Electrophysiological assessment of the central lemniscal pathway in man[1]

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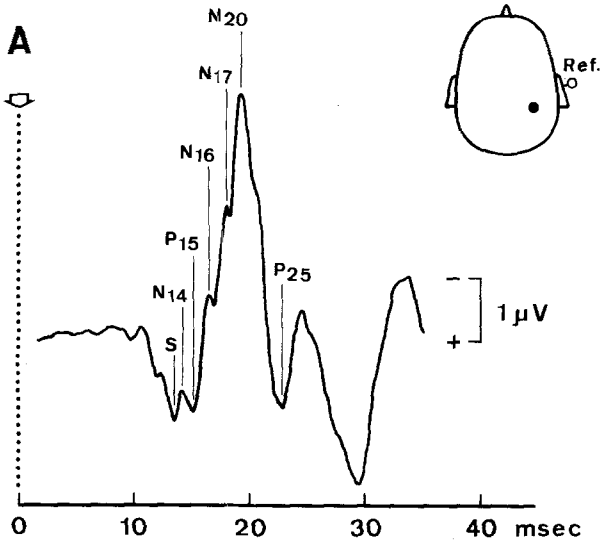
Summary. A new method to evaluate the conduction time of the central somatosensory pathway is described. Normative data established in a group of young subjects are reported.

A large number of diseases of the central nervous system can affect the lemniscal pathway, thus reducing the conduction velocity of the somesthesic afferent impulses. Therefore the assessment of the conduction time of the central somatosensory pathway (from the spinal cord to the primary cortex) may bear a great relevance as a clinical screening test, especially in the face of equivocal symptoms or signs. A tentative evaluation of such a conduction time, obtained by the difference in latency between the response evoked at the scalp and at the axilla has recently been proposed[2]. However, the ideal electrophysiological method to localize the site of a lesion along an afferent pathway is the scalp detection of potentials reflecting the progressive activation of such a pathway at different levels, as clearly demonstrated by the clinical application of the brain stem auditory response[3-7].

It has recently been contended that the early components of the somatosensory evoked potential (SEP) may be far-field responses arising in the central lemniscal pathway[8-10]. Moreover, it is well known that the latency of the somatosensory primary response depends on the height of the subject[11]. The same might be true even for the short-latency components of the human SEP, thus reducing their possible diagnostic relevance. The adequacy of the SEP short-latency components was therefore investigated: in particular, the peak latencies of SEP components preceding the primary response, as well as some relevant SEP interwave latencies, were statistically evaluated as a function of the height of subjects (152–187 cm; mean: 172.4) in order to identify chronological not height-dependent parameters. In 20 healthy volunteers (16 males, 4 females), aged 25–37, scalp recordings of cerebral somatosensory potentials evoked by median nerve stimulation at the wrist were performed, according to the methods described elsewhere[10].

Results and discussion. The primary response of the somatosensory cortical potential (i.e. the N20-P25 complex) evoked by the median nerve stimulation at the wrist was



B LATENCIES

S	N_{14}	P_{15}	N_{16}	N_{17}	N_{20}	P_{25}
13.1 ±0.9	13.9 ±0.8	14.6 ±0.9	15.9 ±0.9	17.2 ±0.9	18.7 ±1.1	21.3 ±1.2

Fig. 1. To show (A) the early components of the somatosensory evoked response to left median nerve stimulation at the wrist and (B) their mean latencies. Stimuli consisted of square pulses (duration: 0.2 msec; intensity: 3 to 4 times thumb motor threshold) generated at a rate of 1 c/s. The SEP was led off from the exploring electrode, placed over the left hand projection area, and the reference (Ref) on the right ear lobe. Analysis times of 25 or 50 msec were used and usually 512 or 1024 responses were summated.

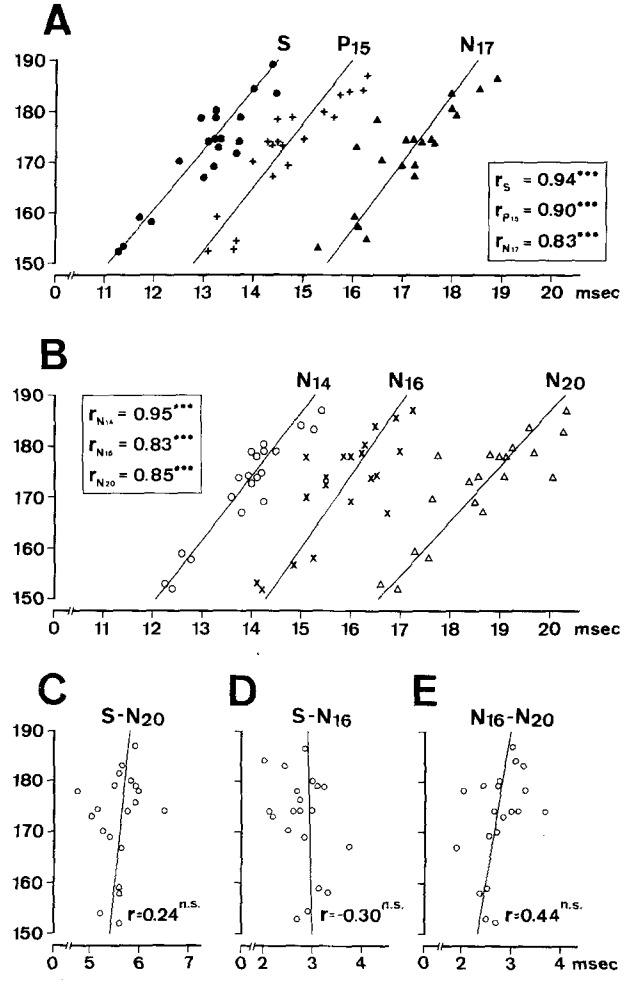


Fig. 2. Scatter diagrams relating peak latencies of SEP components (A and B) and 3 different SEP interwave latencies (C, D and E) to the height of subjects. Each line is fitted to the data points by the method of least squares. Correlation coefficients (r) and their statistical significance are reported in each diagram.

constantly preceded by a sequence of deflections of low amplitude[10] (figure 1, A), the latencies of which (figure 1, B) were significantly related to the height of subjects (figure 2, A and B). On the opposite, the interwave latencies S wave-N20 (5.6±0.4 msec), S wave-N16 (2.8±0.4 msec) and N16-N20 (2.8±0.4 msec) did not show a significant relationship to the height of subjects (figure 2, C–E), their distribution curves being normal, at least within the age-range investigated.

It has thus been shown that the latency of each SEP component under investigation (i.e. S wave through N20) is significantly related to the height of the subject. Therefore, changes in latency of the SEP components, per se, would be scarcely adequate to detect abnormalities in conduction time of the central segment of the lemniscal pathway, especially if the disease process is so diffuse (or disseminated), as to affect bilaterally conduction of afferent impulses. To this purpose, three interwave latencies seem to be particularly suitable, i.e. S wave-N20, S wave-N16 and N16-N20, all of them bearing no significant relationship to the height of subjects. As to the functional significance of such intervals, it should be recalled that several lines of evidence gathered from both animals[12] and humans[13] support the hypothesis that the sequence of small waves, consistently preceding the primary response, are far-field potentials generated in the cervical posterior columns (S wave), the medial lemniscus and/or the dorsal columns nuclei (N14-P15 complex), the thalamus (N16) and the thalamocortical radiation (N17). Specifically it is postulated that: a) S wave-N20 reflects the time interval between the transit of an afferent volley through the posterior columns of the spinal cord and its arrival at the contralateral somatic area: therefore it represents the central conduction time of the lemniscal pathway; b) S wave-N16 and N16-N20 correspond respectively to the spino-thalamic and thalamocortical conduction times, though further evidence is needed to confirm this assumption. Studies are now in progress to find out to what extent the central conduction time of the lemniscal pathway may be affected by aging. Thereafter, the effective clinical relevance of these data will be verified.

1 Part of these results has been presented at the XX National Congress of the Italian Society of Neurology, held in Rome on November 24–26, 1977.
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Normal liver actually possesses a high vascular plasminogen activator activity

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Summary. Blood vessels isolated from the liver of the rat, guinea-pig, rabbit, dog and pig showed histochemically a more or less high plasminogen activator activity. In whole liver sections, the abundant release and diffusion of inhibitors of fibrinolysis from the liver parenchyma during the histochemical procedure, partially or totally mask this high vascular activity.

Human[1–7] and animal[8–12] liver is regarded as an organ which normally shows a very low vascular fibrinolytic activity[2–3,7–12] or no activity at all[1,4–6,8–9]. This conclusion was drawn by histochemical studies on whole liver sections. Comparative histochemical studies on whole organs and isolated tissue layers have shown, however, that in whole organ sections the release and diffusion of inhibitors of fibrinolysis from layers rich in inhibitors during the histochemical procedure partially or totally mask the fibrinolytic activity in adjacent tissue layers[13]. Normal liver is very rich in plasmin inhibitors[7,12]. Therefore, this study was undertaken to compare the fibrinolytic activity and plasmin inhibition on whole liver sections and sections of blood vessels isolated from corresponding areas of the liver in several species.

Materials and methods. Liver specimens from 18 adult, normal Wistar rats, 15 guinea-pigs, 10 White New Zealand rabbits, 10 dogs and 8 pigs were taken immediately after the animals were killed. From corresponding anatomical areas of each liver, blood vessels of various diameter were isolated as gently as possible. Liver specimens and isolated blood vessels were washed in saline, frozen and stored at a temperature of −20 °C or below. Both tissue preparations were studied simultaneously within 1 week after the initial freezing.

Fibrinolytic activity. Fibrinolytically active sites were located in frozen sections (6 µm) by the histochemical fibrin slide technique as described before[14], using bovine plasminogen-rich and plasminogen-free fibrinogen (Poviet, Organon-Teknika, Oss, The Netherlands). An average of 50 sections were examined from each specimen. The evaluation of the fibrinolytic activity was made as described before[14].

Plasmin inhibition. An average of 50 sections from the same specimen were examined with Noordhoek Hegt and Brakman's[7] fibrin slide 'sandwich' technique as follows: Frozen sections (16 µm), collected on precleaned microscope slides, were covered with a layer of fibrin by mixing and spreading 100 µl of a solution of plasminogen-free bovine fibrinogen and 20 µl of a solution of bovine thrombin (Leo pharmaceuticals, Denmark) in saline (20