

# Conduction Velocity in a Brain Nerve Pathway of Normal Adults Correlates With Intelligence Level

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This is a detailed report of the first demonstration in normal individuals of a correlation between intelligence level ("IQ") and nerve conduction velocity (NCV) in a brain nerve pathway. A total of 147 postsecondary students were tested for nonverbal IQ and latency of the P100 (a visually evoked potential recorded over the primary visual cortex); this latency was used to estimate an approximate NCV in the visual pathway (retina to visual cortex). The correlation between this NCV and IQ is +.26 ( $p = .002$ ); after correction for the restricted IQ range (but not for test ceiling or attenuation), it is +.37. Three recent studies of mentally retarded patients, using similar stimulation and recording, also showed increased P100 latencies relative to controls. These results, plus those of other IQ studies using choice reaction time or long-latency evoked potentials, are all explainable by positive correlations between brain NCV and speed of information processing and between this speed and intelligence level.

## INTRODUCTION

Although the exact nature of human intelligence remains a subject for debate (Sternberg & Detterman, 1986), interest in, and systematic study of, *variations* in intelligence—ranging from severe mental retardation to giftedness—has continued from the early work of Francis Galton (1883) to the present. Galton also suggested that higher intelligence is a result of greater "mental speed," which would be reflected by a shorter reaction time (RT), where RT is the time from a stimulus (visual or auditory) to a response, such as pressing a button. Galton was not successful in relating RT to level of intelligence, but in the last 25 years the predicted negative correlation between RT and intelligence level has become well established (reviewed by Jensen, 1982), having been demonstrated in many

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different laboratories (Jensen, 1982; Vernon, 1987). This relationship has been interpreted as a positive correlation between speed or efficiency of information processing and intelligence, but a specific mechanism is still unknown (Vernon, 1987).

In theory, interindividual differences in speed of information processing could be due to differences in brain (cerebral cortex) "design," or in average cortical nerve conduction velocity (NCV), or in average cortical speed of synaptic transmission (of impulses between neurons), or most likely, to differences in all of these. Because nothing is known about design in relation to cognition (Goldman-Rakic, 1987; Hillyard & Picton, 1987) and, of the other two factors, only subcortical brain NCV (a surrogate for cortical NCV) has become experimentally accessible in normal subjects (this article), we focus here on brain NCV (see Discussion for further details).

Variation in brain NCV as a possible mechanism to explain the RT-IQ relationship follows from the suggestion (Reed, 1984, 1988a, 1988b) that increased NCV in brain nerve axons would increase the speed of information processing, and consequently, the level of intelligence, and vice versa. Recent evidence clearly supporting this suggestion is now becoming available from electrophysiological studies of mental retardation. These studies determined the latency [time from a visual stimulus—checkerboard pattern reversal—to the arrival of an evoked electrical potential (P100) at the scalp over the primary visual cortex]. This visual evoked potential (VEP) was studied in children (Korinthenberg, Ullrich, & Füllenkemper, 1988; Landi et al., 1987) and adults (Creel & Buehler, 1982) with phenylketonuria. In those children the mean P100 latency was significantly increased relative to age-matched controls. In the adult phenylketonurics, 4 of 6 patients had long latencies. Consequently, the corresponding mean NCV over the brain nerve pathway studied is slower in those retarded subjects.

For the P100 (latency about 90–115 ms), as well as for the less-studied N70 VEP (latency around 70 ms), the nerve pathway is the visual pathway: retina to thalamus to primary visual cortex (PVC, Area 17). Following each visual stimulation (pattern reversal) about 50 ms is required for intraretinal processing (including retinal cone transduction) (Baylor, Nunn, & Schnapf, 1987; Chiappa, 1990, pp. 97–99; Lowitzsch, 1989, pp. 82–87). Beginning with the retinal ganglion cell-optic nerve synapse, there is only one additional synapse (optic tract-lateral geniculate nucleus of the thalamus) before this pathway terminates (synapses) in Layer 4 of the PVC (Kandel & Schwartz, 1985, pp. 352, 362). Because the total transmission time for these three chemical synapses is probably less than 3 ms (Hubbard, Llinás, & Quastel, 1969; Kandel & Schwartz, 1985, p. 92), almost all of the latency between the retina and the PVC is nerve conduction time.

Positron emission tomography demonstrates that the pattern-reversal visual stimulation used in the preceding clinical studies produces a sharply localized

response within the PVC (Fox, Miezin, Allman, Van Essen, & Raichle, 1987), in agreement with the neuroanatomy of this nerve pathway. The cortical origin of both the N70 and the P100 VEPs is also demonstrated by the binocular fusion of two monocular random-dot stimuli (Bodis-Wollner, Mylin, & Frkovic, 1989; Skrandies, 1987, respectively). The N70 VEP is thought to be the earliest response from the PVC following pattern-reversal stimulation (Bodis-Wollner et al., 1989). Earlier studies of VEP latencies and intelligence usually used potentials with longer latencies that do *not* relate to specific brain nerve pathways or potential generators. The well-known study of Ertl and Schafer (1969), for example, had electrodes near the *vertex* of the scalp (therefore, not recording from the PVC) and measured potentials as late as 250 ms. Before our investigation, no study of VEP latency and intelligence in normal subjects used P100 latency or pattern-reversal stimulation. These facts may account for the failures to confirm clearly such earlier claims of a VEP-latency-IQ relation (reviewed by Callaway, 1975).

We now report an extension of the preceding clinical results to the normal population. In 147 normal young adult male students whose VEP latencies were measured using the same methodology as in the studies of retarded phenylketonurics, the P100 latency and its corresponding NCV are correlated with a standard measure of nonverbal intelligence. This finding strengthens the view that variation in brain NCV is a factor in the variation in level of intelligence. Together with the RT-IQ correlation, this result can be interpreted as showing that speed of information processing is one of the factors determining the level of intelligence, and that brain NCV is a factor affecting this speed.

## METHODS

### Subjects

The subjects were students from three postsecondary educational institutions in the eastern San Francisco Bay region of California; 75 were from a university whereas 72 were from two community colleges (2-year institutions accepting any high school graduate). All were male, between 18 and 25 years of age, of European ancestry, and in apparent good health. Subjects using corrective glasses wore them during testing. Each gave informed consent. These subjects, plus others, were also tested for NCV in the median nerve of the arm and for RT; those results were published in Reed and Jensen (1991).

### Personal Characteristics and Intelligence

Subjects were questioned on their handedness and visual acuity and were measured for height, weight, head length (using a cephalometer caliper in the sagittal plane with the blunt tips at the glabella and opisthocranium) (Olivier, 1969) and oral temperature (during P100 testing).

The university students were given the Raven's (1983a) Advanced Progressive

Matrices intelligence test; the college students were given the Standard Progressive Matrices version (Raven, 1983b). These tests were given without time limit; most students took between 30 and 60 min. For comparability, the Raven scores were converted to equivalent Otis-Lennon IQ scores (general population,  $M = 100$ ,  $SD = 16$ ; Jensen, Saccuzzo, & Larson, 1988).

### VEP Testing

Each subject was tested for two medium-latency ( $< 120$  ms) VEPs, N70 and P100, evoked by pattern-reversal stimulation. These VEPs are the earliest well-defined potentials recordable over the PVC using this stimulation; only P100 is used clinically (Chiappa, 1990; Lowitzsch, 1989). Using a two-channel clinical test instrument (TD20, TECA Corp., Pleasantville, NY), standard VEP testing procedures (Chiappa, 1990) were followed, except that subjects were tested binocularly instead of monocularly. Stimuli were black and white checkerboard patterns (squares 12.5 mm on a side, subtending a visual angle of  $43'$  at 1 m), reversing (black to white, white to black) at 2 Hz, presented on a video monitor (visual angle of  $14^\circ \times 18^\circ$ ). Luminance level was constant and in the usual clinical range (W. Adamson, personal communication, August 1987). All subjects were tested in the same quiet, darkened room while fixating on a spot in the center of the screen. Gold cup electrodes were applied to four scalp sites:  $O_z$ ,  $F_z$ ,  $C_z$ , and  $F_{pz}$  (10-20 System). Impedances were usually below 4 k $\Omega$ ; a band pass of 2–100 Hz was used. Channel 1 was  $O_z$  (neg.) and  $F_z$  (pos.); Channel 2 was  $C_z$  (neg.) and  $F_z$  (pos.);  $F_{pz}$  was ground. The difference between Channels 1 and 2, equivalent to  $O_z - C_z$ , where  $O_z$  is over the occipital (visual) cortex and  $C_z$  is at the vertex of the head (both in the sagittal plane), was used to detect N70 and P100. [This electrode montage and procedure follows the manufacturer's recommendations. The subtraction corrects for the small potential sometimes present at  $C_z$  (Stockard, Hughes, & Sharbrough, 1979). The  $O_z - F_z$  waveform usually resembled that of  $O_z - C_z$  very closely.] Usually, 100–200 pattern reversals were given in each of two trials, with a rest period of 2–3 min between trials. Artifactual potentials, from eye blinks and other sources, were automatically rejected. The signal-averaged output was scored (blindly with respect to IQ test results) for latencies to the nearest millisecond by the first author, using electronic cursor readings, and including only well-defined peaks. The N70 and P100 values analyzed were the mean values of the two trials.

### Visual Pathway NCVs

In order to calculate approximate NCVs for the visual pathway (optic nerve–optic tract–optic radiation) from the N70 and P100 latencies, the length of this pathway was roughly approximated (see the following) by the subject's head length. This distance varied from 182–214 mm ( $M = 199.9$ ,  $SE = 0.51$ ) and was not correlated with IQ score ( $r = .12$ ,  $p = .16$ ). Dividing this distance by the N70 or P100 latency gives an approximate corresponding NCV, V:N70 or

V:P100. As noted before, latencies between the retina and PVC are almost entirely due to nerve conduction time. It is, therefore, proper to speak of NCV for the visual pathway.

### **Rationale for Using Estimates of Visual Pathway NCVs**

These NCVs underestimate the true (but unknown) NCVs because, as noted before, retinal processing requires some 50 ms of the observed latencies. The N70 latency, therefore, actually represents about 20-ms conduction time in the visual pathway whereas the P100 represents about 50 ms of conduction time. The true NCVs for this pathway are consequently about two to four times greater than the estimates. Although not an accurate measure of the true NCVs, these approximate estimates can still be used for *correlation* analysis because, among subjects, the measured head length should be closely proportional to the length of the actual visual pathway and the mean retinal processing time, say about 50 ms, to a first approximation can be considered a constant that can be subtracted from the variable of interest (observed VEP latency) without greatly affecting the correlation.

### **Statistical Analyses**

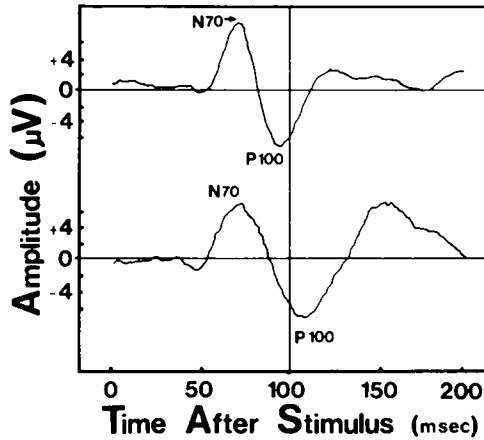
Standard tests for parameter distribution and Pearson correlation were used. All *p* values are two-tailed.

## **RESULTS**

Oral temperature did not significantly affect the two latencies nor did age, height, weight, handedness, or need for corrective glasses. Repeat tests ( $n = 14$ , several days to several weeks later, show that P100 latency is quite constant within an individual over time, the correlation between first test and repeat test being  $+ .801$  ( $p = .0006$ ) and the mean difference (repeat - first) being  $-.036 \pm .507$  (*SE*). N70 latency ( $n = 10$ ) may be somewhat less stable, the corresponding correlation being  $+ .732$  ( $p = .016$ ) and the mean difference  $+ .400 \pm .897$ .

N70 and P100 peaks for 2 subjects, selected to illustrate variability of P100 latency, are shown in Figure 1 (p. 264). Table 1 (p. 264) shows the distributions of sources, ages, and IQ scores of the two student groups and the total population of 147 subjects. As expected for these selected student samples, the IQs are usually well above the general population mean of 100 and the standard deviations are reduced from the population mean of 16. The IQ distribution of the total sample is normally distributed about the mean of 117.9 (skewness and kurtosis are not significant).

Table 2 (p. 265) gives the means and distributions of the two VEP latencies, N70 and P100, and the two corresponding visual pathway NCVs, V:N70 and V:P100, for the 147 subjects. (Means for the two groups differ only for V:P100, where university students slightly exceed community college students, 2.013 vs. 1.983,  $p = .043$ .) The N70 and P100 mean latencies are 72.33 and 100.17 ms,



**Figure 1.** Representative VEP responses from 2 subjects. Each curve is a signal-averaged response over the first 200 ms following the stimulus (reversal of the checkerboard pattern, black to white, white to black). *Upper curve:* from 122 reversals, N70 and P100 peak latencies 70 and 95 ms, respectively. *Lower curve:* from 140 reversals, N70 and P100 latencies 71 and 106 ms, respectively.

respectively. The P100 mean agrees well with the range of normal values in the literature (Chiappa, 1990; Lowitzsch, 1989). (Normal values for N70 are not available.) The standard deviations and ranges show moderate variability among subjects in these latencies. The mean conduction velocities for V:N70 and V:P100, 2.770 and 1.998 m/s, respectively, as noted in Methods, underestimate the true (but unknown) NCVs by a factor of about 2–4. Each NCV shows a limited variability about its mean. There appear to be no published estimates for these NCVs.

Table 3 shows the correlations among IQ score, VEP latencies, and visual pathway NCVs for the 147 subjects. N70 latency does not correlate significantly

**TABLE 1**  
Description of Subjects

Source	n	Age		IQ Score <sup>a</sup>			
		M	(SE)	M	(SE) <sup>b</sup>	SD	Range <sup>b</sup>
University	75	20.4	(0.2)	123.8	(0.9)	8.0	102–136
Community College	72	19.9	(0.2)	111.7	(1.2)	9.9	87–134
Total	147	20.2	(0.2)	117.9	(0.9)	10.8	87–136

*Note.* All subjects were normal male students of European ancestry between 18 and 25 years of age from the eastern San Francisco Bay region of California.

<sup>a</sup>IQ score: Equivalent Otis-Lennon IQ (Intelligence) score from Raven's Progressive Matrices score (nonverbal test; Jensen, Saccuzzo, & Larson, 1988). <sup>b</sup>Five university students and 2 college students received the maximum possible scores; consequently, the means and maxima should be somewhat greater than shown.

TABLE 2  
VEP Latencies and Visual Pathway NCVs in the 147 Subjects

Statistic	VEP Latencies (ms)		Visual Pathway NCVs (m/s) <sup>a</sup>	
	N70	P100	V : N70	V : P100
Mean	72.33	100.17	2.770	1.998
Standard Error	0.30	0.30	0.012	0.007
Standard Deviation	3.65	3.61	0.150	0.089
Minimum	63.5	92	2.44	1.75
Maximum	82	112.5	3.20	2.22

*Note.* The means for university and community college students do not differ significantly (two-tailed) for N70, P100, V : N70, or head length. They do differ significantly ( $p = .043$ , two-tailed) for V : P100, being 2.013 and 1.983, respectively. P100 means differ significantly ( $p = .034$ ) for a one-tailed test, latencies being 99.63 and 100.72, respectively.

<sup>a</sup>See text for details.

with IQ but P100 latency does ( $r = -0.21$ ,  $p = .010$ ). Both NCVs correlate with IQ, the V:N70 correlation being  $+ .18$  ( $p = .025$ ) and the V:P100 correlation being  $+ .26$  ( $p = .0017$ ). Using the IQ standard deviation observed in our student population (10.79), we can estimate what the IQ–NCV correlations would be in the general unselected population (McNemar, 1964, pp. 203–205). These corrected correlations are *IQ–V:N70*:  $+ .27$ ; *IQ–V:P100*:  $+ .37$ . Correcting for “ceiling effects” in IQ scores (see Table 1) would further increase these correlations.

We can examine the nature of the positive IQ–V:P100 correlation by observing the mean IQ of subjects selected for different V:P100 values. Figure 2 (p. 266) shows mean IQs for V:P100 quintiles. It is seen that the positive relation holds over the entire range of V:P100 values; mean IQ steadily increases from 113.8 in the lowest quintile to 122.4 in the highest, with no significant deviation

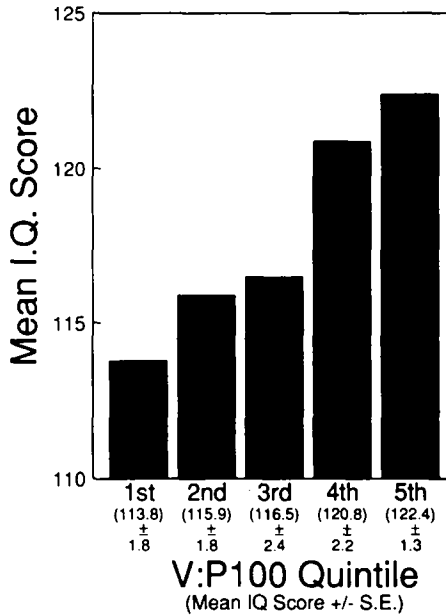
TABLE 3  
Correlations Among IQ Score, VEP Latencies, and Derived  
Visual Pathway NCVs in the 147 Subjects

	IQ	N70 Latency	P100 Latency	V : N70 NCV
N70 Latency	$-.117^a$	—	—	—
P100 Latency	$-.212^b$	$+.389^{**}$	—	—
V : N70 NCV	$+.184^c$	$-.823^{**}$	$-.296^{**}$	—
V : P100 NCV	$+.256^{d**}$	$-.185^*$	$-.714^{**}$	$+.525^{**}$

*Note.* See text for correction of NCV–IQ correlations for restricted range of IQ scores.

<sup>a</sup> $p = .159$ , exact two-tailed probability. <sup>b</sup> $p = .010$ , exact two-tailed probability. <sup>c</sup> $p = .025$ , exact two-tailed probability. <sup>d</sup> $p = .0017$ , exact two-tailed probability.

\* $p < .05$ , two-tailed. \*\* $p < .002$ , two-tailed.



**Figure 2.** Distribution of mean IQ scores in V:P100 quintiles. The distribution of V:P100 values of the 147 students, from lowest (1.75) to highest (2.22), was divided into quintiles. Quintile 1 contains the 20% of students with the lowest V:P100 values; Quintile 2 contains the 20% of students with values between the 20th and 40th percentile, and so on. The cutting points were 1.920, 1.980, 2.028, and 2.064. The linear regression of individual IQ on quintile number (1–5) has a slope of 2.21 IQ points per quintile ( $p = .0004$ ) with no significant deviation from linearity ( $p = .87$ ).

from linearity. The regression of IQ on quintile number has a slope of 2.21 IQ points per quintile ( $p = .0004$ ).

## DISCUSSION

This study appears to be the first attempt, in normal subjects, to estimate directly one of the determinants (brain NCV) of information-processing speed and to correlate it with a measure of level of intelligence. As noted in the Introduction, of the three factors (design, mean brain synaptic transmission speed, mean brain NCV) determining processing speed and, consequently, to some degree, the level of intelligence, only brain NCV has become noninvasively accessible in normal subjects. None of the earlier studies of VEP latencies and IQ, for example, that of Ertl and Schafer (1969), could estimate brain NCVs because their latencies were not for specified brain nerve pathways.

The brain NCV we studied—visual pathway NCV—is indirectly estimated, and because variations in cortical design and synaptic speed are (necessarily) ignored here, we would not expect a high correlation between our estimate



(V:P100) and IQ; in fact, the correlation is modest. Against this we may put the observed agreement with two a priori expectations: (1) positive correlation between speed of information processing and IQ (because of the established RT-IQ correlation), and (2) interindividual variation in mean brain NCV. The latter expectation follows from the fact that every human nerve studied, cranial or peripheral, shows significant variation among normal adults in NCV; the coefficient of variation is usually around 10%, (Ma & Liveson, 1983). With the realization of these prior expectations in mind, our significant "modest" correlations have more meaning than a first assessment might indicate.

We have presented data showing that the increased P100 latency found in mental retardation due to phenylketonuria (if not optimally treated) has a close parallel in normal young adult students: the less intelligent students, on average, have longer P100 latencies than the more intelligent. The P100-latency-IQ relation thus appears to be a general one. The finding that the NCV estimates for the visual pathway, V:N70 and V:P100, derived from N70 and P100 latencies, correlate more highly with intelligence than do the latencies themselves, supports the view that NCV, not latency, is the primary variable of interest here.

It should be noted that V:P100 mainly estimates the NCV of the optic radiation, the band of nerve fibers from the lateral geniculate nucleus of the thalamus to the PVC because most of the conduction time between the retina and cortex occurs in this segment of the visual pathway. In the cat (and presumably in humans) these nerve fibers are small diameter axons conducting at speeds of 1–10 m/s (Martin, 1984), whereas the optic nerve and tract are composed of larger fibers conducting at a mean NCV of about 25 m/s (Stanford, 1987). In ferrets, the range of NCVs in the optic nerve and tract is 10–45 m/s (Baker & Stryker, 1990). After correcting for intraretinal processing time (see Methods), the V:P100 estimate of 2.00 m/s gives an NCV estimate of about 4 m/s, in agreement with the preceding range for the optic radiation.

Most of the many earlier studies of VEP latencies and intelligence or mental retardation are not directly relevant to this study because they differ significantly from it in one or more methodologies (e.g., including a wide age range of subjects, using flash stimulation (all did), latencies > 120 ms, no electrodes over the visual cortex, mixed types of subjects). Several studies do have indirect relevance. Galbraith, Gliddon, & Busk, (1976) studied 24 heterogeneous mental retardates, including Down's syndrome (DS), using flash stimulation and visual cortex recording, and found significantly increased late-latency VEPs from the retardates (322 vs. 270 ms for controls). Using similar techniques, DS alone has shown similar delayed late-latency VEPs in other studies (Dustman & Callner, 1979; Dustman, Schenkenberg, & Beck, 1976; Gliddon, Busk, & Galbraith, 1975). The many developmental abnormalities of the brains of DS patients (Benda, 1969), however, may lessen the relevance of these VEP findings to the study here. (In contrast, note that the brains of phenylketonurics are *normal* at birth.)

More directly relevant was a study of normal children, aged 10 or 11 years, who were either bright or dull intellectually (Dustman et al., 1976). Using the preceding techniques, a late (ca. 250 ms) VEP was significantly delayed in the dull children, in agreement with other studies (reviewed in Callaway, 1975). The common thread through these earlier studies is that, when long-latency VEPs differ, the less intelligent group has the longer latency. Because flash-induced VEPs reach the cortex within 70 ms (Dustman & Callner, 1979), latencies over 200 ms are due mainly to conduction time *within the cortex*. These between-group latency differences are therefore due primarily to differences in speed of cortical conduction.

Another long-latency evoked potential (evoked auditorily, visually, or somatosensorially), the P3 (also called P300, due to a "surprising" or meaningful event, ranging in latency from 250–600 ms depending on the stimulus and paradigm, recorded at or near the vertex of the scalp; Hillyard & Picton, 1987; Oken, 1990), agrees with the previous long-latency VEP findings. In normal university students the visual P3 latency and choice RT are positively correlated (Kutas, McCarthy, & Donchin, 1979), suggesting that the P3, like RT, is a measure of speed of information processing correlated with intelligence level. Like the long-latency VEPs, most of the P3 latency occurs after the stimulus reaches the cortex. Clinical studies on demented and retarded patients show an increased P3 relative to controls (Hillyard & Picton, 1987; Oken, 1990). An increased P3 latency is also found in DS (Blackwood, St. Clair, Muir, Oliver, & Dickens, 1988; Galbraith et al., 1976) and in mental retardation due to fragile X chromosomes (Blackwood et al., 1988). Again, the groups with lower intelligence have increased cortical latencies. Brain pathology in these patients may complicate interpretation of these results but, even so, it is reasonable to expect that such pathology would decrease the speed of information processing, and consequently, increase the P3 latency *and* decrease intelligence, thus producing the observed correlation.

### **Hypotheses and Supporting Data**

We suggest that two hypotheses, each supported by several kinds of evidence, can adequately explain our observed correlation between intelligence and visual pathway NCV.

**H1:** The visual pathway NCV (estimated here by V:P100) is positively correlated with the mean NCV of the cerebral cortex.

**H2:** Intelligence requires the processing of information and the level of intelligence is positively correlated with the speed of this processing. This speed, in turn, is correlated with mean cortical NCV.

1. Evidence for H1 comes from several sources. Neurons in the visual pathway and in the cortex share a common origin and environment. The optic radia-

tion (whose NCV V:P100 primarily estimates) and cortical neurons both have very small caliber axons and conduct at similar speeds (Martin, 1984); they are, consequently, very similar. In the peripheral nervous system, NCVs of different nerves appear to be positively correlated within individuals (Hegmann, 1979). In the optic tract of ferrets, the NCVs of different classes of nerve fibers appear to be positively correlated (Baker & Stryker, 1990). On these grounds, it is probable that H1 is true: Individuals with a lower visual pathway NCV should, on average, have a lower mean cortical NCV, and vice versa. Experimental support for H1 comes from the increased cortical latencies of the late-latency VEPs of dull, retarded, and demented patients, and increased P3 latencies of retarded and demented patients, as discussed before. Because these patients also have an increased P100 latency in the visual pathway, these findings offer direct evidence for H1.

2. The following considerations support H2:

a. It is obvious that "intelligent activity," such as problem solving, requires the processing of information supplied to the cortex through one or more of the senses. If one person can consistently solve sets of problems in, say, half the time that another can, we usually think of him or her as being more intelligent, at least for problem solving. The significant negative correlation between choice RT and intelligence (Jensen, 1982; Vernon, 1987) provides support for H2 here.

b. Information processing requires the transfer of information, represented by action potentials in cortical neurons, along axons and across synapses, in local cortical regions and between cortical regions. This axonal transfer, which is the traveling action potential, occurs at some NCV for each axon. NCVs vary among cortical neurons but, in principle, may be aggregated into some mean cortical value. The faster this mean NCV, the greater the speed of information processing, and vice versa.

The V:P100-IQ correlation is about  $+ .4$  (after correcting for restricted IQ range); it would be higher after correcting for the ceiling effect and unreliability of the IQ determinations. Therefore, V:P100 variation may account for at least 20–25% of the IQ variance. But V:P100 itself is a quite indirect measure of mean cortical NCV, which, by the preceding arguments, is the ultimate variable of interest to us here. Consequently, variation in cortical NCV probably accounts for 25% or more of the general population variance in IQ scores. Other factors, such as variation in brain design (see the following), may account for a larger fraction of the total variance.

It may be useful to attempt to place our findings and interpretations on V:P100, cortical NCV, and speed of information processing within the larger framework of cognitive operations of the cortex in general. Although the neurophysiological and neuroanatomical details of even elementary cognitive opera-

tions cannot be specified (Goldman-Rakic, 1987; Hillyard & Picton, 1987), and therefore, a fortiori, intelligence cannot be analyzed into these components, it is obvious that the information processing required for intelligence uses large groups of neurons interconnected in specific patterns. The organization (design) of cortical neurons into a cognitively functioning organ, the cerebral cortex, is clearly a basic requirement for intelligence. Our results and views in no way challenge this importance of design. Our point here is that, for a *given* brain design, the mean cortical NCV can affect the speed of information processing and, in turn, the level of intelligence as reflected by a test score. This is so because information can only be transferred from one cortical region to another along axons (at some velocity) and across synapses (with some delay). The cumulated axonal conduction time and cumulated synaptic delay (time) involved in information processing are probably within an order of magnitude of each other (based on estimates of cortical NCV, mean length of axons, and synaptic delay; Reed, 1988b). Consequently, cortical NCV should be an important component of information processing speed. If two brains have the same cortical design but one has a mean cortical NCV 20% greater than the other, the one with the greater NCV will process information faster. The relative importance of between-individual variation in cortical NCV and between-individual variation in cortical design and in synaptic delay, for producing the observed variation in IQ scores is, of course, completely unknown.

Although an adequate, functioning cortical design is the *sine qua non* of intelligence, there are no data, so far, showing that in the "normal population," say IQ range 70–130, cortical design varies appreciably from one individual to another. In contrast, it is now clear that normal persons vary in their visual pathway NCVs and, as argued before, very probably in their mean cortical NCVs. This, we feel, is the potential importance of our results and interpretation: an available approach for understanding normal *variation* in intelligence at the neurophysiological level.

Finally, we believe that our results with normal subjects logically complement the results found in mentally impaired patients. Taken together, these findings increase our understanding of cognitive function in both groups. Because the visual pathway can serve as an accessible and useful surrogate for some aspects of cortical nerve pathways, particularly the NCV, it seems desirable that further studies of NCV in the visual pathway be carried out both in cognitively well-defined normal and nonnormal populations.

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