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A Somatosensory Latency Between the Thalamus and Cortex Also Correlates With Level of Intelligence

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As part of a study on speed of information processing and intelligence, 205 young adult postsecondary students were tested for somatosensory evoked potential (SEP) latencies and intelligence. Following stimulation at the wrist, latencies of three SEPs were determined: N13, generated in the cervical spinal cord/medulla region; N19, generated in the thalamus; P22, generated in the arm region of the somatosensory (parietal) cortex. These latencies and two latency differences, N19 − N13 and P22 − N19, were tested for correlation with a nonverbal measure of intelligence; only P22 − N19 significantly correlated ($r = -.217$; $p = .013$, two-tailed). Comparing this latency difference in students in the first IQ quartile (mean IQ = 103.4) with that of students in the fourth quartile (M = 131.2) showed mean differences of 4.13 ms versus 3.21 ms, respectively ($p = .0034$, two-tailed).

P22 − N19 measures time for signal transmission from the thalamus to the sensory cortex. These results agree with considerably more extensive data on visually evoked potentials showing a negative correlation between IQ and the latency for a visual stimulus of the retina to produce a signal at the visual cortex (most of this latency is between the thalamus and the cortex; Reed & Jensen, 1992). The findings here agree with the visual results and strongly suggest that the IQ–latency correlation occurs because the latency indexes cortical nerve conduction velocity, an important component of information-processing speed.

INTRODUCTION

To date, our studies on speed of information processing and intelligence in normal young adults have reported on correlations between IQ and reaction time (RT; Jensen & Reed, 1990) arm nerve conduction velocity (NCV; Reed & Jensen, 1991), and visual pathway (retina to thalamus to primary visual cortex) latency and NCV (Reed & Jensen, 1992). We found, as expected, IQ correlations

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with RT but not with arm NCV. The visual pathway latency for a visually evoked potential (VEP), P100, and the visual pathway NCV derived from this latency, were also correlated with IQ. The P100 correlation confirmed what had previously been observed in three studies of mental retardates; more intelligent subjects (normal controls) have shorter latencies (higher visual pathway NCV) than less intelligent subjects (patients), and vice versa (Reed & Jensen, 1992). We now report another (final) set of IQ correlations from this study: correlations with somatosensory evoked potentials (SEPs).

SEPs are electrical signals, usually recorded from the scalp over the relevant part of the somatosensory (parietal) cerebral cortex, following stimulation of some peripheral nerve, for example, the median nerve of the arm. Following the same general procedures used for VEPs, modern instruments using signal averaging can easily and reliably record several short-latency SEPs (< 30 ms after stimulation at the wrist) from normal persons. Determination of such SEPs in patients has become an established procedure in neurology (Chiappa, 1990, pp. 307–437; Stöhr, 1989). SEPs have not, to our knowledge, been used in relation to cognitive levels of either normal or abnormal subjects.

Following stimulation of the median nerve at the wrist, three SEPs, N13, N19, and P22, are usually observed in normal subjects (Chiappa, 1990; Stöhr, 1989). N13, measured over the cervical vertebrae, is generated in the region of the cervical spinal cord/medulla; N19, measured over the contralateral arm region of the somatosensory cortex, is generated in the thalamus; P22, measured with the same electrode as for N19, is generated in the somatosensory cortex (reviewed by Chiappa, 1990, pp. 376–391). The latency difference P22 − N19 thus represents the time for potentials to travel from the thalamus to the parietal cortex and is entirely within the brain, as is the VEP P100 latency. Both latencies are due almost entirely to nerve conduction time, not to synaptic transmission time (for P22 − N19 there is only one synapse included because N19 is a postsynaptic signal in the thalamus and P22 is postsynaptic in the cortex). We present evidence here that, in normal young adults, the P22 − N19 latency, like the visual P100 latency, is negatively correlated with IQ.

SUBJECTS AND METHODS

Subjects

The subjects were students from three postsecondary educational institutions in the eastern San Francisco Bay region of California; 117 were from a university and 88 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.

Personal Characteristics and Intelligence

Subjects were questioned on their handedness and were measured for height, weight, head height (using the Todd head spanner), and arm and oral temperatures.

The university students were given the Raven's Advanced Progressive Matrices intelligence test (Raven, 1983a); 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 mins. For comparability, the Raven scores were converted to equivalent Otis-Lennon IQ scores (general population mean = 100, *SD* = 16; Jensen, Saccuzzo, & Larson, 1988).

Somatosensory Evoked Potentials (SEPs)

Three SEPs, N13, N19, and P22, were determined for each subject following standard clinical procedure for upper limb stimulation (median nerve at the wrist; Chiappa, 1990; Stöhr, 1989). (N and P refer to negative or positive; numbers refer to approximate latencies in milliseconds following stimulation.)

Subjects were tested while reclining comfortably (head on a small pillow) on their backs on a padded wooden clinical examining table in a quiet darkened room. Stimulation was with 0.05-ms pulses at 5 Hz and was just sufficient to produce a slight twitch in the thumb. Usually between 300 and 600 pulses were given and the signal-averaged waveform (recorded on a TD20 instrument, TECA Corporation, Pleasantville, NY) was used for measurements. Two recording electrodes (C'_3 or C'_4 and C_7) and one reference electrode (F_{pz}) were used (designations of the international "10–20 System"). C'_3 and C'_4 are scalp locations over the arm regions of the somatosensory cortex contralateral to the stimulated arm (the preferred arm for writing). C_7 is over the seventh cervical vertebra (back of the neck); F_{pz} is a midline scalp location in the high forehead. Scalp preparation and use of electroencephalographic (EEG) type gold cup electrodes followed standard procedure. Electrode impedances were below 7 kΩ. Filters were set for a band pass of 20 Hz to 2 kHz. The electrode montage was: Channel 1, C'_3 or C'_4 (for right- or left-handed subjects, respectively) negative, F_{pz} positive; Channel 2, C_7 negative, F_{pz} positive.

Following one successful recording session, N19 and P22 were usually displayed in Channel 1 of the TD20 and N13 was in Channel 2. Electronic cursors were used to measure the three latencies to the nearest 0.1 ms and also the amplitude difference between N19 and P22; the waveforms, latencies, and amplitude were recorded on a strip printer. After a rest of about 2 min, the procedure was repeated. An oral temperature was then taken. Only well-defined (having a definite maximum value) SEP peaks were used; this requirement resulted in the loss of some subjects (eliminated blindly with respect to IQ level). The mean latencies and amplitudes of the two trials were used and two interpeak latencies, N19 − N13 and P22 − N19, were calculated. N19 − N13, also termed central conduction time (Cant & Shaw, 1986), measures the conduction time from the spinal cord at the level of the seventh cervical vertebra to the thalamus. This is almost identical to the medulla–thalamus latency because the C_7–medulla latency is only about 0.17 ms (Stöhr, 1989). The P22 − N19 latency, from thalamus to somatosensory cortex, as explained before, is the latency of major interest.

To correct for the known effects of temperature and physical size on the latencies, as well as correcting for the possible effects of other covariates, each latency and amplitude was regressed on age, arm and oral temperatures, height, weight, arm span, head height, and handedness (right vs. left). Covariates significant at the .01 probability level were corrected for by regressing the subjects' measurements to the population mean values.

RESULTS

The numbers, mean ages, and mean IQs for the community college and university students are shown in Table 1. As expected for the this student population, the IQs are well above 100 and the standard deviations are reduced from the normal 16. Arm and oral temperatures and arm span affected N13 and N19 latencies whereas oral temperature and arm span affected P22 latency. These effects were removed by regression. Table 2 shows these latencies, the two interpeak latencies, and the P22 − N19 amplitude for the two groups and the total. There are no significant differences between groups for any of the variables. The considerably greater standard deviation of P22 (and P22 − N19) is due, at least in part, to its longer nerve pathway. This provides a greater opportunity for spreading of its peak (due to variation in conduction times among axons) and so making the location of the peak less definite. The N13, N19, P22, and N19 − N13 latency means for the total agree quite well with published latencies (Chiappa, 1990; Stöhr, 1989), although each of our means is a few tenths larger. The amplitude mean is between the two published values.

The correlations in the total population between IQ and SEP latencies and amplitude are shown in Table 3. Only the P22 − N19 correlation, −.217, is significant (p = .013, two-tailed). Since a negative correlation is predicted, a one-tailed p can be used here: .007.

Comparing latency differences of students in the first IQ quartile (IQ range = 87–109; M = 103.4) with those in the fourth IQ quartile (range = 127–136; M = 131.2) offers another way to look for possible IQ–latency associations. Table 4 shows the mean latencies of the N19 − N13 and P22 − N19 differences for the

TABLE 1
Description of Subjects

		Age[a]		IQ[b]	
Source	***n***	***M***	***SD***	***M***	***SD***
Community College	88	20.0	1.9	112.0	9.1
University	117	20.6	2.1	123.8	8.3
Total	205	20.3	2.0	118.7	10.4

Note. Each subject has one or more well-defined SEP latencies.
[a]Range = 18–25 years; the means differ at the .05 level. [b]Equivalent Otis-Lennon scores (Jensen et al., 1988); the means differ at the .001 level.

TABLE 2
Short-Latency SEP Latencies and Amplitudes[a]

Source	Latencies (ms)					Amplitude (μV)
	N13	N19	P22	(N19 − N13)	(P22 − N19)	(P22 − N19)
Community College						
M	13.84	19.86	23.49	5.89	3.73	3.51
(*SD*)	(0.43)	(0.53)	(1.57)	(0.44)	(1.60)	(1.48)
University						
M	13.97	19.87	23.16	5.84	3.38	3.34
(*SD*)	(0.44)	(0.51)	(1.22)	(0.45)	(1.19)	(1.21)
Total						
M	13.92	19.86	23.29	5.86	3.53	3.41
(*SD*)	(0.44)	(0.52)	(1.38)	(0.45)	(1.38)	(1.32)

[a]Values of mean and (standard deviation), corrected for arm span and temperature. Only well-defined peaks are included (see text for definitions). Sample sizes for group means range from 54 to 101. There are no significant differences between groups for any variable at the .05 level, two-tailed.

TABLE 3
Correlations in Total Population Between SEP Values and IQ

SEP Value	*r*	SEP Value	*r*
N13 latency	.067	(N19 − N13) latency	.032
N19 latency	.042	(P22 − N19) latency	−.217[a]
P22 latency	−.156[b]	(P22 − N19) amplitude	−.099

[a]$p = .013$, two-tailed ($n = 130$). [b]$p = .057$, two-tailed ($n = 149$).

TABLE 4
SEP Latency Differences (in ms) for Students in the First and Fourth IQ Quartiles

Latency Difference	First IQ Quartile[a]			Fourth IQ Quartile[b]		
	(*n*)	*M*	*SE*	(*n*)	*M*	*SE*
N19 − N13	(33)	5.79	0.07	(49)	5.85	0.06
P22 − N19	(25)	4.13[c]	0.34	(42)	3.21[c]	0.12

[a]Mean IQ = 103.4, range = 87–109. [b]Mean IQ = 131.2, range = 127–136. [c]These means differ at $p = .0034$, two-tailed.

first and fourth quartiles. The latencies do not differ for N19 − N13 but do differ for P22 − N19, being significantly less ($p = .0034$, two-tailed) in the fourth quartile.

DISCUSSION

Because N13 measures the latency of nerve tracts that are entirely outside the brain (wrist to cervical spinal cord) and a previous study of this population (Reed & Jensen, 1991) did not find any correlation between arm NCV and IQ, it is not surprising that N13 also is uncorrelated with IQ. N19 includes this tract plus the short tract to the thalamus whose latency is measured by N19 − N13; N19 and N19 − N13 also were uncorrelated with IQ. Only the latency for the tract between the thalamus and parietal sensory cortex, measured by P22 − N19, clearly correlates (negatively) with IQ.

This thalamocortical tract has a formal neuroanatomical similarity to the corresponding tract in the visual pathway, the optic radiation, which runs from from the thalamus to the primary visual cortex. A latency over the visual pathway, P100, between the time of retinal stimulation to recording over the visual cortex, was used to derive an NCV that is significantly ($p = .0017$, two-tailed) negatively correlated with IQ in this student population. Three studes of mental retardates also show P100–IQ correlation (Reed & Jensen, 1992). Most of the nerve conduction time of the P100 occurs in the optic radiation (OR) because its fibers are of small diameter and conduct slowly (relative to the larger and faster fibers in the optic nerve and tract). Reed and Jensen (1992) argued that because these fibers in the OR are similar in size, conduction speed, and origin to cortical fibers, they can serve as an accessible surrogate for the inaccessible cortical tracts whose NCV we wish to know.

The possible similarity in thalamocortical fibers of the sensory and visual pathways can be explored by calculating the SEP NCV and comparing it to the VEP NCV. We note that the vertical distance from the thalamus to the vertex of scalp is about one-half the vertical distance from the medulla to the vertex and the medulla is in line with the two external ear openings (from magnetic resonance imaging; Gademann, 1984, p. 47). Because the Todd head spanner used measures head height as the vertical distance from the external ear openings to the vertex, the straight line distance from the thalamus to the vertex is about half this head height. The velocity of nerve conduction from the thalamus to the parietal sensory cortex is therefore about 0.5 × (headheight in mm)/(P22 − N19 in ms): $M = 21.8$, $SD = 5.5$ m/s. (This probably is a minimal estimate because the sensory thalamocortical tract is not a straight vertical line). The mean NCV in the OR (adjusted for retinal processing delay) is about 4–5 m/s (Reed & Jensen, 1992), so the NCV is probably four or more times faster (and the nerve fibers are correspondingly thicker) in the sensory thalamocortical tract than in the OR. This calculation suggests that the fibers in the sensory tract are not as similar to cortical fibers as are the fibers in the VEP tract; perhaps this accounts for the lower correlation with IQ.

Jensen and Reed (1990), studying this student population, found that discriminative (Oddman) RT was negatively correlated with IQ ($p < .01$); subtracting simple RT from Oddman RT increased this correlation ($r = -.274$, $p < .001$). Reed and Jensen (1993) found that in spite of RT and visual pathway NCV each being separately correlated with IQ, RT (as Oddman RT or Oddman RT − simple RT) did not appear to correlate with visual pathway NCV. It is, therefore, of interest to examine the Oddman RT : (P22 − N19) correlation: It is +.03, also nonsignificant. It was suggested (Reed & Jensen, 1993) that there are two independent processes affecting information-processing sped, one RT linked and one cortical NCV linked. The lack of correlation of the P22 − N19 latency with RT may be another example of this.

Increased cortical NCV (reflected by increased thalamocortical NCV) would carry information faster along the cortical nerve fibers and therefore increase information-processing speed and, consequently, level of intelligence, and vice versa. Decreased total cortical nerve path length (between visual cortex and motor cortex), required for a correct RT decision (for example), might also increase information-processing speed, and vice versa (Reed & Jensen, 1993; the possibility of normal subjects having different cortical nerve path lengths for a simple standard task is shown by the work of Gevins et al., 1989).

In summary, the sensory thalamocortical latency (P22 − N19) results agree with the more extensive VEP latency data (largely thalamocortical) in correlating with IQ. These results are also in agreement with long-latency VEP and P3 (P300) latency data; shorter latencies (higher cortical NCV) go with high IQ, and vice versa (reviewed by Reed & Jensen, 1992). The evidence for negative correlation between brain latencies, over defined nerve pathways, and IQ, and the interpretation of this as a consequence of positive correlation between information-processing speed and IQ, is further strengthened by these SEP findings.

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