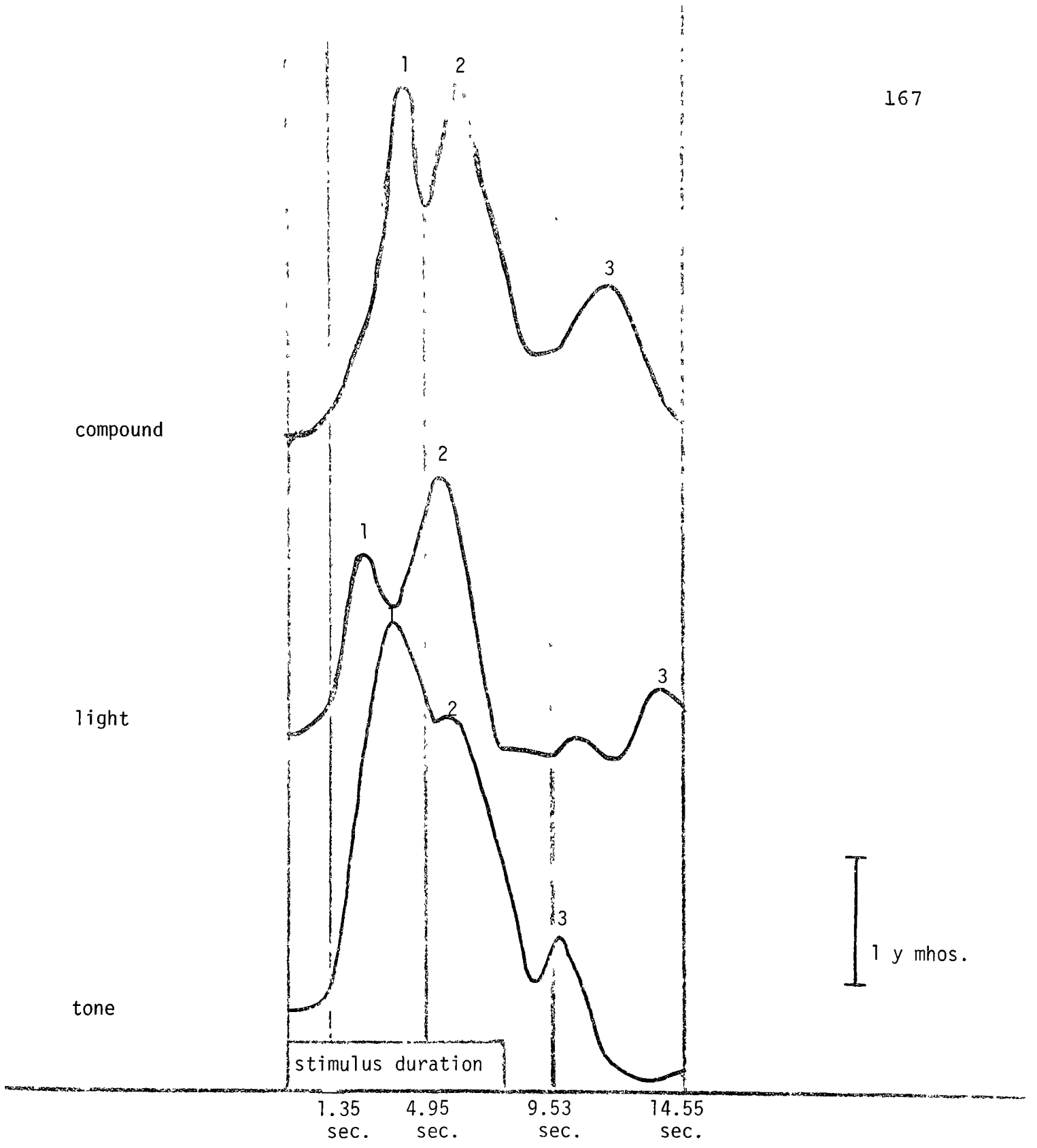
Example of first, second and third interval responding by an asymptotically trained subject (no. 4) during interpalated compound conditioning.



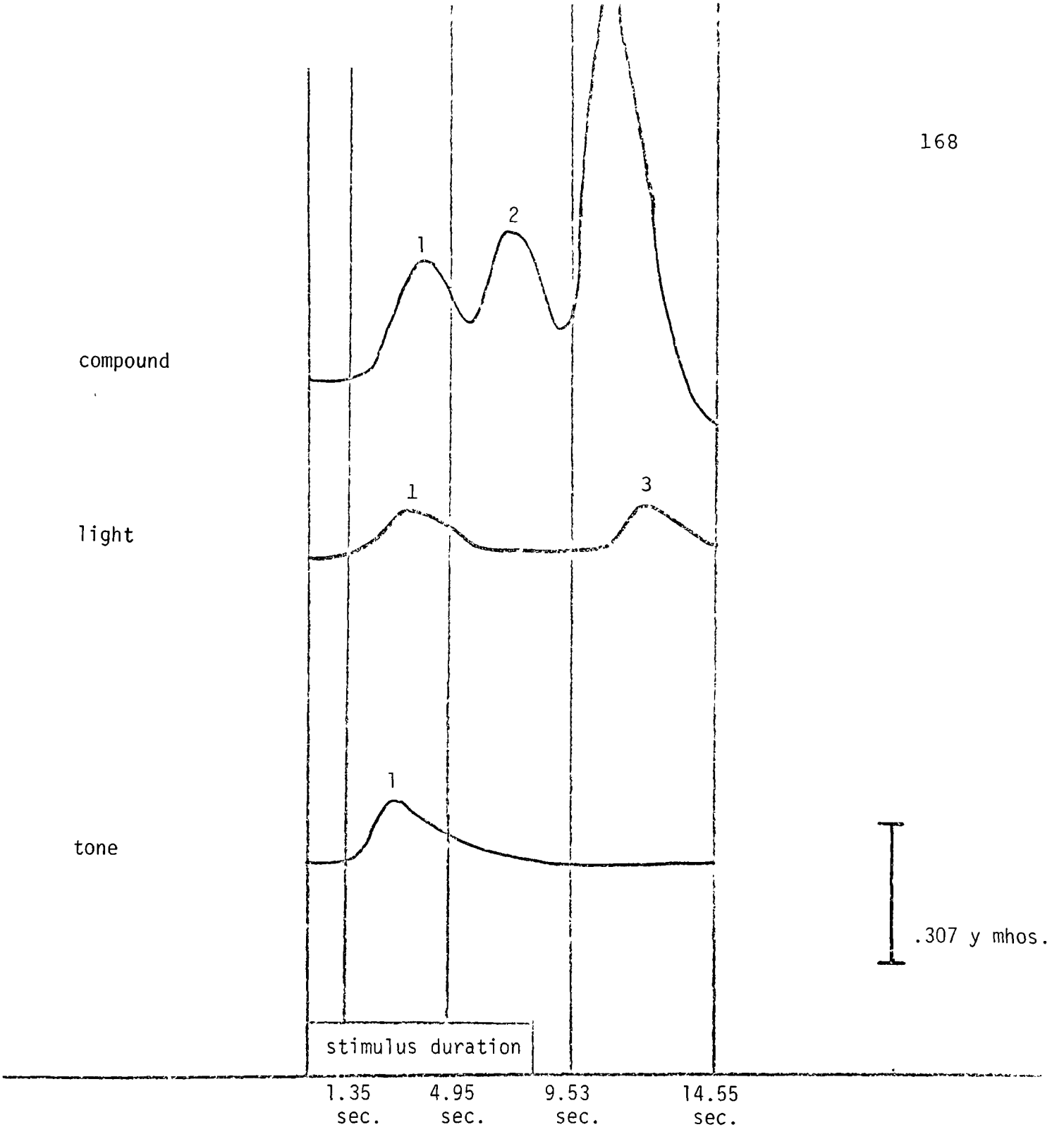
Example of first, second and third interval responding by an overtrained subject (no. 12) during interpolated compound conditioning.



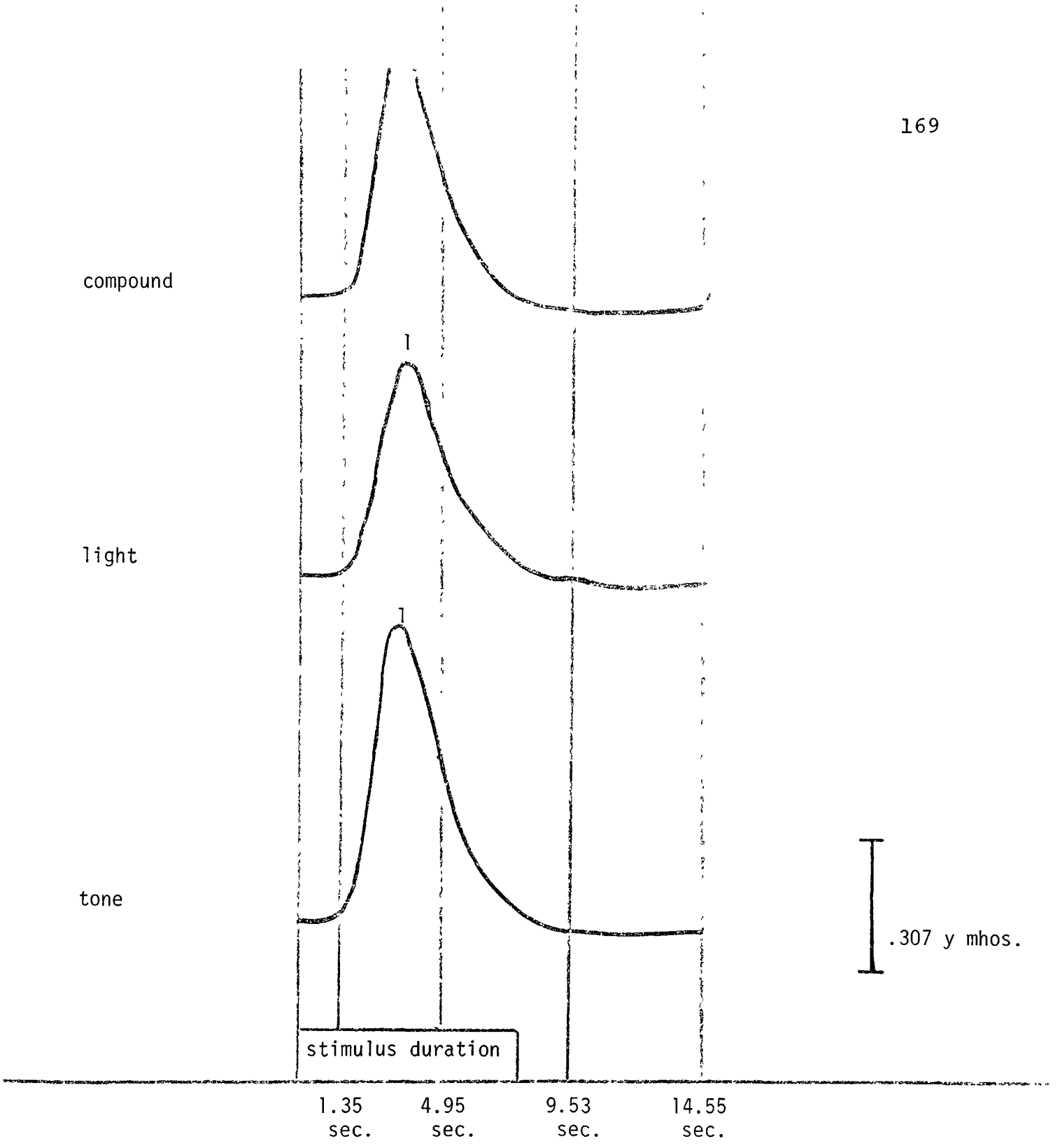
Example of first, second and third interval responding by subject no. 10 who had received the truly random control treatment during interpolated training.



Examples of first, second and third interval responding by an asymptotically trained subject (no. 4) during configurational testing.



Examples of first, second and third interval responding by an overtrained subject (no. 12) during configurational testing.



Example of first, second, and third interval responding by a truly random control subject (no. 10) during configurational testing.

APPENDIX 2

ANALYSIS OF VARIANCE TABLES

F Ratios for CT X TB X SC Analysis of Variance - First Interval GSR Magnitude in Adaptation

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	151.0087	50.04572	1	50.04572
2	CT	R(CT)	.1226	.08125639	2	.04062819
3	TB	TB,R(CT)	6.3798	.2873219	1	.2873219
4	SC	SC,R(CT)	19.4439	.3925516	2	.1962758
5	R(CT)			10.93651	33	.3314095
6	CT X TB	TB,R(CT)	1.3698	.1233812	2	.0616906
7	CT X SC	SC,R(CT)	1.0703	.04321766	4	.01080441
8	TB X SC	TB,SC,R(CT)	1.0729	.02544624	2	.01272312
9	TB X R(CT)			1.486185	33	.04503591
10	SC X R(CT)			.666235	66	.01009447
11	CT X TB X SC	TB,SC,R(CT)	.9018	.04277575	4	.01069394
12	TB X SC X R(CT)			.7826885	66	.01185891

First Interval GSR Mean Magnitudes for CT X TB X SC Analysis of Variance Cell Values in Adaptation

CT = Control

SC =	C	L	T
TB = 1	.60975	.57950	.47292
2	.47017	.43533	.37267

CT = Asymptotic

SC =	C	L	T
TB = 1	.57350	.54542	.49875
2	.53100	.44175	.40675

CT = Overtrained

SC =	C	L	T
TB = 1	.48917	.43392	.45750
2	.54450	.41558	.38617

F Ratios for CT X TB X SC Analysis of Variance -
Second Interval GSR Magnitude in Adaptation

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	41.6111	2.360816	1	2.360816
2	CT	R(CT)	2.0668	.2345167	2	.1172584
3	TB	TB,R(CT)	.8392	.01692463	1	.01692463
4	SC	SC,R(CT)	.7227	.01749799	2	.008748993
5	R(CT)			1.872261	33	.05673518
6	CT X TB	TB,R(CT)	1.9425	.07835364	2	.03917682
7	CT X SC	SC,R(CT)	.8503	.04117277	4	.01029319
8	TB X SC	TB,SC,R(CT)	.0631	.001630329	2	.0008151643
9	TB X R(CT)			.6655505	33	.02016819
10	SC X R(CT)			.7989790	66	.01210574
11	CT X TB X SC	TB,SC,R(CT)	1.3719	.07088923	4	.01772231
12	TB X SC X R(CT)			.8526068	66	.01291828

Second Interval GSR Mean Magnitudes for CT X TB X SC
Analysis of Variance Cell Values in Adaptation

CT = Control

	SC = C	L	T
TB = 1	.15742	.14258	.13000
2	.06942	.09975	.04625

CT = Asymptotic

	SC = C	L	T
TB = 1	.05300	.02283	.09575
2	.08000	.07283	.05217

CT = Overtrained

	SC = C	L	T
TB = 1	.17392	.13400	.11108
2	.16492	.09608	.17983

F Ratios for CT X TB X SC Analysis of Variance -
Third Interval GSR Magnitude in Adaptation

Source	Error Term	F	Sum of Squares	df	MS
1 Mean	R(CT)	55.5086	4.326069	1	4.326069
2 CT	R(CT)	.9497	.1480276	2	.07401377
3 TB	TB,R(CT)	4.6420	.07425719	1	.07425719
4 SC	SC,R(CT)	1.8597	.04046294	2	.02023147
5 R(CT)			2.571856	33	.07793498
6 CT X TB	TB,R(CT)	.7092	.02269077	2	.01134539
7 CT X SC	SC,R(CT)	.8377	.03645346	4	.009113364
8 TB X SC	TB,SC,R(CT)	1.1123	.02376929	2	.01188464
9 TB X R(CT)			.5278978	33	.01599690
10 SC X R(CT)			.7180116	66	.01087896
11 CT X TB X SC	TB,SC,R(CT)	.1238	.005292892	4	.001323223
12 TB X SC X R(CT)			.7052100	66	.01068500

Third Interval GSR Mean Magnitudes for CT X TB X SC
Analysis of Variance Cell Values in Adaptation

CT = Control

	SC = C	L	T
TB = 1	.13250	.12158	.10392
2	.11633	.08200	.07092

CT = Asymptotic

	SC = C	L	T
TB = 1	.20617	.17517	.19183
2	.16992	.09692	.11108

CT = Overtrained

	SC = C	L	T
TB = 1	.14925	.20725	.15292
2	.17867	.15967	.12133

F Ratios for CT X TB X SC Analysis of Variance -
First Interval GSR Magnitude in Testing

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	234.4939	71.87862	1	71.87862
2	CT	R(CT)	1.1720	.7184868	2	.3592434
3	TB	TB,R(CT)	.0727	.001184907	1	.001184907
4	SC	SC,R(CT)	16.4617	.7188506	2	.3594253
5	R(CT)			10.11538	33	.3065265
6	CT X TB	TB,R(CT)	2.0824	.06792212	2	.03396106
7	CT X SC	SC,R(CT)	1.9624	.1713891	4	.04284728
8	TB X SC	TB,SC,R(CT)	.4330	.01069129	2	.005345643
9	TB X R(CT)			.5381904	33	.01630880
10	SC X R(CT)			1.441041	66	.02183395
11	CT X TB X SC	TB,SC,R(CT)	1.1014	.05439174	4	.01359794
12	TB X SC X R(CT)			.8148319	66	.01234594

First Interval GSR Mean Magnitudes for CT X TB X SC
Analysis of Variance Cell Values in Testing

CT = Control

SC =	C	L	T
TB = 1	.72667	.65717	.53892
2	.69158	.76308	.57217

CT = Asymptotic

SC =	C	L	T
TB = 1	.62225	.56533	.51158
2	.58892	.50125	.45517

CT = Overtrained

SC =	C	L	T
TB = 1	.61892	.48733	.48475
2	.63517	.49033	.47308

F Ratios for CT X TB X SC Analysis of Variance -
Second Interval GSR Magnitude in Testing

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	68.6609	8.979362	1	8.979362
2	CT	R(CT)	2.8367	.7419615	2	.3709807
3	TB	TB,R(CT)	3.9965	.06232017	1	.06232017
4	SC	SC,R(CT)	15.8211	.8271912	2	.4135956
5	R(CT)			4.315682	33	.1307783
6	CT X TB	TB,R(CT)	1.5089	.04705739	2	.02352870
7	CT X SC	SC,R(CT)	4.9985	.5226833	4	.1306708
8	TB X SC	TB,SC,R(CT)	2.9350	.06248736	2	.03124368
9	TB X R(CT)			.5145871	33	.01559355
10	SC X R(CT)			1.725368	66	.02614195
11	CT X TB X SC	TB,SC,R(CT)	1.0333	.04399961	4	.0109999
12	TB X SC X R(CT)			.7025948	66	.01064537

Second Interval GSR Mean Magnitudes for CT X TB X SC
Analysis of Variance Cell Values in Testing

CT = Control

	SC = C	L	T
TB = 1	.12142	.07425	.17433
2	.12883	.13575	.12692

CT = Asymptotic

	SC = C	L	T
TB = 1	.42117	.24892	.22792
2	.33917	.21275	.16408

CT = Overtrained

	SC = C	L	T
TB = 1	.35792	.11408	.24792
2	.36900	.08808	.11758

F Ratios for CT X TB X SC Analysis of Variance -
Third Interval GSR Magnitude in Testing

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	150.2363	14.71203	1	14.71203
2	CT	R(CT)	3.0262	.5926897	2	.2963448
3	TB	TB,R(CT)	4.2341	.1245576	1	.1245576
4	SC	SC,R(CT)	24.6264	1.255950	2	.6279750
5	R(CT)			3.231556	33	.09792590
6	CT X TB	TB,R(CT)	3.8772	.2281135	2	.1140567
7	CT X SC	SC,R(CT)	2.5808	.2632370	4	.06580925
8	TB X SC	TB,SC,R(CT)	.1701	.004945755	2	.002472878
9	TB X R(CT)			.9707753	33	.02941743
10	SC X R(CT)			1.683001	66	.02550001
11	CT X TB X SC	TB,SC,R(CT)	1.2568	.07306641	4	.01826660
12	TB X SC X R(CT)			.9592517	66	.01453412

Third Interval GSR Mean Magnitudes for CT X TB X SC
Analysis of Variance Cell Values in Testing

CT = Control

	SC = C	L	T
TB = 1	.20000	.20442	.09992
2	.27017	.19783	.16425

CT = Asymptotic

	SC = C	L	T
TB = 1	.49008	.28758	.28292
2	.42550	.17250	.22167

CT = Overtrained

	SC = C	L	T
TB = 1	.46708	.23600	.29700
2	.35783	.19233	.13067

F Ratios for CT X EP X SC Analysis of Variance -
First Interval GSR Magnitude in Adaptation and Testing

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	290.3579	60.37112	1	60.37112
2	CT	R(CT)	.5924	.2463593	2	.1231796
3	EP	EP,R(CT)	4.3727	.4885823	1	.4885823
4	SC	SC,R(CT)	28.7469	.5419568	2	.2709784
5	R(CT)			6.861346	33	.2079195
6	CT X EP	EP,R(CT)	.7029	.1570804	2	.07854021
7	CT X SC	SC,R(CT)	2.5512	.09619331	4	.02404833
8	EP X SC	EP,SC,R(CT)	1.0542	.01346153	2	.006730765
9	EP X R(CT)			3.687207	33	.1117335
10	SC X R(CT)			.6221399	66	.009426359
11	CT X EP X SC	EP,SC,R(CT)	.5511	.01407552	4	.003518879
12	EP X SC X R(CT)			.4213941	66	.006384756

First Interval GSR Mean Magnitudes for CT X EP X SC Analysis
of Variance Cell Values in Adaptation and Testing

CT = Control

	SC = C	L	T
EP = 1	.54000	.50558	.42258
2	.70908	.71000	.55500

CT = Asymptotic

	SC = C	L	T
EP = 1	.55242	.49400	.45233
2	.60492	.53308	.48300

CT = Overtrained

	SC = C	L	T
EP = 1	.51650	.42467	.42200
2	.62658	.48533	.47917

F Ratios for CT X EP X SC Analysis of Variance -
Second Interval GSR Magnitude in Adaptation and Testing

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	79.4720	5.047905	1	5.047905
2	CT	R(CT)	1.1136	.1414725	2	.07073623
3	EP	EP,R(CT)	19.0663	.5623105	1	.5623105
4	SC	SC,R(CT)	11.5627	.2327370	2	.1163685
5	R(CT)			2.096095	33	.06351799
6	CT X EP	EP,R(CT)	5.5459	.3271265	2	.1635633
7	CT X SC	SC,R(CT)	3.2290	.1299902	4	.03249756
8	EP X SC	EP,SC,R(CT)	10.4204	.1850571	2	.09252852
9	EP X R(CT)			.9732485	33	.02949238
10	SC X R(CT)			.6642345	66	.01006416
11	CT X EP X SC	EP,SC,R(CT)	4.1627	.1478534	4	.03696334
12	EP X SC X R(CT)			.5860511	66	.008879561

Second Interval GSR Mean Magnitudes for CT X EP X SC Analysis
of Variance Cell Values in Adaptation and Testing

CT = Control

	SC = C	L	T
EP = 1	.11325	.12092	.08792
2	.12542	.10500	.15025

CT = Asymptotic

	SC = C	L	T
EP = 1	.05850	..04758	.08192
2	.38050	.23050	.19617

CT = Overtrained

	SC = C	L	T
EP = 1	.14667	.12000	.13992
2	.36350	.10075	.18300

F Ratios for CT X EP X SC Analysis of Variance -
Third Interval GSR Magnitude in Adaptation and Testing

	Source	Error Term	F	Sum of Squares	df	MS
1	Mean	R(CT)	154.7147	8.702169	1	8.702169
2	CT	R(CT)	2.9689	.3339860	2	.1669930
3	EP	EP,R(CT)	24.6989	.7775562	1	.7775562
4	SC	SC,R(CT)	22.9638	.4306227	2	.2153113
5	R(CT)			1.856136	33	.05624655
6	CT X EP	EP,R(CT)	.6729	.04236835	2	.02118418
7	CT X SC	SC,R(CT)	1.9685	.07382673	4	.01845668
8	EP X SC	EP,SC,R(CT)	12.1729	.2155805	2	.1077902
9	EP X R(CT)			1.038888	33	.03148145
10	SC X R(CT)			.6188231	66	.009376105
11	CT X EP X SC	EP,SC,R(CT)	2.1518	.07621729	4	.01905432
12	EP X SC X R(CT)			.5844253	66	.008854926

Third Interval GSR Mean Magnitudes for CT X EP X SC Analysis
of Variance Cell Values in Adaptation and Testing

CT = Control

	SC = C	L	T
EP = 1	.12433	.09625	.08708
2	.23458	.20075	.13175

CT = Asymptotic

	SC = C	L	T
EP = 1	.18758	.13517	.15183
2	.45717	.23008	.25233

CT = Overtrained

	SC = C	L	T
EP = 1	.16408	.18317	.13700
2	.41225	.21367	.21392

APPENDIX 3

RAW DATA

First Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Random Control Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	1.244	.797	1.165	1.030	1.035	1.014	1.089	.988	.909	.873	.564	.987	.179	.545	.849	.898	.674	.305
S_2	.534	.422	.399	.382	.471	.355	.422	.341	.355	.355	.382	.179	.411	.436	.179	.482	.411	-
S_3	.790	1.030	.566	.668	.723	.587	.706	.479	.732	.724	.973	.492	.506	.580	.338	.603	.430	.348
S_4	-	.712	-	-	-	-	-	-	-	-	-	-	.113	-	-	-	-	-
S_5	.607	.503	.503	.482	.545	.355	.458	.411	.458	.436	.305	.534	.355	.411	.458	.341	-	.482
S_6	1.078	1.181	1.055	.803	.446	.697	.534	1.078	.552	.797	.790	.227	.341	.179	-	.399	.201	.179
S_7	.687	.549	.300	.596	.500	.536	.400	.263	.506	.430	.437	.405	.437	.422	.348	.479	.472	.500
S_8	1.048	1.152	.645	.997	.935	.734	1.058	1.058	.720	1.181	.935	.852	1.290	.911	.839	1.272	1.426	1.305
S_9	.903	.913	.355	.848	.617	.179	.665	.253	.140	-	.850	-	.959	.422	.655	.783	.687	.725
S_{10}	.453	.422	.355	.253	.285	.276	.329	.211	.318	.405	.358	.160	.318	.276	.318	.405	.374	.310
S_{11}	.712	.482	.650	.399	.399	.269	.503	.382	.305	.292	.269	.290	.269	.446	.201	-	.326	-
S_{12}	.720	.642	-	.564	.650	.658	-	-	.326	.290	-	.459	-	-	.229	.307	.179	.269

First Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Asymptotic Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.720	.355	.411	.617	.201	.290	.355	.446	-	.201	-	.326	.459	.201	.355	.179	-	.355
S_2	.823	.712	.775	.756	.742	.503	.642	.592	.873	.727	.534	.179	1.018	.534	.370	.422	.292	.253
S_3	.888	1.025	.888	.948	.841	.880	.617	.574	.674	.762	.658	.783	.572	.436	.370	.898	.635	.436
S_4	.674	.756	.699	.674	.580	.810	.307	.446	.399	.697	.608	.471	.482	.482	.545	.459	.399	.307
S_5	.545	.382	.650	.515	.503	.552	.617	.411	.370	.545	.436	.446	.617	.482	.624	.734	.564	.545
S_6	.534	.642	.608	.515	.326	.436	.494	.399	.482	.436	.355	.446	.324	.524	.494	.624	.140	.545
S_7	.530	.276	.358	.253	.276	.211	.276	.263	.318	.365	.430	.422	.338	.391	.318	.338	.329	.253
S_8	1.253	1.018	.705	1.085	.948	.969	.993	1.174	.892	1.253	.892	.580	1.137	1.108	.503	.860	.691	.796
S_9	.665	.649	.430	.365	.374	.437	.113	.241	-	.467	.391	.391	.113	.113	.113	.310	-	.179
S_{10}	.564	.996	.650	.674	.574	.796	.642	.674	.608	.564	.771	.697	1.055	.943	.971	1.301	.960	.705
S_{11}	.545	.253	.341	.411	.341	-	.290	.341	.179	.399	.179	.399	-	-	-	.241	.391	.300
S_{12}	.229	.399	.624	.534	.608	-	-	.305	.140	-	.358	-	-	.381	-	.241	.318	.179

First Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Overtrained Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	-	.381	.400	.530	.400	.113	.487	.285	.192	.566	-	.338	.504	.348	.766	.649	.716	.519
S_2	1.124	.503	.598	.503	.823	.201	-	.598	.416	.540	.422	.549	.467	.310	.620	.629	.211	-
S_3	.459	.503	.471	-	.269	.341	.411	.382	.292	.201	.382	-	.524	.105	-	.436	.201	-
S_4	.624	.665	.624	.771	.727	.624	.691	.552	.658	.616	.665	.580	.552	.750	.592	.756	.608	.399
S_5	1.024	.650	.841	.955	.720	.742	1.080	1.174	.750	1.269	.292	1.177	1.261	1.108	.868	1.174	1.327	1.181
S_6	.620	.358	.861	.191	.310	.405	-	.479	.263	.422	.224	.559	1.042	1.002	.674	1.028	.307	.503
S_7	.642	-	-	-	-	-	-	-	-	-	.253	.305	.552	.436	.370	.459	.370	.326
S_8	.775	.201	.459	.645	.487	.500	.422	.358	.487	.211	.603	.374	-	.160	.285	.492	.437	-
S_9	.487	-	-	-	.224	.596	.191	.179	.263	-	-	-	.071	-	.224	.133	.179	-
S_{10}	.444	.592	.522	.358	.437	.358	.276	.405	.530	.596	.276	.430	.766	.592	.620	.716	.668	.580
S_{11}	.823	.564	.985	.564	.534	.411	.552	.341	.436	.324	.524	.436	.446	.446	.399	.382	.326	.229
S_{12}	.829	.829	1.154	.534	.201	.471	.617	.503	.515	.927	-	-	.545	.251	-	.355	.471	-

First Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Random Control Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.682	.494	.545	.885	.803	.665	1.003	.848	.411	.503	.459	.727	.783	.525	-	.756	.903	.341
S_2	.732	.783	.348	.807	.624	.338	.580	.693	.241	.580	.890	.858	.745	.453	.160	.759	.911	.286
S_3	.559	.750	.400	.639	.639	.596	.745	.620	.838	.884	.723	.761	.629	.687	.665	.511	.771	.679
S_4	.736	.536	.642	.759	.993	.355	.741	1.042	.224	1.017	1.039	.973	.860	1.112	.978	.903	.515	.592
S_5	.503	.552	.783	.617	.660	.564	.756	.545	.682	.771	.756	.796	.734	.482	.742	.837	1.009	.699
S_6	1.069	.727	.635	.399	.750	.253	.665	.292	.140	.592	1.028	.545	.987	.987	.411	.650	.503	-
S_7	.759	.540	.587	.603	.649	.766	.701	.649	.969	.790	.834	.790	.714	.552	.569	.569	.787	.511
S_8	.864	1.136	.677	1.181	1.098	1.143	1.413	1.152	.911	.887	1.398	1.266	1.172	1.207	1.088	1.058	1.559	.9577
S_9	.580	.355	.860	.927	.552	.608	.903	-	-	.411	.617	.932	1.038	1.038	.370	.341	.777	.471
S_{10}	.530	.453	.487	.487	.338	.300	.358	.444	.522	.487	.437	.472	.472	.444	.253	.365	.241	.133
S_{11}	.324	.482	.699	.494	.574	.436	.471	.399	-	.436	.140	.446	.290	.411	.229	.422	.598	.179
S_{12}	.635	.868	.503	1.009	.783	.705	1.063	.837	.564	.803	.810	.494	.524	1.030	.873	.617	.848	.355

First Interval GSR √Micromhos Conductance Change for Asymptotic Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.338	.160	.140	.191	.113	.071	.263	.224	.133	.211	.133	.191	.300	-	-	.211	-	.160
S_2	.909	.928	1.042	.564	.617	.880	.764	.552	.650	.816	.948	.201	.691	.908	.658	.370	.927	.253
S_3	.898	.796	.642	1.009	.796	.837	.816	.705	.524	.783	.665	.503	.635	.580	.682	.564	.608	.885
S_4	.943	1.131	.790	1.244	.782	.598	1.290	.887	1.097	.776	.790	.802	.935	.750	.790	.911	.761	.677
S_5	.459	.524	.470	.482	.503	.459	.624	.482	.411	.524	.524	.545	.617	.564	.617	.697	.598	.459
S_6	.598	.650	.524	.459	.370	.650	.691	.534	.608	.783	.580	.617	.598	.650	.592	.734	.642	.534
S_7	.701	.679	.596	.405	.649	.530	.620	.607	.668	.492	.624	.607	.566	.530	.391	.300	.416	.487
S_8	.796	.868	.524	.837	.705	.635	1.181	1.088	.665	1.169	.515	.201	.908	.608	.382	.823	1.095	.179
S_9	.253	.160	-	-	-	-	.113	-	-	.318	-	-	.405	-	-	.224	.113	.405
S_{10}	.987	.742	.552	1.117	.665	.756	.960	.921	.482	.398	.179	.873	1.048	.635	.682	1.285	.206	.592
S_{11}	-	-	.211	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.133
S_{12}	.503	.712	.734	.720	.971	.734	.674	.837	.816	.140	.996	.837	.771	.635	.617	.691	.873	.848

First Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Overtrained Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.759	.620	.607	.566	.179	.629	.741	.732	.607	.467	.725	.437	.649	.552	-	.615	.738	-
S_2	.810	.023	.382	.459	.837	.382	.783	.201	.105	.552	.668	.253	.374	.378	.241	.725	.587	1.124
S_3	.411	.482	.292	.564	.411	.324	.326	.422	.179	.459	.503	-	.399	.341	-	-	.411	.482
S_4	.668	.500	.603	.861	.487	.472	.741	.683	.738	.841	.620	.875	.766	.381	.732	.815	.633	.500
S_5	-	-	-	-	.133	.276	-	.348	.160	.179	.253	.211	.348	.113	.160	.492	.374	.179
S_6	.580	.727	.635	.803	.372	.399	.892	.382	.742	.853	.545	.803	.966	.515	.617	.580	.253	.598
S_7	.598	.459	.399	.598	.503	.617	.580	.552	.635	.848	.665	.545	.796	.742	.580	.860	.545	.534
S_8	.996	.411	.699	.943	.552	.459	1.108	.355	.816	1.038	-	-	.592	.229	.355	.459	.650	1.055
S_9	.771	.534	.766	.503	.608	.382	.796	.552	.503	.764	.382	.624	.927	.642	.341	1.287	.574	.691
S_{10}	.823	.741	.810	.615	.741	.788	.800	.800	.457	.800	.716	1.012	.492	.706	.716	.820	.714	.723
S_{11}	.816	.803	.705	.783	.771	.564	.764	.459	.742	.756	.697	.691	.764	.524	.816	.948	.436	.712
S_{12}	.329	.211	.160	.500	.160	.253	-	-	.191	.338	.133	-	.113	.224	.179	.191	.253	.253

Second Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Random Control Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.624	-	-	-	-	.140	-	-	-	-	-	-	-	.382	.598	-	-	-
S_2	.140	.307	-	-	-	.140	-	-	-	-	-	-	-	-	-	-	-	-
S_3	.477	-	-	.348	.519	-	-	.416	-	-	-	-	-	-	-	-	.211	-
S_4	.422	-	.482	.285	.576	.191	.381	.318	.530	.530	-	.365	-	.133	.113	.285	.133	.133
S_5	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.324	-
S_6	-	.624	.932	-	-	-	.326	.269	-	.201	-	-	-	.179	-	-	.140	.105
S_7	.511	.338	.160	-	-	.253	-	.253	-	-	-	-	-	-	-	-	-	-
S_8	-	-	-	.645	.500	.321	-	-	.500	.405	-	.354	-	.815	-	.321	.321	-
S_9	-	.269	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{10}	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{11}	.436	-	-	-	-	-	-	.179	-	-	-	-	-	-	-	.382	-	-
S_{12}	.524	-	1.025	-	-	-	.552	.564	-	-	.503	-	.382	.459	-	-	-	-

Second Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Asymptotic Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.446	-	-	-	-	-	.179	-	-	-	-	-	-	-	-	-	-	-
S_2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_3	.688	-	-	-	.253	-	-	-	.471	.140	.525	-	.411	-	-	-	.382	-
S_4	-	-	-	-	.572	-	-	-	-	-	-	-	.617	-	-	.355	.446	-
S_5	.399	-	.269	-	-	-	-	-	.179	-	-	.201	.436	.411	-	.355	-	.370
S_6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.105	-	-	.140
S_7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_8	-	-	.810	-	-	-	-	-	1.009	-	-	.382	-	.411	.682	-	.269	-
S_9	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{10}	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.564	-	-
S_{11}	-	-	.515	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{12}	-	-	.201	-	-	-	-	-	-	-	-	-	-	.179	-	-	-	-

Second Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Overtrained Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.649	-	-	-	-	-	-	-	-	.596	-	-	-	-	-	-	-	.725
S_2	.477	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	.113
S_3	.290	-	.370	-	-	-	-	-	.140	.459	-	-	.201	-	.494	.253	-	-
S_4	.253	.201	-	-	-	-	-	-	-	-	-	-	.816	-	-	-	-	-
S_5	.382	.848	.880	-	-	-	-	-	.411	-	-	-	-	-	-	-	-	-
S_6	-	-	-	.179	-	-	-	-	-	-	.506	.329	.374	-	.269	-	.459	.411
S_7	-	.436	.399	.411	.355	.382	.534	.382	.399	.580	-	-	-	-	-	-	-	-
S_8	-	-	-	.649	.766	.834	-	.522	.191	.624	-	.365	1.012	.732	.587	.706	.672	.841
S_9	-	.437	-	-	.405	-	-	.108	-	-	-	-	-	-	-	-	-	-
S_{10}	-	-	-	-	.365	-	.348	-	-	-	.329	.179	-	-	.273	-	.759	-
S_{11}	.482	-	-	-	-	-	-	-	-	-	-	-	-	-	.382	-	-	-
S_{12}	-	-	-	-	-	-	-	-	-	.324	-	1.244	-	-	.269	-	-	-

Second Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Random Control Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	-	.515	.399	-	-	.324	-	-	-	-	-	-	.503	-	-	.269	-	.269
S_2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_3	.374	-	.381	.607	.336	.400	-	-	.552	-	-	-	-	.437	.487	.133	-	-
S_4	-	-	-	.133	-	-	.374	-	-	-	-	-	.179	-	-	.727	.229	-
S_5	-	-	.270	-	.201	.459	-	-	-	.292	-	-	-	-	.179	-	.399	-
S_6	-	-	-	-	-	.179	-	-	.253	-	-	-	-	-	-	-	.292	-
S_7	-	.687	.160	-	-	.430	-	.276	-	-	.179	.224	.381	.276	.587	-	.467	-
S_8	.455	.455	.321	.455	-	.852	.405	-	-	-	.381	.479	1.382	.876	.678	.352	-	.645
S_9	.712	-	-	-	-	-	-	-	-	-	-	-	-	-	.341	-	-	.253
S_{10}	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{11}	-	.201	-	.229	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{12}	.105	-	.682	.534	-	-	-	-	.617	-	.742	.436	.436	.624	-	-	-	-

Second Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Asymptotic Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.300	-	-	.160	-	.224	.191	-	.160	.133	-	-	-	-	-	.300	-	-
S_2	.800	-	-	-	-	-	.617	-	-	.422	-	-	-	-	-	.179	-	-
S_3	.269	.592	-	.734	.635	.179	.534	.446	.201	.326	.611	-	.370	-	-	.665	.253	-
S_4	.943	-	-	.955	1.154	.503	1.336	.997	.955	.852	.887	.628	1.050	.761	.519	1.030	.734	.354
S_5	.269	.307	.324	.229	.253	.292	.370	.382	.341	.382	.382	.382	.326	.290	.399	.324	.253	-
S_6	.446	.382	.564	.650	.635	.720	.399	-	-	-	.482	.179	.534	.253	.650	.494	.253	-
S_7	.500	.693	.679	.615	-	.603	.479	-	-	-	-	-	.519	-	.191	.211	.358	.224
S_8	-	-	-	-	.436	-	-	-	-	-	.564	-	-	.140	-	-	.697	-
S_9	.391	.224	.263	.310	.179	.133	-	-	.133	-	.133	.179	.437	-	-	-	-	-
S_{10}	-	-	.650	.564	-	-	.783	-	-	-	-	.674	.918	-	.635	.908	-	.140
S_{11}	.329	.224	.241	.133	-	-	.253	-	.113	.263	.133	-	.191	-	-	-	-	-
S_{12}	.253	.635	.382	.691	.370	.326	.682	.422	.229	.564	-	.446	.324	.494	.326	.503	-	-

Second Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Overtrained Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.576	.285	.693	.430	.285	-	-	.506	-	.504	-	.537	.338	-	-	-	-	-
S_2	.471	-	.494	.251	-	-	.205	-	.290	.540	-	-	-	-	.276	-	-	-
S_3	.572	.105	.269	.503	-	.105	.574	-	.269	.624	.324	-	.436	.140	.140	.513	.179	.515
S_4	-	-	-	.310	-	.300	-	-	-	.530	.422	-	-	-	-	.453	-	-
S_5	.307	.290	.269	.179	-	-	.566	-	-	-	-	-	-	-	-	.253	-	-
S_6	.446	.251	.253	.810	-	.305	.545	.307	.460	.665	-	-	.564	-	.382	.382	.201	-
S_7	.459	.422	.253	.326	-	.253	.411	.411	.355	.598	-	-	.564	.503	-	.650	-	-
S_8	.572	.229	.534	1.069	.201	.032	1.099	-	.662	1.133	-	-	.326	-	-	.515	.446	-
S_9	.305	.524	1.048	-	.292	.411	.140	-	.494	.635	.422	.574	.355	.534	.382	.253	-	-
S_{10}	-	-	.519	.633	-	.492	-	-	-	.596	-	.744	.437	-	-	.620	-	.576
S_{11}	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{12}	.629	-	-	.358	-	-	.160	-	.179	.374	-	.113	.224	-	-	.211	-	-

Third Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for
Random Control Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.494	-	-	-	.411	-	-	.382	.355	-	-	-	.201	-	.341	-	-	.422
S_2	-	.140	.140	.140	.229	.179	.326	-	-	.253	-	.482	-	-	-	.105	-	-
S_3	.241	.741	-	.391	-	-	.479	.276	.338	-	-	-	.453	.300	.329	-	-	-
S_4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_5	.201	-	-	.105	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_6	-	-	.323	.253	-	-	.326	-	-	.665	.229	-	.253	-	-	-	.307	-
S_7	.300	.467	-	.179	.160	.253	-	.160	.179	.241	.113	.113	-	.391	.113	.276	.133	-
S_8	-	-	-	.537	.611	.500	.352	-	.381	.628	-	.354	.537	.691	-	.381	.790	.405
S_9	.355	.292	-	-	-	-	-	-	.290	-	-	-	-	-	-	.211	-	-
S_{10}	-	.140	.105	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{11}	-	-	.253	-	-	-	.105	-	-	-	-	-	-	-	-	-	-	-
S_{12}	-	.382	.453	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-

Third Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Asymptotic Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	-	-	-	.503	-	-	.436	-	-	-	-	.269	-	-	-	-	.105	-
S_2	-	-	.446	.292	-	-	-	-	.290	-	-	-	-	-	-	-	-	-
S_3	.627	.725	1.146	.665	.580	.482	.617	.290	.803	.324	-	-	.771	.771	.436	.399	-	-
S_4	.253	.292	.290	.341	.446	.201	-	.269	.229	-	-	.292	.253	.341	.201	.253	-	.411
S_5	.459	-	.446	.105	.290	.370	.105	-	-	.382	-	-	.422	-	.292	.436	-	.341
S_6	.422	.253	.341	.446	.370	-	.253	-	-	-	-	-	-	.229	-	.460	.253	-
S_7	-	-	.113	-	-	-	-	-	-	.179	.241	-	.211	-	.179	-	.160	-
S_8	.399	.564	-	-	-	-	.860	-	1.235	.229	.370	1.315	.817	.307	-	-	-	-
S_9	-	-	.113	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{10}	-	.436	-	-	.720	-	-	.341	-	-	-	-	-	-	-	.665	.515	-
S_{11}	.326	.382	.269	.140	.203	-	.179	-	.140	-	-	-	-	-	-	.191	.191	-
S_{12}	-	.140	-	-	-	-	-	-	-	-	-	.160	-	-	.113	.133	-	-

Third Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Overtrained Subjects in Adaptation

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	-	-	-	-	-	-	.224	-	-	-	-	-	-	.179	-	-	-	-
S_2	-	-	.305	.253	-	-	-	-	-	.422	-	-	.422	.297	-	-	-	-
S_3	.411	.370	.341	-	.269	.229	-	-	.229	.269	.201	.269	-	-	.307	.290	-	-
S_4	.370	.482	.370	-	.436	-	-	.307	.229	-	.436	-	.370	.229	.253	-	.253	.446
S_5	.699	.650	-	.665	.691	.524	-	.617	.524	.290	-	.292	.459	-	-	.665	-	-
S_6	-	.318	-	.160	.329	.391	-	-	.160	.113	.276	.620	.391	.572	-	-	-	.482
S_7	-	-	.307	-	-	.179	-	-	-	-	-	-	-	-	.482	-	-	-
S_8	-	.742	-	.179	-	-	.276	-	-	.253	.338	-	-	-	.310	.318	-	-
S_9	.310	.519	-	-	.318	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{10}	.263	.329	-	.224	-	.263	.211	.191	.318	.400	.500	.160	.338	.253	.253	.437	.329	.224
S_{11}	-	.382	-	.355	.179	-	-	-	-	.179	.269	-	-	.179	-	.140	-	.105
S_{12}	.582	.341	.341	.201	-	-	-	-	.803	-	1.112	-	-	-	-	.699	.324	.179

Third Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Random Control Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	-	.459	.892	.494	-	-	.382	.324	-	-	-	-	.253	-	-	.712	-	-
S_2	-	-	-	-	.338	-	-	-	-	.710	-	-	-	.179	.133	-	-	-
S_3	-	.437	-	.358	.566	-	-	.318	.566	.253	.500	.500	.566	-	-	.668	.329	.400
S_4	-	-	-	-	.276	-	-	.405	-	.191	.911	.191	.592	-	.771	.494	-	-
S_5	.229	-	-	-	-	.201	-	.229	-	.269	.307	-	.459	-	.201	.727	.251	.727
S_6	.471	-	.382	.482	.382	.140	.411	.355	.253	.253	.411	.140	-	-	-	-	.140	-
S_7	.519	.338	-	.587	-	.467	-	.453	.263	-	.374	.300	.437	.453	-	.263	.741	.310
S_8	.430	-	-	.405	.628	-	.706	.677	-	.997	-	.576	.677	1.136	-	.645	-	-
S_9	-	.307	-	-	-	-	-	-	-	-	.411	-	.140	-	-	-	.253	.201
S_{10}	.191	.263	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
S_{11}	-	.201	-	.399	.411	-	.355	-	-	-	.459	.305	-	.269	.179	-	-	.179
S_{12}	-	-	.179	.494	-	.269	.292	-	-	.422	-	.253	-	-	.355	-	-	.201

Third Interval GSR $\sqrt{\text{Micromhos Conductance Change}}$ for Asymptotic Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.391	-	-	.224	-	-	.362	-	.285	-	-	-	.300	-	.738	-	-	-
S_2	.453	-	-	.783	-	-	-	-	-	-	-	-	.436	-	.290	.545	-	-
S_3	1.030	.873	.665	.777	.382	.399	.482	-	.471	.580	-	-	.324	.140	.399	.624	.307	-
S_4	.977	.898	-	1.024	.292	.742	.790	.500	.381	.900	.405	.430	.887	.285	.706	-	.611	.595
S_5	.253	.269	.326	.422	.399	.355	.503	.370	.355	.436	.290	.482	.370	.292	.422	.411	.290	-
S_6	.697	.459	.471	-	.524	.370	.574	.355	.399	.552	.459	.665	.382	.545	.750	.269	.446	.355
S_7	.615	.492	.592	.679	.511	.549	.540	.552	.549	.487	-	.224	.338	.253	.224	.530	-	-
S_8	.771	-	-	.253	.399	.503	.201	-	-	-	-	.140	.598	-	.140	.292	-	-
S_9	.358	.276	.348	-	-	.500	.133	-	.191	.191	.276	-	-	-	-	.133	-	.241
S_{10}	.810	-	-	.727	.885	.580	1.112	.494	-	.705	.290	-	1.017	.201	-	1.310	-	-
S_{11}	.457	-	-	.391	.113	.160	.113	-	.179	-	-	-	.133	-	-	.133	-	-
S_{12}	.229	.471	.140	-	.355	.370	.524	.494	.326	.617	-	.674	.829	.580	.524	.987	.545	-

Third Interval GSR √Micromhos Conductance Change for Overtrained Subjects in Testing

	Triads																	
	1			2			3			4			5			6		
	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T	Cp	L	T
S_1	.750	.253	.668	.285	.629	.422	.566	-	.639	-	-	.160	.160	.273	.365	.211	-	-
S_2	.592	.459	-	.699	.341	.422	-	-	-	.253	.133	-	.537	-	.160	-	.133	-
S_3	.552	.355	-	.459	-	-	.436	-	.140	.422	-	.179	.494	.163	-	.482	-	-
S_4	.467	.487	-	.750	.160	.607	.665	.552	.276	.790	.687	.422	.741	.263	.492	.519	.318	-
S_5	-	-	-	.133	.133	-	.224	-	-	.113	.133	-	.113	.113	-	.381	.318	-
S_6	.545	-	.399	.341	.307	.290	.691	.292	.608	.810	.292	.292	.292	-	-	.868	.635	-
S_7	.650	.253	-	.503	-	.459	.253	.436	.355	-	.201	.503	.305	-	-	.534	-	.503
S_8	.885	-	.742	.783	-	-	1.038	-	-	.608	-	-	.436	-	-	-	-	-
S_9	.650	.229	.519	.307	-	.422	.524	.341	.411	.545	.552	-	.290	.229	.253	-	.727	.324
S_{10}	.716	.592	.519	.639	.639	.453	.391	.224	.348	.596	.300	-	-	-	.253	.559	.348	.211
S_{11}	.564	.592	.290	.370	.459	.515	-	-	.292	.382	.269	.229	.253	.324	.370	.598	.307	-
S_{12}	.224	.467	.300	-	-	.358	.179	.300	.253	.607	-	-	-	.211	-	-	-	-

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Asymptotic Subjects during Compound Conditioning Acquisition

	Trials									
	1	2	3	4	5	6	7	8	9	10
S_1	.000	.503	.201	.430	.276	.114	.191	.179	.114	.160
S_2	.105	.253	1.001	.269	.823	.750	.494	.000	.503	.000
S_3	.174	.837	1.112	1.019	.411	.873	.624	.609	.459	.609
S_4	.382	.399	.423	.382	.459	.459	.341	.201	.253	.140
S_5	.618	.524	.674	.564	.552	.552	.503	.618	.503	.552
S_6	.292	.691	.580	.545	.618	.534	.437	.342	.370	.326
S_7	.285	.358	.430	.338	.444	.472	.330	.309	.348	.318
S_8	.860	1.109	.459	1.186	1.070	1.207	1.058	.628	.539	.864
S_9	.114	.620	.592	.487	.522	.492	.552	.559	.492	.179
S_{10}	.908	1.168	1.207	1.305	1.143	1.030	1.030	1.048	.677	1.020
S_{11}	.285	.000	.355	.292	.000	.000	.000	.114	.114	.000
S_{12}	.179	.503	.515	.000	1.414	.564	.342	.269	.140	.370

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Overtrained Subjects during Trials One through Ten of Compound Conditioning Acquisition

	Trials									
	1	2	3	4	5	6	7	8	9	10
S_1	.766	.831	.839	.790	.771	.649	.545	.494	.545	.515
S_2	.816	.503	.503	.574	.503	.000	.503	.503	.307	.370
S_3	.423	.253	.552	.140	.503	.179	.423	.140	.355	.201
S_4	.660	.783	.873	.728	.660	.790	.598	.399	.515	.524
S_5	1.256	1.312	1.336	1.207	1.376	1.360	1.469	1.239	1.351	1.312
S_6	.796	.750	1.117	.927	1.099	.830	.993	.750	.837	.624
S_7	.342	.482	.355	.000	.326	.355	.290	.342	.269	.269
S_8	.552	.574	.796	1.020	.611	.628	.628	.790	.245	.539
S_9	.000	.687	.348	.365	.430	.000	.000	.500	.348	.330
S_{10}	.693	1.025	.828	.766	.566	.732	.453	.630	.615	.655
S_{11}	.524	.000	.699	.382	.253	.201	.399	.382	.423	.342
S_{12}	.764	.324	.342	.000	.355	1.287	.494	.179	.382	.355

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Overtrained Subjects during Trials Eleven through Twenty of Compound Conditioning Acquisition

	Trials									
	11	12	13	14	15	16	17	18	19	20
S_1	.524	.534	.411	.460	.308	.435	.582	.435	.398	.289
S_2	.356	.236	.000	.374	.407	.524	.505	.275	.678	.238
S_3	.325	.482	.000	.000	.460	.325	.289	.250	.176	.204
S_4	.384	.705	.609	.411	.460	.609	.600	.176	.576	.356
S_5	1.390	1.019	1.019	1.352	1.289	1.240	1.189	.888	1.273	1.442
S_6	.897	1.044	.690	.783	.460	.000	.590	.697	.340	.289
S_7	.370	.325	.384	.370	.325	.000	.459	.451	.319	.339
S_8	.432	.248	.398	.000	.626	.340	.000	.176	.204	.823
S_9	.339	.000	.000	.275	.238	.319	.288	.000	.000	.000
S_{10}	.639	.649	.565	.706	.438	.629	.609	.565	.609	.374
S_{11}	.356	.264	.194	.524	.565	.451	.459	.530	.505	.374
S_{12}	.250	.204	.000	.460	.384	.000	.505	.137	.252	.194

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Overtrained Subjects during Trials Twenty-one through Thirty of Compound Conditioning Acquisition

	Trials									
	21	22	23	24	25	26	27	28	29	30
S_1	.541	.211	.422	.310	.391	.487	.374	.511	.540	.330
S_2	.422	.506	.179	.668	.285	.487	.437	.430	.224	.430
S_3	.179	.290	.104	.292	.290	.342	.000	.105	.342	.179
S_4	.552	.437	.292	.416	.993	.292	.253	.574	.229	.742
S_5	1.009	1.163	.736	.864	1.538	.000	.922	.876	.924	1.280
S_6	.635	.355	.290	.179	.000	.534	.534	.459	.000	.326
S_7	.348	.659	.559	.467	.639	.714	.422	.695	.566	.655
S_8	.515	.229	.253	.000	.241	.437	.635	.000	.545	.269
S_9	.000	.358	.000	.253	.000	.000	.241	.329	.000	.000
S_{10}	.659	.587	.580	.655	.540	.725	.630	.710	.639	.540
S_{11}	.241	.374	.587	.405	.607	.645	.896	.564	.796	.326
S_{12}	.000	.179	.133	.179	1.140	.300	.000	.348	.603	.000

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Control Subjects during Trials One through Ten of Compound Conditioning Acquisition

	Trials									
	1	2	3	4	5	6	7	8	9	10
S_1	-	-	-	.996	-	.823	.000	.304	-	-
S_2	-	-	-	-	-	.292	-	.437	.105	.292
S_3	-	-	.603	.587	-	.511	-	-	.329	-
S_4	.000	-	-	.000	.000	-	.000	-	-	.000
S_5	.305	-	.000	.201	.000	.397	-	.471	-	-
S_6	-	-	-	.229	-	.269	-	-	.459	-
S_7	.511	.437	-	.540	-	-	.500	-	.422	-
S_8	-	-	1.058	-	-	1.019	-	1.078	-	-
S_9	.728	-	.860	-	.860	.903	.666	-	.552	-
S_{10}	-	-	-	-	-	-	.263	-	.276	-
S_{11}	-	.524	-	.459	-	.446	.370	.355	.482	.292
S_{12}	-	-	.000	-	.000	-	-	.211	-	.114

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Control Subjects during Trials Eleven through Twenty of Compound Conditioning Acquisition

	Trials									
	11	12	13	14	15	16	17	18	19	20
S_1	.545	.609	.459	-	.545	-	.810	.000	.324	-
S_2	.000	-	.201	-	-	.382	-	-	-	.140
S_3	-	-	.388	.630	.261	.241	.241	.300	.374	-
S_4	-	-	-	.133	.000	.000	.000	.000	.000	.000
S_5	-	-	-	-	.355	-	.307	.355	-	-
S_6	.000	-	.422	.459	-	.355	.564	.140	.000	-
S_7	-	.000	-	.338	-	.133	.160	-	.358	.319
S_8	-	1.040	1.136	.677	1.040	-	1.305	-	1.030	1.116
S_9	.459	-	.000	-	.771	.635	-	.756	.000	-
S_{10}	.285	.133	.000	.071	-	-	-	-	-	-
S_{11}	.000	.307	-	.140	-	.000	-	.272	-	.204
S_{12}	-	-	.405	.000	.000	.679	.133	-	-	-

First Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Control Subjects during Trials Twenty-one through Thirty of Compound Conditioning Acquisition

	Trials									
	21	22	23	24	25	26	27	28	29	30
S_1	.742	-	.000	-	.500	-	-	.976	-	.000
S_2	-	.000	.325	-	-	-	-	-	.113	-
S_3	.775	-	.264	-	.758	-	.438	. 451	-	.451
S_4	-	.000	-	.000	-	.000	-	-	.109	.000
S_5	-	-	-	.459	.370	.503	-	-	.590	-
S_6	.503	-	-	-	.460	-	.000	.448	-	.651
S_7	.109	.319	.225	.357	.319	-	-	-	.000	-
S_8	.934	.840	1.456	-	-	.676	-	.805	-	-
S_9	.000	-	-	.000	-	.682	-	-	.000	-
S_{10}	.191	.160	.300	.310	.178	.109	-	-	-	.366
S_{11}	.204	-	.176	-	-	.272	-	-	-	.000
S_{12}	.194	.383	-	.238	-	-	.400	.178	.158	-

Second Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Asymptotic Subjects during Compound Conditioning Acquisition

	Trials									
	1	2	3	4	5	6	7	8	9	10
S_1	.000	.382	.229	.000	.000	.179	.160	.160	.000	.000
S_2	.000	.000	.000	.201	.000	.000	.000	.000	.140	.000
S_3	.179	.201	.635	.705	.435	.842	.307	.728	.000	.503
S_4	.000	.750	.515	.494	.503	.423	.382	.269	.201	.342
S_5	.292	.342	.618	.399	.305	.342	.326	.253	.355	.290
S_6	.000	.000	.270	.000	.370	.000	.229	.307	.253	.564
S_7	.000	.416	.318	.179	.391	.179	.276	.160	.160	.276
S_8	.000	.000	.000	.411	.000	.247	.000	.000	.000	.000
S_9	.000	.000	.506	.624	.000	.577	.437	.400	.457	.375
S_{10}	.000	1.169	1.172	.900	.997	.802	.677	.706	.761	.405
S_{11}	.000	.179	.437	.000	.179	.000	.285	.000	.000	.114
S_{12}	.000	.201	.618	.000	.326	.524	1.039	.000	.201	.201

Second Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Overtrained Subjects during Trials One through Ten of Compound Conditioning Acquisition

	Trials									
	1	2	3	4	5	6	7	8	9	10
S_1	.000	.668	.695	.657	.444	.348	.515	.000	.482	.574
S_2	.000	.000	.000	.000	.642	.437	.494	.000	.355	.000
S_3	.515	.230	.000	.253	.290	.399	.201	.269	.355	.324
S_4	.000	.691	.712	.370	.564	.459	.503	.515	.000	.355
S_5	.000	.922	.887	.852	1.152	.776	.000	.989	.720	.706
S_6	.000	1.063	.885	.728	.823	.564	.494	.000	.742	.325
S_7	.000	.000	.411	.382	.355	.370	.355	.269	.290	.253
S_8	.868	.342	.762	.802	.802	1.290	.628	.577	.645	.876
S_9	.000	.000	.000	.374	.000	.624	.487	.000	.000	.000
S_{10}	.000	.000	.566	.695	.603	.716	.732	.374	.310	.382
S_{11}	.000	.503	.000	.000	.000	.229	.000	.000	.446	.000
S_{12}	.000	.000	.269	.290	.253	.756	.873	.459	.292	.580

Second Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Overtrained Subjects during Trials Eleven through Twenty of Compound Conditioning Acquisition

	Trials									
	11	12	13	14	15	16	17	18	19	20
S_1	.204	.460	.000	.250	.000	.325	.000	.000	.000	.482
S_2	.000	.000	.275	.000	.576	.000	.000	.309	.383	.309
S_3	.460	.411	.289	.000	.340	.325	.000	.000	.000	.204
S_4	.250	.000	.460	.250	.144	.000	.000	.000	.451	.460
S_5	.000	.611	.456	1.223	.956	.611	.815	.628	.000	1.144
S_6	.697	.742	.682	.720	.000	.435	.582	.000	.667	.617
S_7	.000	.398	.000	.325	.000	.000	.000	.000	.194	.178
S_8	.988	.864	.204	.651	.810	.000	.674	.000	.356	.000
S_9	.000	.209	.391	.000	.309	.000	.000	.158	.000	.252
S_{10}	.391	.438	.158	.264	.319	.000	.723	.000	.000	.414
S_{11}	.370	.275	.000	.000	.000	.414	.000	.000	.000	.383
S_{12}	.000	.000	.600	.289	.000	.000	.530	.264	.000	.000

Second Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Overtrained Subjects during Trials Twenty-one through Thirty of Compound Conditioning Acquisition

	Trials									
	21	22	23	24	25	26	27	28	29	30
S_1	.000	.000	.000	.000	.405	.330	.000	.253	.000	.382
S_2	.566	.253	.000	.000	.382	.000	.000	.179	.000	.000
S_3	.179	.229	.370	.201	.324	.326	.229	.000	.253	.292
S_4	.000	.140	.000	.479	.318	.201	.000	.000	.000	.000
S_5	.519	1.239	.456	.790	.000	.000	.000	.382	.840	.405
S_6	.000	.742	.305	.810	.524	.382	.307	.728	.000	.253
S_7	.191	.000	.382	.492	.000	.000	.540	.531	.338	.000
S_8	.253	.000	.000	.000	1.025	.853	.000	.000	.000	.000
S_9	.472	.000	.318	.358	.310	.416	.310	.348	.365	.285
S_{10}	.264	.382	.191	.695	.253	.000	.000	.318	.511	.000
S_{11}	.222	.000	.000	.356	.356	.318	.467	.000	.292	.000
S_{12}	.000	.000	.000	.000	.224	.000	.000	.437	.000	.816

Second Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Control Subjects during Trials One through Ten of Compound Conditioning Acquisition

	Trials									
	1	2	3	4	5	6	7	8	9	10
S_1	-	-	-	.000	-	.437	.000	.000	-	-
S_2	-	-	-	-	-	.326	-	.000	.000	.253
S_3	-	-	.253	.348	-	.191	-	-	.000	-
S_4	.318	-	-	-	.000	-	.000	-	-	.318
S_5	.000	-	.515	.000	.459	.000	-	.000	-	-
S_6	-	-	-	.000	-	.000	-	-	.000	-
S_7	.179	.000	-	.000	-	-	.000	-	.071	-
S_8	-	-	1.207	-	-	.479	-	.628	-	-
S_9	.000	-	.000	-	.000	.000	.000	-	.000	-
S_{10}	-	-	-	-	-	-	.000	-	.000	-
S_{11}	-	.000	-	.000	-	.399	.000	.000	.000	.000
S_{12}	-	-	.000	-	.000	-	-	.000	-	.000

Second Interval GSR $\sqrt{\text{Micromhos Conductance Changes}}$ for Control Subjects during Trials Eleven through Twenty of Compound Conditioning Acquisition

	Trials									
	11	12	13	14	15	16	17	18	19	20
S_1	.000	.000	.000	-	.000	-	.000	.000	.000	-
S_2	.000	-	.000	-	-	.000	-	-	-	.399
S_3	-	-	.000	.000	.000	.000	.000	.000	.000	-
S_4	-	-	-	.000	.191	.000	.160	.000	.000	.000
S_5	-	-	-	-	.000	-	.000	.000	-	-
S_6	.000	-	.000	.000	-	.000	.000	.000	.000	-
S_7	-	.000	-	.224	-	.000	.000	-	.000	.000
S_8	-	.839	.000	.000	.500	-	.577	-	.000	.382
S_9	.000	-	.665	-	.697	.000	-	.000	.000	-
S_{10}	.000	.211	.000	.000	-	-	-	-	-	-
S_{11}	.000	.000	-	.000	-	.000	-	.000	-	.000
S_{12}	-	-	.000	.000	.531	.000	.000	-	-	-

Second Interval GSR $\sqrt{\text{Micromhos}}$ Conductance Changes for Control Subjects during Trials Twenty-one through Thirty of Compound Conditioning Acquisition

	Trials									
	21	22	23	24	25	26	27	28	29	30
S_1	.000	-	.000	-	-	.000	-	.000	-	.000
S_2	-	.292	.000	-	-	-	-	-	.179	-
S_3	.000	-	.357	-	.479	-	.000	.192	-	.212
S_4	-	.451	-	.000	-	.459	-	-	.486	.000
S_5	-	-	-	.000	.144	.399	-	-	.000	-
S_6	.000	-	-	-	.000	-	.000	.000	-	.000
S_7	.000	.298	.194	.252	.238	-	-	-	.339	-
S_8	.248	.000	.456	-	-	.000	-	.304	-	-
S_9	.000	-	-	.250	-	.000	-	-	.000	-
S_{10}	.000	.000	.000	.000	.077	.000	-	-	-	.000
S_{11}	.000	-	.000	-	-	.000	-	-	-	.000
S_{12}	.000	.000	-	.000	-	-	.000	.000	.000	-