Coding of stimulus parameters in autistic, retarded, and normal children: evidence for a two-factor theory of autism

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INTRODUCTION

The hypothesis that perceptual and physiological abnormalities in sensory integration and selective attention underly the observed behavioral deficits of autistic children has received increasing attention in recent years. Despite this, a literature survey conducted by James and Barry (1980) suggested that the psychophysiological underpinnings of perceptual orientation in such children had received scant empirical attention. In particular, it was noted that few studies had undertaken a systematic investigation of a range of phasic response measures as a function of variations in stimulus parameters. However, sufficient evidence was available to suggest that the striking behavioral patterns found in autism might be associated with abnormal autonomic activity.

In response to the paucity of reliable data in this area, a number of studies have been undertaken in this laboratory to systematically investigate the psychophysiological reactivity of autistic children. James and Barry (1980b) investigated several phasic response measures using visual stimuli in a simple habituation paradigm. We reported that the autistic group displayed a marked impairment in the habituation of respiratory pause when compared with matched retarded and normal children. In addition to this specific deficit in processing reductions in stimulus novelty, the autistic group also displayed enhanced response magnitudes in the peripheral and cephalic vasculature. A second study of response magnitudes in normal preschoolers and university students showed significant age effects in peripheral and cephalic pulse amplitude responses, suggesting that
the overresponding of the autistic group could be attributed to developmental delay.

The data base and conclusions derived from this study were extended further by James and Barry (1984). Phasic changes in respiratory period, electrodermal activity (GSR), the evoked cardiac response (ECR), and the vasoconstrictive peripheral pulse amplitude response (PPAR), were examined in a much larger \( (n = 40) \) sample of autistic children of a wide age range, with matched control groups of retarded and normal children. Prior work was extended from the visual to the auditory modality and results were also analysed as a function of chronological age (CA) in order to more closely examine previous evidence of developmental delay.

Results of this study clearly indicated marked differences in the phasic responsiveness of autistic children. The specific deficit in the processing of stimulus novelty was confirmed in both the respiratory pause and GSR measures. The autistic group also displayed enhanced response magnitudes in the non-habituating measures, despite age-matching with the control groups, and did not display the diminution of response level with increased age that was characteristic of both the control groups. These effects were obtained across both modalities. In general, this study indicated that the findings previously reported by James and Barry (1980b) could be replicated across modalities with a significantly larger sample size and an increased range of measures.

On the basis of these results, James and Barry (1980b, 1984) have suggested a two-factor theory of the abnormal psychophysiological reactivity of autistic children. Firstly, there appears to be a specific qualitative deficit in the ability to process reductions in stimulus novelty, as indicated by the significant impairment of habituation rates. Secondly, there is strong evidence of a general quantitative developmental delay, manifested as an overall hyperresponsivity and delay in the maturational reduction of response amplitude. Together, these impairments could be expected to contribute importantly to the cognitive, language, and emotional problems of autistic children.

Other studies from this laboratory have separately examined each of these two deficits. James and Barry (1980a) hypothesised that the specific deficit related to habituation rates, and the lack of affective lability that is recognised as a cardinal symptom of autism, might both result from impaired functioning of the brainstem reticular activating system. This hypothetical linkage between failure to habituate normal stimuli and flattening of affect was tested in the normal population by examining habituation rates in groups differing on a personality measure of affect lability, the P-scale of the Eysenck Personality Questionnaire. Two experiments were reported which indicated that low affect-lability subjects demonstrated significantly slower habituation of respiratory pause and GSR than high affect-lability subjects. These results were interpreted as supporting the hypothesis that rate of habituation of autonomic response measures to simple stimuli may be related to personality differences in affect lability, with differences in both variables being determined by brainstem differences. Further work by Barry (1980) supported the relationship between the personality measure used, Eysenck and Eysenck's P-scale, and the processing of affective stimuli.

The notion of developmental delay in autistic children was discussed in detail by James and Barry (1981). We concluded that evidence from clinical descriptions, experimental learning and cognitive performance studies, and other literature relating to maturational factors in autism, supported the suggestion that disturbances in developmental rates and sequences may be an essential primary feature of the disorder. Suggestions from the psychophysiological studies outlined above, that autistic children suffer from a generalised developmental delay in those mechanisms responsible for the regulation of phasic response amplitudes in the central nervous system (CNS), led to further experimental studies of developmental delay at the CNS level.

Barry and James (1978), in a study of the development of handedness in autistic children, reported a developmental lag in the establishment of manual preference that distinguished these children from mentally retarded and normal control groups. This suggestion of a delay in the establishment of cerebral lateralisation was further
investigated by James and Barry (1983). Using magnitude of dominant ear advantage as an indicator of relative cerebral dominance, unwarned simple reaction time (RT) to monaural presentation of tones was investigated in matched groups of autistic, retarded, and normal children. Results indicated that autistic children showed significant developmental delay in both RT and the establishment of cerebral dominance, compared to the control groups. Since the development of cerebral dominance is allied to maturity of the CNS, this study reinforces previous evidence from our psychophysiological studies that a primary immaturity in CNS organisation is an essential feature of the profile of autistic children.

The series of studies from this laboratory, outlined above, provides strong support for the postulated two-factor deficit in autism, viz. a general developmental delay in CNS maturity and a specific brainstem deficit reflected in an inability to adequately process reductions in stimulus novelty. Evidence from other laboratories providing support for this two-factor deficit theory has been extensively outlined in our previous reports, and need not be duplicated here. However, since this deficit pattern was first proposed by James and Barry (1980b, 1984), some supporting evidence has appeared in the literature, although differences in methodologies preclude definite comparisons.

Kootz and Cohen (1981) and Kootz et al. (1982) examined cardiovascular correlates of attention in autistic children during resting, social interaction and RT tasks. They reported a pattern of increased peripheral blood flow, decreased peripheral vascular response and elevated HR, that was interpreted as indicating a heightened state of sensory rejection in autism. However, it is difficult to assess whether such a cardiovascular response pattern is indeed indicative of autism per se or is merely a function of maturational delay, since neither study incorporated adequate control groups. (A detailed discussion of necessary control groups in autistic research has been presented by James and Barry, 1981.) Indeed, Kootz et al. (1982) reported no control groups at all, thus making any conclusions about their data extremely difficult. Kootz and Cohen (1981) included an unmatched normal control group, but failed to include any controls for I.Q. or mental age (MA) factors. The reported finding of a significantly elevated tonic HR level, therefore, need not, as the authors contend, be interpreted as suggesting that autistic children are in a state of heightened autonomic nervous system arousal. Rather, it can be taken as another indication of an overall developmental lag in the establishment of normal psychophysiology.

Van Engeland (1984), in an examination of electrodermal orienting responses in higher functioning autistic children (mean I.Q. 71), concluded that their electrodermal response pattern was similar to that derived from schizophrenic samples. He reported a significant number of non-responders in the autistic group compared to unmatched normal, psychiatric and mentally retarded control groups. The electrodermal pattern of those autistic children who did respond was characterised by large amplitudes and fast recovery. He also reported no differences between groups in the number of spontaneous fluctuations, suggesting that arousal levels did not differ between autistic and other children, in agreement with our previous findings. These results were interpreted as providing support for the notion that autistic children are unusually sensitive to stimuli and prone to sensory overload.

This 'schizophrenic' pattern of electrodermal responding in autistic children was not confirmed by Stevens and Gruzelier (1984). They were unable to isolate any features of electrodermal activity that distinguished autistic children from matched retarded and normal control groups. There was only some suggestion of slower habituation rates and marginally higher levels of skin conductance that increased over time, interpreted as a tendency towards over-arousal. Analysis of bilateral response patterns indicated a higher incidence of autistic children with larger left- than right-hand responses. This was interpreted as indicative of a narrowed attention mode characterised by left hemisphere dominance. Such findings are readily accommodated in our two-factor theory as indicators of developmental delay (Barry and James, 1978).

In general, there still appears to be a scarcity of
reliable data relating to psychophysiological response patterning in autism. There has been a consistent failure in most studies to incorporate adequate control groups, and there has been no effort to establish a firm data base by replicating studies, using identical experimental procedures and stimulus characteristics.

Working within the framework developed in this laboratory, the present study was designed as a further replication and extension of previous studies. In particular, indications in previous work that autistic children displayed a deficit in the processing of stimulus novelty suggested that other stimulus parameters should also be investigated. Thus this study was aimed at investigating the relationship between variations in stimulus magnitude and phasic responsiveness, as well as a continuing examination of the effects of stimulus novelty and chronological age. A variety of physiological indicators was thus selected within the context of Barry’s recent theory of preliminary processes in orienting response (OR) elicitation (Barry, 1984). Reports from our laboratory, in supporting this theory, have consistently reported that respiratory pause reflects the processing of stimulus novelty, that the PPAR is an absolute indicator of stimulus magnitude, and that the GSR is an OR indicator reflecting both stimulus novelty and magnitude.

Response magnitudes of these measures were investigated as a function of stimulus novelty, modality, and magnitude, CA, and group membership. On the basis of previous results from this laboratory, the following specific predictions can be made concerning the behavior of each phasic response indicator. Since respiratory pause is sensitive to stimulus novelty only, habituation will occur in the control groups, but habituation rates will be significantly reduced for the autistic group because of their hypothesised brainstem impairment. PPAR will reflect stimulus magnitude variations, but not novelty, in the control groups; the behavior of this measure in autistic children is unknown. The GSR will reflect both stimulus magnitude and novelty in the control groups, with impaired habituation and unknown magnitude effects in the autistic group. Finally, significant age effects are expected in these measures for the control groups, with evidence of developmental delay in the autistic group. For the present purposes, ‘habituation’ is operationally defined as response decrement over trials, following previous usage in this laboratory.

METHODS

Subjects
Thirty-two autistic children (26 males and 6 females), ranging in age from 4 years 9 months to 17 years 2 months, formed the autistic group. Each child was a non-residential day pupil enrolled at the Vern-Barnett Diagnostic Teaching Centre (Autistic Children’s Association of N.S.W.), and its three area schools in the Sydney metropolitan district. Each was previously diagnosed and currently treated as autistic, and, for the purpose of inclusion in this study, satisfied all five of the diagnostic criteria established by Ritvo and Freeman (1978):

1. Signs and symptoms present prior to 30 months of age.
2. Disturbances of developmental rate and/or sequence.
3. Disturbance of responsiveness to sensory stimuli.
4. Disturbances of speech, language, and cognitive capacities.
5. Disturbances of relating to people, events, and objects.

This group was divided into two subgroups on the basis of CA. The ‘young’ group consisted of 16 subjects aged from 4.9 years to 9.11 years, while the remaining 16 subjects formed the ‘old’ group, aged from 12.0 years to 17.2 years. The distribution of ages and IQs for the sample is indicated in Table I. (IQs used in this study were supplied by the participating schools and were based on at least two independent assessments of each child undertaken within the preceding year.)

A mentally retarded control group was formed from day pupils attending a special school for mentally handicapped children administered by the N.S.W. Department of Education. This group matched the autistic sample child-by-child on sex, CA, and I.Q. Comparison of these two groups
TABLE I
Age and I.Q. distribution for the autistic group

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<tr>
<td></td>
<td>I.Q.</td>
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<tr>
<td>Intervals (years and months)</td>
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<tr>
<td>'Young'</td>
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<tr>
<td>4.0–4.11</td>
<td>1</td>
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<tr>
<td>5.0–5.11</td>
<td>4</td>
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<tr>
<td>6.0–6.11</td>
<td>0</td>
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<td>7.0–7.11</td>
<td>2</td>
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<tr>
<td>8.0–8.11</td>
<td>4</td>
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<tr>
<td>9.0–9.11</td>
<td>1</td>
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<tr>
<td>'Old'</td>
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<tr>
<td>10.0–10.11</td>
<td>0</td>
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<tr>
<td>11.0–11.11</td>
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<td>12.0–12.11</td>
<td>6</td>
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<tr>
<td>13.0–13.11</td>
<td>1</td>
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<td>14.0–14.11</td>
<td>2</td>
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<td>15.0–15.11</td>
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<tr>
<td>16.0–16.11</td>
<td>2</td>
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<tr>
<td>17.0–17.11</td>
<td>1</td>
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<tr>
<td>Level of retardation (WHO, 1977)</td>
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<tr>
<td>Near normal</td>
<td>84–90</td>
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<tr>
<td>Borderline</td>
<td>71–83</td>
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<tr>
<td>Mild</td>
<td>50–70</td>
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<tr>
<td>Moderate</td>
<td>35–49</td>
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<tr>
<td>Severe</td>
<td>21–34</td>
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yielded correlation coefficients exceeding 0.99 for CA and 0.95 for I.Q. A normal control group was formed from children in the normal school population, also matched with the autistic children child-by-child on sex and CA. Analysis yielded correlation coefficients for CA exceeding 0.99 between this and the other groups. These groups thus effectively controlled for intellectual and maturational variables in the manner proposed by James and Barry (1981). No child in any group was in receipt of medication immediately prior to, or during, the period of testing.

Apparatus and procedure
Subjects were tested using a within-subjects repeated presentation of simple visual and auditory stimuli. Visual stimuli consisted of 2 large (size 9 × 9 cm) and 5 small (4.5 × 4.5 cm) white squares (luminance 137 cd/m²) on a black background (luminance 0.60 cd/m²), and were presented in an alternating order by an Exidy microcomputer on a small (22.5 × 17.5 cm) black and white video monitor. Auditory stimuli were 2400 Hz tones at 50 and 33 dB SPL re 20 μN/m², presented by the microcomputer in an alternating sequence, via circumaural headphones. These innocuous low to moderately-intense stimuli were chosen to be compatible with previous OR work from this laboratory. During the auditory half of the session, a visual pattern of high complexity was continually present on the screen, as detailed by James and Barry (1984). Stimulus durations were 2.0 s, with ISI randomly varying among 30, 40, and 50 s. Within each subgroup, the initial stimulus was counterbalanced over magnitude and modality. The entire experimental procedure lasted approximately 30 min.

Subjects sat approximately 70 cm from the monitor, and testing was carried out in quiet darkened rooms on their respective school premises. Each autistic child had previously undergone a lengthy familiarisation period with both the experimental setting and with the experimenter. Each child was encouraged to maintain attention on the video monitor to reduce gross body movements. AC-coupled skin resistance changes * were recorded using 10 μA from a StoeIting GSR preamplifier with a time constant of 4 s. Beckman-type Ag/AgCl electrodes were attached to the volar surfaces of the medial phalanges of the second and third digits of the subject’s right hand. Commercially-available felt corn pads limited the contact area to 0.71 cm², and 0.05 M KCl in an inert viscous ointment base was used as electrolyte (following James and Barry, 1984). Respiration was recorded from a StoeIting bellows pneumograph attached around the subject’s chest. A Narco Bio-systems photo-electric pulse pickup was attached to the distal volar surface of the fourth digit of the subject’s right hand, which rested on the knee of an experimental assistant. (This was necessitated by the inability of our autistic subjects to refrain from hand move-

* Previous work by Barry (1981) established the comparability of such measures with skin conductance response measures within an OR context.
ments without some minimal physical restraint. The procedure was generalised to the control groups for experimental uniformity.) These physiological measures were recorded on 3 channels of a Stoelting Multiscribe at a paper speed of 3.0 mm/s. Stimulus events were recorded on a fourth channel.

RESULTS

All physiological measures were manually scored from the protocols. Approximately 5% of responses from the autistic protocols were considered to be unreadable or doubtful, due to movement artifacts. These were replaced by averages of responses to the preceding and following stimuli of the same type. (This procedure was necessary because the ANOVA program employed contained no provision for missing data. The averaging process was chosen because it would not affect the presence or absence of habituation effects.) This procedure did not apply to the retarded and normal groups, where all protocols were sufficiently readable. Data were analysed with a 5-way planned-comparisons ANOVA over group membership, age, modality, stimulus magnitude, and trials, with repeated measures on the last three variables. Within trials, only linear and quadratic trends were analysed. Specific planned contrasts between groups allowed comparisons of autistic with retarded, autistic with normal, and ‘young’ with ‘old’ children. In order to increase the value of positive findings reported here, in the context of the theory testing involved, a significance level of $P < 0.01$ was chosen. The overall method of analysis followed the multivariate approach of Poor (1973), involving fewer assumptions about homogeneity of variances than the traditional repeated measures ANOVA procedure.

Respiratory pause

The respiratory response was defined as the percentage increase in the period of the respiratory cycle containing the stimulus onset, compared with the immediately prestimulus cycle (following Barry, 1984). These values were subjected to the 5-way ANOVA discussed above.

Fig. 1. Respiratory pause, averaged over stimulus magnitude, as a function of trials. The left panel shows data from auditory stimuli, and visual data is shown in the right panel. Results are shown separately in this and other figures for autistic (A), retarded (R), and normal (N) groups. The failure of the autistic children to show habituation is readily apparent.

Fig. 1 shows the mean response magnitude, averaged across stimulus magnitude, as a function of trials. Data are shown separately for each group, and for stimuli of each modality. There was a strong overall habituation effect represented by both linear, $F_{1,90} = 924.70$, $MS_e = 28.79$, and quadratic, $F_{1,90} = 137.69$, $MS_e = 18.78$, trends over trials. The linear trend differed as a function of stimulus modality, $F_{1,90} = 7.13$, $MS_e = 12.91$. Fig. 1 suggests that this was due to a greater initial response magnitude associated with the auditory stimuli, coupled with similar habituated response levels to stimuli of both modalities. The autistic group showed little evidence of habituation, differing significantly from the retarded and normal groups on both linear ($F_{1,90} = 328.35$ and 339.09 respectively) and quadratic ($F_{1,90} = 67.56$ and 32.62 respectively) trends over trials, as shown in Fig. 1. The autistic children displayed significantly larger mean respiratory responses than the retarded children, $F_{1,90} = 487.14$ and 484.10, respectively, $MS_e = 92.40$. Although this is mainly attributable to the marked difference in trial effects between the groups, a separate analysis over the first presentation of each stimulus type indicated that substantial differences existed even in the initial responses. For autistic children compared with retarded children, the difference approached significance ($F_{1,90} = 5.55$, $P < 0.025$), whereas the autistic children were significantly more responsive than the normal children, $F_{1,90} = 8.56$, $MS_e = 48.69$. 
As shown in Fig. 2, there were no significant main effects of modality, or stimulus magnitude. However, the modality X stimulus magnitude effect differed between autistic and normal children, \( F_{1,90} = 9.01, \text{MS}_e = 18.05 \). Fig. 2 suggests that, while the normal group shows a direct stimulus magnitude effect with auditory stimuli, and an inverse effect with visual stimuli, the autistic group shows opposite stimulus magnitude effects in each modality. Since these effects producing the interaction are small, and this interaction is of no apparent theoretical significance, it will not be discussed further. Although there appears, from Fig. 2, to be a slight reduction in response magnitude with increasing age in the normal and retarded groups, and a contrasting increase in response magnitude with increasing age in the autistic children, none of these effects or interactions approached statistical significance. However, the separate analysis carried out over the responses to the first stimulus presentations, discussed above, suggested that habituation might have masked these effects: the first-trial age x group interactions approached significance, \( F_{1,90} = 3.48, P < 0.07 \), and \( F = 4.10, P < 0.05 \), respectively. These effects are shown by the dashed lines in Fig. 2 (right panel).

**PPAR**

The PPAR was scored as the percentage decrease in pulse amplitude from the prestimulus pulse to the smallest pulse occurring between beats 7 and 12 poststimulus, following previous convention in this laboratory. These values were submitted to the 5-way ANOVA described above. The autistic group displayed significantly larger response magnitudes than the retarded and normal groups (\( F_{1,90} = 82.58 \) and 104.68 respectively, \( \text{MS}_e = 75.72 \)), for stimuli of both modalities, as shown in Fig. 3. It can also be noted from Fig. 3 that there were no significant trial effects in either modality.

Fig. 4 illustrates the main effect of stimulus magnitude, which was statistically significant, \( F_{1,90} = 104.92, \text{MS}_e = 42.91 \). As is also apparent from Fig. 4, this effect did not differ between the
Fig. 5. Electrodermal responses as a function of trials. Again, the failure of the autistic children to demonstrate response habituation is clearly demonstrated, as is their hyperresponsivity.

Fig. 6. Left panel shows stimulus magnitude effects in the GSR, again indicating that all groups code stimulus magnitude similarly. The right panel displays the GSR as a function of trials for different stimulus magnitudes (S, L), illustrating the significant interaction between the quadratic trend over trials and stimulus magnitude.

There was a significant main effect of age, $F_{1,90} = 53.94$, with the younger group exhibiting greater responsiveness, as shown in Fig. 4. This age effect differed between the autistic and normal groups, $F_{1,90} = 7.01$, and between the autistic and retarded groups, $F_{1,90} = 11.41$. Fig. 4 indicates that these differences were due to a markedly reduced age effect in the autistic children compared with the control groups. No other main effects or interactions approached significance.

$GSR.$

The skin resistance response, defined as any measurable drop in resistance with latency between 1 and 5 s following stimulus onset, was used as a measure of the GSR. Due to the marked skewness in distribution of scores, values were subjected to a square root transformation prior to analysis, following James and Barry (1984). All values (including ‘zero’ responses) were submitted to the repeated-measures ANOVA outlined above. A marked habituation effect was indicated by both linear ($F_{1,90} = 656.14$, $MS_e = 224.91$) and quadratic ($F_{1,90} = 221.37$, $MS_e = 162.54$) trends over trials. The linear trend over trials differed as a function of stimulus modality, $F_{1,90} = 12.12$, $MS_e = 111.11$. Fig. 5 suggests that this reflects a greater initial response magnitude elicited by auditory stimuli compared with visual stimuli, coupled with similar habituated response levels. The autistic group differed significantly from the retarded and normal groups on both linear ($F_{1,90} = 180.20$ and 172.89 respectively) and quadratic ($F_{1,90} = 33.08$ and 50.15 respectively) trends, as illustrated in Fig. 5. There was a significant difference in mean response magnitudes between the autistic and retarded groups ($F_{1,90} = 277.44$, $MS_e = 2578.82$), and between the autistic and normal groups ($F_{1,90} = 311.19$), with the autistic children displaying larger response magnitudes. This is generally attributable to the marked difference in trial effects between the groups, although a separate analysis over the first response to each stimulus type confirmed that the apparent group differences were significant, $F_{1,90} = 53.18$ and 56.37 respectively, $MS_e = 872.04$.

There was a significant stimulus magnitude effect ($F_{1,90} = 75.80$, $MS_e = 424.61$) which did not differ between the groups. This is shown in Fig. 6. A significant interaction between quadratic trend over trials and stimulus magnitude, $F_{1,90} = 10.69$, $MS_e = 69.29$, is also illustrated in Fig. 6. This effect appears to arise from a slightly faster ‘bottoming-out’ of responses with repetition of the higher-magnitude stimuli, although the response magnitudes continue to reflect stimulus magnitudes. No other main effect or interaction reached significance.

$Tonic arousal level$

Of the measures employed in this study, only respiratory rate lent itself to employment as an index of tonic arousal level. For each subject, the
The duration of 10 respiratory cycles immediately prior to the first stimulus presentation was measured in mm. Analysis as a function of group, with the planned group contrasts employed throughout this study, indicated that only the age factor approached significance \( F_{1.90} = 4.13, P < 0.05, \text{MS}_e = 9.38 \). Conversion of the duration measures to rates indicated that respiratory rates decreased from 25.3 to 23.2 cycles/min as the age of the groups increased.

DISCUSSION

This study was designed to allow an examination of the effects of stimulus novelty and magnitude, within the visual and auditory modalities, upon a range of physiological measures. By selecting the particular measures within an OR context provided by preliminary process theory, a clear set of predictions about normal functioning and development was available. Examination of these predictions in older and younger autistic children, and in tightly-matched control groups, was expected to illuminate the nature of stimulus coding processes in this syndrome, and to provide evidence relevant to a two-factor theory of autism.

The reduction of stimulus novelty, as a stimulus is repeatedly presented, is normally reflected in response habituation in certain measures. Significant habituation was noted here in both respiratory pause and electrodermal responses, as expected from previous work on preliminary process theory. As can be seen from Figs. 1 and 5, there was very rapid habituation apparent within the 5 presentations of each stimulus type, at least for the control groups. These figures also indicate how the autistic children differed significantly from both the normal and retarded control groups in their rate of apparent habituation of both these measures. It appears that the autistic children showed little evidence of any reduction in response magnitude over trials, suggesting an inability to adequately process reductions in stimulus novelty. This replicates previous findings from this laboratory, confirming the occurrence of one of the deficits hypothesised in the two-factor theory of autism.

Based on previous work in this laboratory, stimulus magnitude effects were expected in PPAR and the GSR. Such effects are apparent in Figs. 4 and 6, and were found to be statistically significant. These figures also indicate that all three groups of children processed this dimension of the stimulus in a similar fashion, with no significant interaction between the magnitude effect and group. This demonstrates the selective nature of the preceding finding of an impairment in the novelty-processing ability of autistic children. Clearly, that novelty-processing deficiency is not merely symptomatic of a global stimulus-encoding problem. Rather, we have demonstrated an inability of autistic children to adequately code one stimulus dimension (novelty) in a context where they clearly exhibit the ability to correctly code another stimulus dimension (magnitude). These findings apply to stimuli of both the auditory and visual modalities.

The third independent variable focused upon in this study, the effect of subject age, relates to the second deficit postulated in the two-factor theory of autism: developmental delay. Previous work in our laboratory had established that response magnitudes generally decreased with increasing age in normal children. Further, autistic children had been found to be hyperreactive in comparison with normal and retarded control groups of matched ages. In the present study, autistic hyperresponsiveness was apparent in all measures. With the habituating measures, at least part of this effect is attributable to the failure of the autistic children to show significant response reduction over trials, but separate analyses suggested that such hyperresponsiveness occurs even at the first stimulus presentation. With the non-habituating PPAR, there is no doubt of the existence of this effect over all stimulus presentations. While these data support the hypothesised hyperresponsivity of the autistic children, the developmental origin of this characteristic must also be demonstrated. Relevant information comes from a consideration of the age effects obtained here. In the main analyses, significant age effects were obtained only in PPAR. The response decrement with increasing age noted with this measure (see Fig. 4) supports the interpretation of hyperre-
activity as a correlate of developmental delay. Further, the reduced age effect apparent in the autistic group suggests continuing developmental problems for these children: there is evidence not only of developmental delay, but also of a continuing slowness of development. We have assumed in previous reports that these effects are not apparent in the other measures used here because of the floor effects associated with the habituating measures. Some evidence for this assumption was obtained by analysis of the initial responses as a function of group. With respiratory pause, the interactions between age and group, shown in Fig. 2 by the dashed lines, approached significance. However, such evidence was not found with the GSR, and this point is in need of further investigation.

These two group differences were obtained without evidence of arousal differences, since respiration rates reflected only developmental slowing. Our previous studies have also obtained such phasic response differences in the absence of parallel differences in tonic cardiac arousal levels, arguing against an interpretation of our findings in simple arousal terms.

It is important to consider why the two aspects of atypical autonomic functioning in autistic children, reported from this laboratory in a number of studies, have not been as clearly apparent in reports from other centres. After observations of children and discussion with colleagues in Britain and the U.S., we have concluded that the basis for these discrepant observations is the severity of the autistic disorder in our subjects. We believe that our subjects are more severely handicapped than those generally used in similar studies elsewhere. This is supported by the fact that, while we had to physically restrain the hand movements of our subjects in order to obtain reasonably artifact-free records (see Methods), other researchers have been able to achieve the same result through instructions (e.g. Stevens and Gruzelier, 1984). The absence of less-severely handicapped children from our Autistic Centres may reflect aspects of government funding policies in N.S.W. Children officially recognised as ‘autistic’ here are entitled to higher levels of educational support than are provided for the bulk of other handicapped children, and this may have resulted in an official bias against recognising less-severely handicapped children as autistic. It appears reasonable to propose that the magnitudes of the deficits we report would correlate with the severity of a child’s autistic handicap. This proposition deserves empirical investigation.

This study has confirmed, in a severely autistic sample, the existence of the two psychophysiological anomalies previously associated with this syndrome. That is, evidence has been provided that such children fail to adequately process stimulus novelty, and exhibit hyperresponsivity in autonomic measures. The first of these anomalies has been linked, in our theory, with affective flattening as joint outcomes of a brainstem impairment. The second of these anomalies has been shown here to be compatible with the existence of developmental delay in autism. Thus the present study supports our two-factor theory of autism: that the constellation of emotional, cognitive, and behavioural symptoms associated with the syndrome are products of the interaction between a specific brainstem impairment and global developmental delay.

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