Central somatosensory conduction time in severely growth-stunted children

Heike Hesse, Maria-Felix Rivera, Ivette de Díaz, and Gregory J Quirk

ABSTRACT
To examine the effects of chronic malnutrition on central nervous system function, we used the somatosensory evoked potential to measure the central conduction time of 20 children aged 7-8 y with heights below the third percentile for their age and 20 control children in Honduras. The two groups differed significantly in socioeconomic status, achievement in Bender’s neurointegrative test, and hematocrit, but not in birth weight. After median nerve stimulation, the mean central conduction time (interpeak latency between N13 and N20) for the growth-stunted group (6.19 ± 0.52 ms) did not differ significantly from that of the control subjects (6.30 ± 0.58 ms), suggesting appropriate myelination and fiber diameter. Somatosensory tracts may escape damage resulting from postnatal dietary deficiencies because myelination in these tracts is almost complete at birth. Am J Clin Nutr 1998;67:93–6.

KEY WORDS Malnutrition, stunted growth, somatosensory evoked potentials, somatosensory tracts, myelin, central conduction time, children

INTRODUCTION
The World Health Organization estimates that 230 million children worldwide have a low height-for-age, also called growth stunting (1). Height-for-age reflects long-term growth and is altered by chronic nutritional disorders. In Honduras, national surveys indicate that 33.9% of children aged <5 y suffer from growth stunting (1), suggesting a pervasive effect of chronic malnutrition.

Central nervous system (CNS) injuries caused by severe malnutrition can be shown clinically through neurologic signs and symptoms such as dullness, apathy, irritability, muscular weakness and wasting, anxiety, chronic fatigue, hypotonia, hypo- or hyperactivity, attention deficit, and poor school performance (2). In addition to the clinical manifestations observed, electrophysiologic abnormalities have been detected in malnourished children. Changes have been observed in peripheral nerve conduction velocity (3) and brainstem auditory evoked potentials in children with marasmus and kwashiorkor, the most severe forms of acute malnutrition (4, 5). Relatively little is known about the adverse effects of chronic malnutrition on CNS physiology in humans.

There is evidence that the myelination process is impaired in malnourished children and animals. In humans, decreased concentrations of proteolipids, cerebroside, sulfatide, and plasmalogens were found in white matter as well as abnormal cellularity in different CNS sites. Extensive data from rats suggest a severe reduction in the brain myelin concentration due to malnutrition (6). Nevertheless, few studies have assessed the effects of malnutrition on the function of a long myelinated central tract in children.

Somatosensory evoked potentials (SSEPs) constitute a standard clinical technique used to assess the function of the somatosensory peripheral tracts and central projections. A peripheral nerve is stimulated transfcutaneously and the propagated volley is recorded over peripheral and central structures, giving rise to well-characterized components. The cervical potential, reflecting postsynaptic activity in the cervical cord, is a negative wave with a latency of 13 ms (N13). The cortical component (N20) is generated by activation of the primary cortical somatosensory receiving area. Central conduction time can thus be measured by using the interpeak latency between the cervical N13 and the cortical N20, which represents the time necessary for nervous conduction between the cervical cord and the primary somatosensory receiving area (7). We compared the N13-N20 interpeak latency between growth-stunted (malnourished) and control children to assess the effects of chronic malnutrition on central nervous conduction.

SUBJECTS AND METHODS
A group of 20 growth-stunted children with heights below the third percentile for their age and 20 age- and sex-matched control subjects with normal heights were included in the study. The mean (±SD) ages of the control and growth-stunted groups were 7.4 ± 0.3 and 7.6 ± 0.4 y, respectively, and there were 7 girls and 13 boys in each group. The children were contacted through a family dental center where they were being treated. Parents signed informed consent forms, as required by the Ministry of Health in Honduras. Growth stunting was determined by using

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Received February 28, 1997.
Accepted for publication August 5, 1997.
The control and growth-stunted groups differed significantly in height, weight, visuomotor integration, hematocrit, and socioeconomic status, but not in birth weight (Table 1). A representative example of the SSEP for a control child is shown in Figure 1. We observed no significant difference in central conduction time between control and growth-stunted children. The mean (±SD) central conduction times of the control and growth-stunted children were 6.30 ± 0.58 and 6.19 ± 0.52 ms (Figure 2).

DISCUSSION

We carried out the first study of central conduction time in a somatosensory pathway in chronically malnourished children. The growth-stunted children in our sample had a mild but significant decrease in hematocrit, indicating a deficit in hemoglobin, which is usually associated with protein-energy malnutrition (9). Central conduction time in the growth-stunted children did not differ significantly from that in the control children. Central conduction time between the cervical cord and the primary somatosensory cortex involves transmission through 1) the cuneate and anterolateral fasciculi, 2) the medial lemniscus and the spinothalamic fibers to the ventral posterior nucleus of the thalamus, and 3) thalamic radiations to primary somatosensory cortex. The normal conduction times we observed suggest that the fiber diameter and myelination of these tracts were not affected by malnutrition severely enough to induce growth stunting or to lower hematocrit values. Although no conduction deficit was observed with median nerve stimulation, there was the possibility that tibial nerve stimulation might have shown an

![Figure 1](https://example.com/figure1.png)

**FIGURE 1.** Representative traces showing the N13 and N20 waves of the somatosensory evoked potentials recorded simultaneously at positions C5S-EPc and CPe-Ac, respectively, in a control child. Each trace is an average of 1000. Shock artifact begins at the arrow.

### TABLE 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control children</th>
<th>Growth stunted children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>121.07 ± 4.47</td>
<td>106.78 ± 3.91</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>24.39 ± 3.61</td>
<td>18.44 ± 1.89</td>
</tr>
<tr>
<td>Bender Test score</td>
<td>1.41 ± 0.64</td>
<td>2.76 ± 0.45</td>
</tr>
<tr>
<td>Socioeconomic status</td>
<td>2.16 ± 0.00</td>
<td>4.80 ± 0.46</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>0.3931 ± 0.0237</td>
<td>0.3756 ± 0.0292</td>
</tr>
<tr>
<td>Birth weight (kg)</td>
<td>3.41 ± 0.44</td>
<td>3.50 ± 1.22</td>
</tr>
</tbody>
</table>

1 X ± SD.

2, 3 Significantly different from control children: 2 P < 0.001, 3 P < 0.05.
normal birth weights of the stunted children in our study suggest that the nutritional insult was largely postnatal, thereby sparing the rapid, mostly prenatal, myelination of the somatosensory tracts. Myelination in the motor tracts and cerebrum mostly start at late gestation and progress well after birth (12).

Corticospinal fibers may have been at risk in the sample of children in the present study, as suggested by their impaired visuomotor skills. In rats, early postnatal malnutrition reduced the number of myelinated fibers in the corticospinal tract but not in the cuneate fasciculus (13). A previous study from our laboratory showed decreased conduction velocity of corticospinal fibers in adult rats that were malnourished postnatally during the first 3 wk of life (14). Other studies showed slowed conduction in auditory (15) and visual (16) systems of malnourished rats. Thus, the normal function we observed in the somatosensory system may not apply to other systems, especially those that mature postnatally and have long myelinating phases, such as the corticospinal tract.

Many previous studies showed altered neuropsychologic functions in malnourished children (17). An organic injury is thus suspected and may involve subtle changes in dendritic structure and synaptic function (17), in addition to altered nerve conduction. More human and animal studies are needed to adequately assess the neurologic effects of chronic malnutrition and its potential cost to society.

We thank Marco A Zavala and Mayra Ochoa for assistance with the recordings and Winston Mejía (Director of the Laboratory of Neurophysiology) and Miguel Sierra (Director of the Health Center “Alonso Suazo”) for their support.

FIGURE 2. Histograms showing the distribution of central conduction time measured as the interpeak latency between the N13 and N20 potentials of the somatosensory evoked potential for control and growth-stunted children. There were no significant differences.

Our findings appear to support the notion (based largely on brain weight) that the developing CNS may be “spared” during times of nutritional stress (10). The simplest explanation of our findings, however, may relate to the critical periods of development of the tracts in question. All experts in this area agree that myelination of the different tracts takes place at different times and at different rates during development. Yakovlev and Lecours (11) described these variations as myelogenetic cycles (other authors have used the term myelinating phases). Tracts that acquire large amounts of myelin over a short developmental time have short myelinating phases and those that acquire large amounts of myelin over a prolonged time have long myelinating phases. For example, the medial longitudinal fasciculus and the cuneate fasciculus show early myelination and rapid myelinating phases. Myelination of the solitary tracts and cervical corticospinal tracts indicate slow progression from birth to the second postnatal year. Sites with long myelinating phases could be at greater risk of impairment over a greater portion of gestation and postnatal life. The medial longitudinal fasciculus in the human fetus is the first to undergo myelination early in the second half of gestation, followed by the afferent tracts, such as the cuneate fasciculus, gracile fasciculus, medial lemniscus, and lateral lemniscus (12). The

effect due to the increased conduction distance. This was unlikely, however, given that there was no trend toward longer conduction times in our medial nerve data. Nevertheless, future studies might examine the effects of malnutrition on tibial SSEPs as well as the possible differences between spinal and brain components of the pathway.

REFERENCES


