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ABSTRACT

Groups of brain damaged and normal children were subdivided into young (age range 6 to 9 years) and old (age range 10 to 13 years) groups and subjected to an auditory reaction time procedure consisting of regular and irregular preparatory interval conditions. The most important results were as follows: the performances of the young normal, old normal, and old brain damaged groups were superior to that of the young brain damaged group; there was no significant differences between the performance of the old normal and the old brain damaged groups; the set index formula was not more effective in differentiating between the performance of the young brain damaged and young normal groups or between the young brain damaged and old brain damaged groups than were other, more easily derived, indices. The results of the current investigation were highly similar to those obtained in previous studies which employed visual reaction time procedures (Czudner & Rourke, 1970, 1972). The results of all of these studies support the contention that, with advancing years, brain damaged children of the type used in the present study may adapt to and/or recover from the deficit(s) involved in the inability to develop and maintain a state of readiness to respond. (Author)

Age Differences in Auditory Reaction Time of "Brain-Damaged" and Normal Children under Regular and Irregular Preparatory Interval Conditions

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Abstract

Groups of "brain-damaged" and normal children were subdivided into "young" (6-9 years) and "old" (10-13 years) groups and subjected to an auditory reaction-time procedure consisting of regular and irregular preparatory interval conditions. The most important results were as follows: (1) the performances of the young normal, old normal, and old brain-damaged groups were superior to that of the young brain-damaged group; (2) there was no significant difference between the performance of the old normal and the old brain-damaged groups; (3) the set-index formula was not more effective in differentiating between the performance of the young brain-damaged and young normal groups or between the young brain-damaged and old brain-damaged groups than were other, more easily derived, indices. The results of the current investigation were highly similar to those obtained in previous studies which employed visual reaction time procedures (Czudner & Rourke, 1970, 1972). The results of all of these studies support the contention that, with advancing years, brain-damaged children of the type used in the present study may adapt to and/or recover from the deficit(s) involved in the inability to develop and maintain a state of readiness to respond.

Age Differences in Auditory Reaction Time of "Brain-Damaged" and Normal Children under Regular and Irregular Preparatory Interval Conditions[1, 2]

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Introduction

The purposes of the present study were as follows: (1) to compare the performance of brain-damaged and normal children on an auditory reaction-time (RT) task; (2) to determine the relative effectiveness of a set-index formula and other indices as means of differentiating between the performance of brain-damaged and normal children; (3) to determine if, with increasing age, brain-damaged children adapt to and/or recover from the deficit(s) involved in the inability to develop and maintain a state of readiness to respond; and, (4) to determine if the results obtained in a visual RT study (Czudner & Rourke, 1972) were modality-specific.

RT measures have been used as dependent variables in the investigation of a number of theoretical and applied problems. The findings of some of these studies have direct relevance for the current investigation.

Woodrow (1914) reported one of the first systematic works on the application of RT procedures to the study of attentional processes in normal adults. He found a positive relationship between latency and duration of the preparatory interval (PI). (The PI is the interval between the onset of the warning signal and the onset of the RT stimulus.)

Huston, Shakow, and Riggs (1937) and Rodnick and Shakow (1940) employed

RT procedures to assess the ability of schizophrenic adults to develop and maintain a set (i.e., a state of readiness to respond). In connection with this work, Rodnick and Shakow (1940) developed a formula called the "set index," a mathematical expression which includes the length of the PI and the relationship between S's response to regular and irregular warning conditions. They found that it was possible to differentiate between the performance of schizophrenic and normal adults on the basis of this set-index formula.

Most studies involving RT procedures for the determination of the effects of brain damage have utilized adults with relatively acute cerebral lesions. For example, Blackburn and Benton (1955) and De Renzi and Faglioni (1965) found that simple RT can be used as a sensitive indicator of the presence of brain damage in such samples of adults. In a study which led directly to the current investigation, Czudner and Rourke (1970) found that children between the ages of 8 and 14 years who were suffering from relatively chronic cerebral dysfunction could not be differentiated on the basis of a modified version of the set-index formula. (For further explanation of the set-index formula, see Czudner and Marshall, 1967 and Czudner and Rourke, 1970.) However, when the results of the 8- to 10-year-olds were analyzed separately, the performance of only one of the brain-damaged Ss fell within the range obtained for the normal children. Following this study, Czudner and Rourke (1972) composed groups of "young" (6-9 years) and "old" (10-13 years) brain-damaged and normal children and subjected them to a visual RT procedure similar to that employed in the Czudner and Rourke (1970) investigation. The results of the Czudner and Rourke (1972) study supported the contention that, with advancing years, brain-damaged children who suffer from relatively mild, chronic cerebral dysfunction may adapt to and/or recover from the deficit(s) involved in the inability to develop and maintain a state of readiness to respond. Except for the use of auditory rather than visual stimuli and some minor modifications in procedure, the present study was essentially a replication of the Czudner and Rourke (1972) investigation.

Method

Subjects. Two groups of Ss were employed. The first group included 24 children who exhibited anamnestic, neuropsychological, and electroencephalographic evidence of cerebral dysfunction. The "neuropsychological evidence" of cerebral dysfunction meant that one of the investigators (BPR), in a "blind" interpretation of the results of a battery of neuropsychological tests similar to that suggested by Reitan (1966), judged these results to be consistent with the presence of cerebral dysfunction. This group was divided into two age classifications, "young" and "old." The age range for the young group was 7.1 to 9.0 years, with an average age of 7.7 years. The age range for the old group was 10.2 to 13.7 years, with an average age of 11.7 years. For the young group, WISC Full Scale I.Q. ranged from 82 to 109, with a mean of 94.4. For the old group, Full Scale I.Q. ranged from 83 to 115, with a mean of 96.8. The brain-damaged Ss in the present study, where it was possible to determine, had sustained damage to the brain as a result of traumatic head injuries and/or anoxia. Although the extent of damage was difficult to determine, the age of onset of the insult was, for the most part, during the perinatal period or in infancy. In general, the brain-damaged Ss were suffering from what would best be described as relatively mild, chronic cerebral dysfunction. The second (control) group was also made up of 24 children, having no detectable sensory or motor impairment (on the basis of school medical histories and teachers' observations) and matched for mean age and Full Scale I.Q. with the brain-damaged group. There were 10 boys and 2 girls in the young groups and 10 boys and 2 girls in the old groups.

Apparatus. The apparatus consisted of two Hunter Decade interval timers (Model 111C) and one Hunter Klockcounter (Model 120A). A stand was constructed with a telegraph key and two sound sources, one serving as a warning signal (high pitched sound), the other as the RT stimulus (low pitched sound). The Klockcounter was essentially an electrically-operated stop watch arranged in series with a telegraph key in such a way that S had to press the key to set the warning signal. Any release of the key before the sounding of the RT stimulus stopped the apparatus and the trial would then have to be repeated.

Procedure. Each S was escorted to the testing room by E. S was seated in front of the RT apparatus in such a way that he could manipulate the telegraph key conveniently. E situated himself in such a way that he could easily observe S and manipulate the control panel to the rear of the RT apparatus. S was instructed to press the telegraph key and to notice that this activated the high pitched sound (warning signal). Then S was told that he should lift his finger as fast as he could when the low pitched sound (RT stimulus) was heard. The high pitched sound remained on until the low pitched sound was activated. Ss were cautioned that lifting the finger from the telegraph key before the sounding of the RT stimulus would necessitate a repetition of the trial. (This occurred no more than three times for any S.) Before each trial, S was asked if he was ready to begin. If the answer was affirmative, he was instructed to depress the telegraph key and to wait for the RT stimulus before lifting his finger. Ss performed 10 practice trials before the commencement of the test trials.

Ss were exposed to two procedures. In the regular procedure, the PI (time between the two sounds) remained the same for a series of 10 trials before an interval of another length was presented. In the irregular procedure, the PIs were presented for 10 trials each, but in a random manner. The PIs used were 2, 4, and 6 secs. Hence, there were 30 trials for each procedure. There was a 30-sec rest between each change of PI in the regular procedure, a 2-min rest between the two procedures, and a 1-min rest after the first 15 trials of the irregular procedure. Total testing time was 20 minutes. Ss in each group were subdivided such that half began with the regular procedure followed by the irregular procedure, while the order was reversed for the other half.

Results

The data were analyzed by means of six analyses of variance. Two 2 x 2 x 3 analyses were carried out in order to evaluate the performance of the brain-damaged and normal groups separately. In these analyses there were two age classifications (i.e., "young" and "old"), two levels of procedure (i.e., regular and irregular), and three levels of PI (2, 4, and 6 secs). A 2 x 2 x 2 x 3 analysis of variance was carried out in order to assess differences in performance between the brain-damaged and normal children. Finally, three 2 x 2 analyses of variance were carried out, one each for the set index (SI), the highest mean (HM), and the general mean (GM) scores. The main effects in these latter analyses were age (young and old) and group (brain-damaged and normal).

Insert Table 1 about here

Table 1 contains the means and standard deviations for RT under each of the experimental conditions for the brain-damaged and normal groups. The data from Table 1 are plotted in Figures 1, 2, and 3.

Insert Figures 1, 2, and 3 about here

Inspection of Figures 1, 2, and 3 indicates that, except for the old normal group, there was a direct relationship between length of PI and RT in the regular procedure -- that is, as the length of PI increased, RT increased. In the case of the young brain-damaged group, the opposite state of affairs obtained for the irregular procedure -- that is, the shorter the PI, the longer the RT.

The analysis of variance of the brain-damaged groups' performance yielded a statistically significant ($p < .01$) PI x Procedure interaction. Testing for simple effects revealed a significant difference in performance between the regular and the irregular procedures at the 2-sec interval. A statistically significant ($p < .01$) age main effect indicated that the old brain-damaged group exhibited faster RT than did the young brain-damaged group. The significant procedure ($p < .01$) main effect indicated that brain-damaged children generally did better on the regular than on the irregular procedure. The analysis of variance of the normal groups' performance revealed trends similar to those evident in the performance of the brain-damaged groups.

Table 2 contains a summary of the analysis of variance of the young and old brain-damaged and normal groups' performance under the regular and irregular procedures at the three PIs. Inspection of Table 2 indicates the following. There was a statistically significant ($p < .01$) Age x Group interaction. Testing

Insert Table 2 about here

for the simple effects within this interaction yielded statistically significant differences between the performance of the young brain-damaged and young normal groups ($p < .01$) and between the performance of the young brain-damaged and old brain-damaged groups ($p < .01$); there were no statistically significant differences between the performance of the young normal and old normal groups or between the performance of the old brain-damaged and old normal groups. The PI x Procedure interaction was also significant ($p < .01$). An analysis of the simple effects within this interaction indicated that RT latency was faster at the 2-sec interval for the regular procedure than for the irregular procedure.

The set-index (SI) formula employed was as follows: $SI = \frac{1}{2}(M_{4R}/M_{4Ir}) + (M_{6R}/M_{6Ir})$ HM. HM is the highest mean RT obtained for each S over the six conditions. The other terms in the formula represent the average of the ratio of the 4- and 6-sec interval means of the regular (R) procedure to the respective means of the irregular (Ir) procedure. The means for the 4- and 6-sec intervals were employed in this SI formula because previous research with similar populations (Czudner & Rourke, 1970) had demonstrated that performance at these two intervals effected the clearest separation between the brain-damaged and normal groups. An inspection of Figure 4 indicates that, with the application of the SI formula, the performance of one of the young

Insert Figures 4, 5, and 6 about here

brain-damaged Ss fell within the range of performance of the young normal group. There were two such instances when the highest mean (HM) of the six conditions for each S was plotted (Figure 5). Plotting the results in terms of the general mean over all eight experimental conditions (GM) (Figure 6), two young brain-damaged Ss fell within the range of performance of the young normal group. When the performance of the young and old brain-damaged groups was compared employing the SI, HM, and GM scores, there were no overlaps in performance between the groups.

Comparisons of the performance of the old normal and old brain-damaged groups were carried out employing the SI (Figure 7), HM (Figure 8), and GM

Insert Figures 7, 8, and 9 about here

(Figure 9) scores. No clear separation between the groups was obtained on any of these measures. The 2 x 2 analyses of variance for the SI, HM, and GM scores all yielded significant ($p < .01$) age and group main effects and Age x Group interactions. The analyses of the simple effects within these interactions yielded essentially the same information conveyed in Figures 4 through 9.

Discussion

Except for the old normal group, there appeared to be a direct relationship between length of PI and RT latency in the regular procedure. This relationship did not obtain in the case of the irregular procedure. In fact, there was a rather obvious inverse relationship between PI and RT in the irregular procedure in the case of the young brain-damaged group. In the case of the regular procedure, environmental distractions together with a host of other factors may have served to render the maintenance of a state of readiness to respond more difficult as the length of the PI increased. In the case of the irregular procedure, maintenance of this state of readiness to respond was generally more difficult regardless of the length of PI, due in part to the inability of S to predict the duration of the PI.

An inspection of Figures 1, 2, and 3 yields the impression that the slope for the irregular procedure was steeper for the young brain-damaged group than for the other three groups. These findings are similar to those obtained with schizophrenic adults (e.g., Zahn, Rosenthal, & Shakow, 1963) and brain-damaged children (Czudner & Rourke, 1970, 1972).

The significant age main effect for the brain-damaged and the normal groups obtained in the separate analyses for these groups indicated an inverse relationship between age and RT latency. These findings were entirely in accord with those of earlier studies (e.g., Goodenough, 1935; Czudner & Rourke, 1972).

The significant Age x Group interaction (see Table 2) and the results of the subsequent simple effect analyses indicated that the performance of the young normal group was superior to that of the young brain-damaged group and that the performance of the old normal group did not differ significantly from that of the old brain-damaged group. The obtained difference in performance between the young normal and young brain-damaged groups was consistent with the findings of some earlier investigations (e.g., Blackburn & Benton, 1955; De Renzi & Faglioni, 1965). That the performance of the old normal group did not differ significantly from that of the old brain-damaged group in the present study was clearly inconsistent with these latter findings, but identical to the results of Czudner and Rourke (1972). This inconsistency may have been due to crucial differences in the nature, extent, and chronicity of cerebral dysfunction sustained by the brain-damaged Ss in the current study and in the Czudner and Rourke (1972) investigation as compared to that of the Ss used in the Blackburn and Benton (1955) and De Renzi and Faglioni (1965) studies.

The brain-damaged Ss in the present study and in that of Czudner and Rourke (1972), where it was possible to determine, had sustained damage to the brain as a result of traumatic head injuries and/or anoxia. Although the extent of damage was difficult to determine, the age of onset of the insult was, for the most part, during the perinatal period or in infancy. Consequently, the brain-damaged groups were composed of Ss suffering from what would best be described as relatively mild, chronic cerebral dysfunction. However, the brain-damaged Ss employed in the Blackburn and Benton (1955) and De Renzi and Faglioni (1965) studies were, for the most part, suffering from neoplasms or cerebrovascular disease -- that is, lesions much more likely to be acute than were those sustained by the Ss in the current investigation. In this connection, it is also clear that the old brain-damaged Ss in the present study and in that of Czudner and Rourke (1972)had had a much longer period of time than had the young brain-damaged Ss to adapt to and/or recover from the lesion(s) which they had sustained.

Inspection of Figures 4, 5, and 6 indicates that the SI, HM, and GM measures were effective in differentiating between the performance of the young brain-damaged and young normal groups. That both the HM and GM measures can be employed to achieve this differentiation relatively well argues against expending the added effort required to calculate the SI measure for this purpose.

The clear separation between the performance of the young brain-damaged and young normal groups and between the young brain-damaged and old brain-damaged groups, together with the lack of differentiation between the old brain-damaged and old normal groups lend support to the contention of Czudner and Rourke (1970, 1972) that brain-damaged children of the type used in this study may adapt to and/or recover from the deficit(s) involved in the inability to develop and maintain a state of readiness to respond. That the results for both visual RT (Czudner & Rourke, 1972) and auditory RT (present study) procedures were virtually identical indicates that the obtained differences are not modality-specific. It would appear that the neural mechanisms responsible for these patterns of results are clearly central rather than peripheral.

If the deficit in RT latency exhibited by young brain-damaged Ss is as reliable as these results would seem to indicate, visual and/or auditory RT latency may serve as a useful dependent variable for the assessment of the effects of various drugs (e.g., methylphenidate, dexedrine) and/or behavioral procedures which are designed to "increase attention" in children of this age group.

At least two avenues of research related to the current study should be pursued in order to elucidate more fully the obtained differences and relationships. Since the results of the current study, taken together with those of Czudner and Rourke (1972), seem to indicate that the neural mechanisms responsible for the obtained patterns of results are central rather than peripheral, the relationship between RT latency under these conditions and a variety of measures of attention should be determined. Secondly, it may be of interest to compare groups of young and old brain-damaged children and adults with lesions differing in terms of nature, extent, level of chronicity, lateralization, and age of onset in order to explain some of the apparent inconsistencies in the results obtained in RT studies with samples of brain-damaged Ss.

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Footnotes

1. This study was presented at the meeting of the Society for Research in Child Development, Minneapolis, April, 1971.

2. This study was assisted under Grant #195 of the Ontario Mental Health Foundation. Funds were also provided by the Research Division, I.O.D.E. Hospitals, Windsor.

TABLE 1

Reaction Time Means and Standard Deviations (in msecs.) for the Four Groups under Regular and Irregular Preparatory Interval Conditions

Interval (sec.)	Regular Procedure		Irregular Procedure	
	M	SD	M	SD
	Young Brain-Damaged Group			
2	480.6	139.2	666.2	138.6
4	535.7	133.0	573.8	166.7
6	547.7	125.2	528.9	131.3
	Old Brain-Damaged Group			
2	250.5	68.5	322.3	48.1
4	259.5	72.9	271.8	57.6
6	274.8	52.7	263.7	46.9
	Young Normal Group			
2	295.4	52.7	382.6	63.8
4	332.8	64.1	375.6	74.9
6	346.1	57.3	368.6	82.7
	Old Normal Group			
2	227.1	45.5	315.1	52.7
4	247.9	44.7	293.7	66.0
6	259.9	52.4	284.4	62.3

TABLE 2

Analysis of Variance of Young and Old Brain-Damaged and Normal Groups' Performance under Regular and Irregular Preparatory Interval Conditions

Source	df	MS	F
Between Ss	47		
Age	1	2337374.2	66.9*
Group	1	776320.3	22.2*
Age x Group	1	742228.9	21.2*
S within group error	44	34937.1	
Within Ss	240		
PI	2	1690.9	0.5
Age x PI	2	658.9	0.2
PI x Group	2	8490.4	2.6
Age x Group x PI	2	1919.4	0.6
PI x Ss within error	88	3243.7	
Procedure (Pr)	1	173352.5	44.3*
Age x Pr	1	7835.4	2.0
Pr x Group	1	530.8	0.1
Pr x Group x Age	1	9616.5	2.5
Pr x Ss within error	44	3912.5	
PI x Pr	2	68111.3	29.1*
PI x Pr x Age	2	6051.9	2.6
PI x Pr x Group	2	10434.9	4.5
Age x PI x Pr x Group	2	5785.7	2.4
PI x Pr x Ss within error	88	2341.9	

* $p < .01$

Figure Captions

Fig. 1 Mean RT of the young brain-damaged and old brain-damaged groups for the three PIs of the regular and irregular procedures.

Fig. 2 Mean RT of the young brain-damaged and young normal groups for the three PIs of the regular and irregular procedures.

Fig. 3 Mean RT of the old brain-damaged and old normal groups for the three PIs of the regular and irregular procedures.

Fig. 4 Distribution of young brain-damaged and young normal Ss according to the set-index formula.

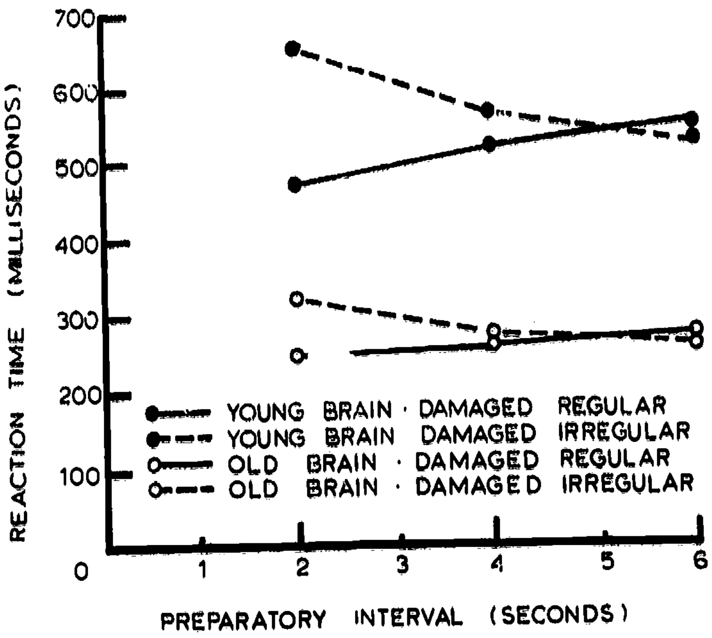
Fig. 5 Distribution of young brain-damaged and young normal Ss according to the highest mean (values are in msecs).

Fig. 6 Distribution of young brain-damaged and young normal Ss according to the general mean (values are in msecs).

Fig. 7 Distribution of old brain-damaged and old normal Ss according to the set-index formula.

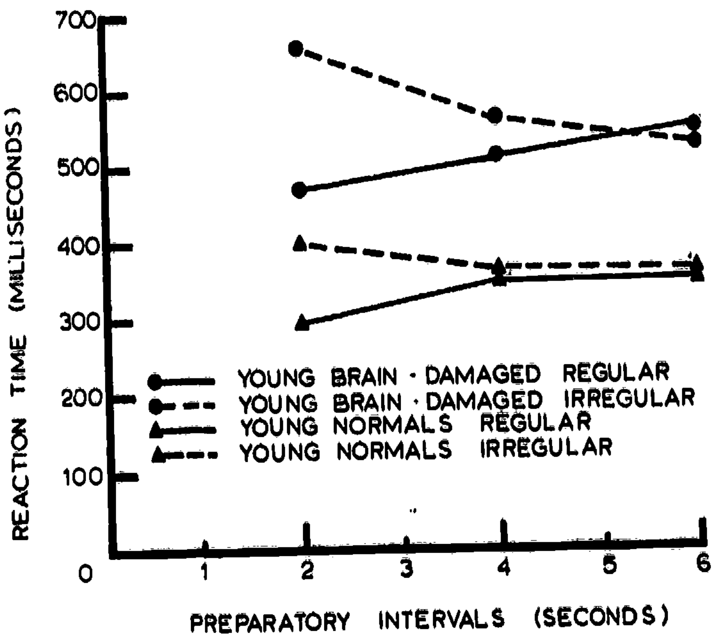
Fig. 8 Distribution of old brain-damaged and old normal Ss according to the highest mean (values are in msecs).

Fig. 9 Distribution of old brain-damaged and old normal Ss according to the general mean (values are in msecs).



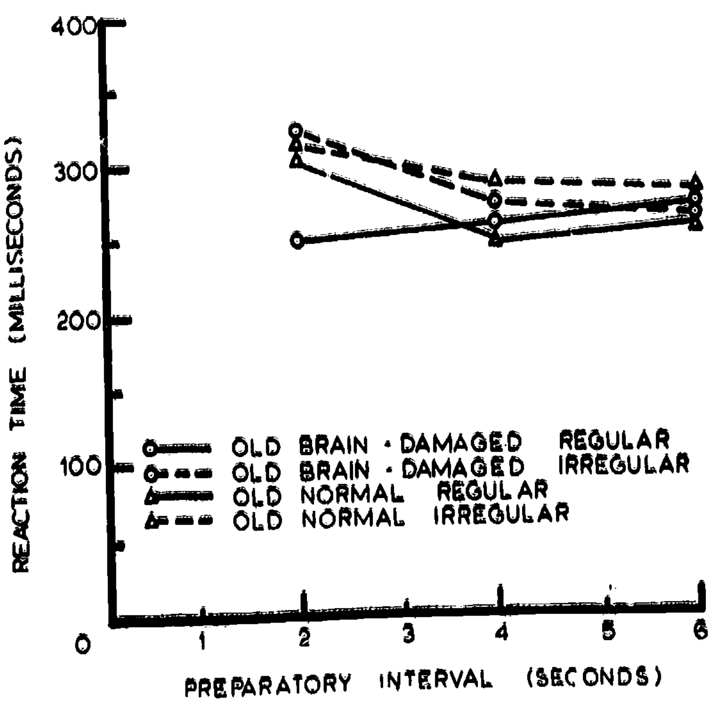
700
600
500
400
300
200
100
0
REACTION TIME (MILLISECONDS)
YOUNG BRAIN · DAMAGED REGULAR
YOUNG BRAIN DAMAGED IRREGULAR
OLD BRAIN · DAMAGED REGULAR
OLD BRAIN · DAMAGED IRREGULAR
1 2 3 4 5 6
PREPARATORY INTERVAL (SECONDS)

1.



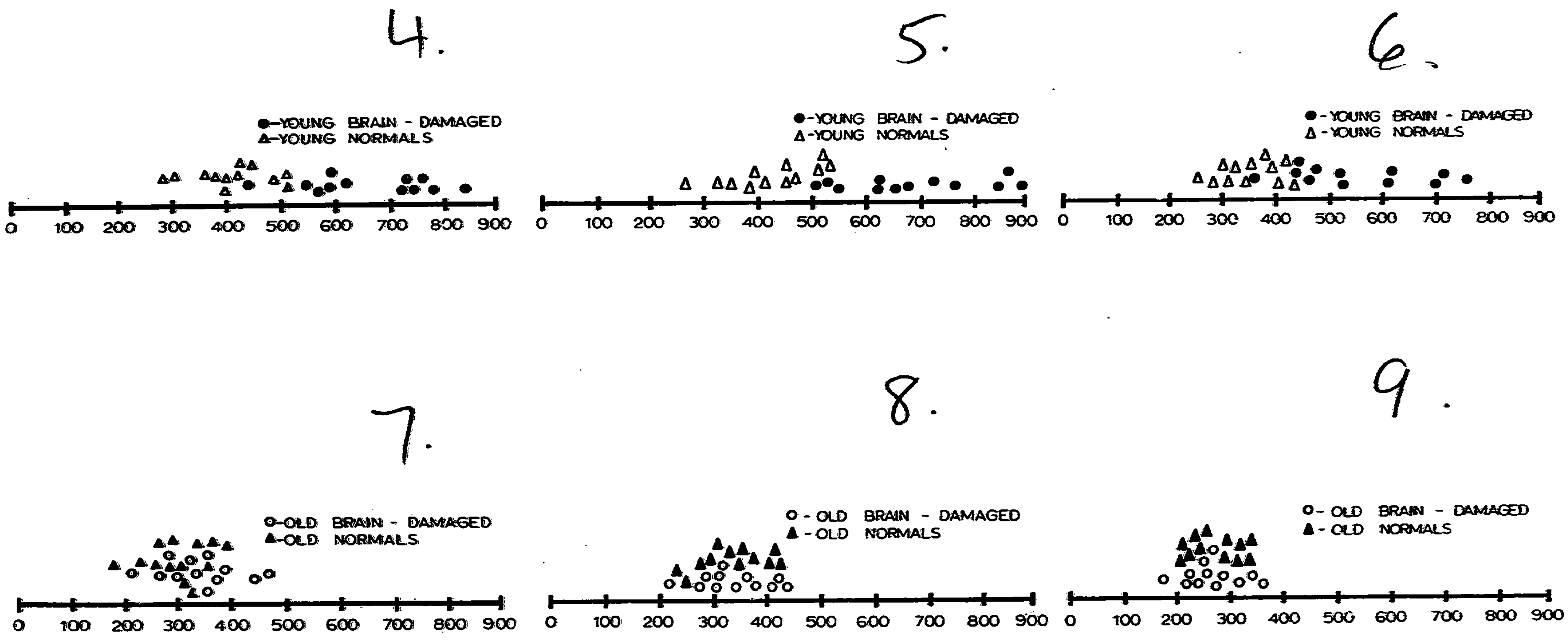
700
600
500
400
300
200
100
0
REACTION TIME (MILLISECONDS)
YOUNG BRAIN · DAMAGED REGULAR
YOUNG BRAIN · DAMAGED IRREGULAR
YOUNG NORMALS REGULAR
YOUNG NORMALS IRREGULAR
1 2 3 4 5 6
PREPARATORY INTERVALS (SECONDS)

2.



400
300
200
100
0
REACTION TIME (MILLISECONDS)
OLD BRAIN · DAMAGED REGULAR
OLD BRAIN · DAMAGED IRREGULAR
OLD NORMAL REGULAR
OLD NORMAL IRREGULAR
1 2 3 4 5 6
PREPARATORY INTERVAL (SECONDS)

3.


4.
●-YOUNG BRAIN - DAMAGED
Δ-YOUNG NORMALS
0 100 200 300 400 500 600 700 800 900
5.
●-YOUNG BRAIN - DAMAGED
Δ-YOUNG NORMALS
0 100 200 300 400 500 600 700 800 900
6.
●-YOUNG BRAIN - DAMAGED
Δ-YOUNG NORMALS
0 100 200 300 400 500 600 700 800 900
7.
O-OLD BRAIN - DAMAGED
▲-OLD NORMALS
0 100 200 300 400 500 600 700 800 900
8.
O-OLD BRAIN - DAMAGED
▲-OLD NORMALS
0 100 200 300 400 500 600 700 800 900
9.
O-OLD BRAIN - DAMAGED
▲-OLD NORMALS
0 100 200 300 400 500 600 700 800 900