THE USE OF SOMATOSENSORY EVOKED POTENTIALS FOR THE DETERMINATION OF SENSORY NERVE CONDUCTION VELOCITIES

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GRAHAM JOHN BALL.

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ABSTRACT

Title: The Use of Somatosensory Evoked Potentials for the Determination of Sensory Nerve Conduction Velocities.

Author: Graham John Ball.

In 1947 Dawson[17] first recorded a somatosensory evoked potential after electrically stimulating the ulnar, median and lateral popliteal nerves. Giblin (1964[27]), using improved techniques, reported an initial negative wave at 14 to 18 msec. followed by a positive potential peaking at 23 to 31 msec.

Using the peak of the first negative wave or the beginning of the positive wave and the peak of the first derivative of the downsweep of the positive wave as latency markers, the sensory conduction velocities of the ulnar, median, peroneal and posterior tibial nerves were measured. The variance of the background noise was used as an indicator of the amplitude of background noise. Statistical tests were performed upon the data to test the randomness and independence of the data. Intraindividual consistency tests were conducted and some tests for distribution of the evoked potential over the scalp were made. Nerve conduction velocities of three clinical patients showed the applicability of the test to aiding clinical diagnosis of sensory nerve problems.

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LITERATURE REVIEW

As far back as 1762, Sauvage[33] arrived at a velocity of the nerve impulse of more than 100,000 metres per second. It was not until 1908 that Piper [54,55] used the muscle action potential in man as an indicator of the arrival of nerve impulses by stimulation of the median nerve at two points and arrived at a motor conduction velocity of 60 to 65 metres per second. The first successful attempt to record percutaneously from a mixed nerve in situ was made by Eichler (1938[25]). Dawson and Scott (1949[22]) used the same procedure and obtained a far better resolution by photographically superimposing 50 traces. Their attempts to measure the conduction velocity proved unsuccessful. The failure was attributed to the difficulty of obtaining pairs of records which had the same shape from electrodes on different sites of the nerve. Dawson (1956[21]) recorded the first purely sensory nerve action potentials. He stimulated the digital nerve and recorded an action potential at the wrist and elbow. Gilliatt and co-workers (1961[28], 1958[29], 1962[30]) recorded sensory conduction time as a diagnostic aid to investigation of patients with peripheral nerve lesions.

In 1947 Dawson[17] conducted an experiment electrically stimulating the ulnar, median, and lateral popliteal nerve and received, after superimposing 50 sweeps, an evoked potential over the corresponding contralateral sensory areas of the central gyrus. The short latency between the beginning of the response of the order of 18 to 20 milliseconds suggested that the response indicated events occurring in the cortex, at, or soon after, the time of arrival of afferent volleys to the cortex. Dawson also indicated that the shorter latency of evoked potential went with a shorter path taken by the impulse. A surface positive wave beginning at 19 milliseconds and which peaked 25 to 28 milliseconds after stimulus at the contralateral ulnar nerve at the elbow, possibly preceded by a small negativity of a few milliseconds duration, was reported by L-E Larson (1953[42]). The negative deflection peaking at 40 milliseconds and a second positive wave lasting 50 to 60 milliseconds was followed by a non-specific response. In Dawson's (1956[21]) recording of evoked potentials, after stimulation of the contralateral ulnar nerve at the elbow, he used the averaging method described by Dawson (1953[19], 1954[20]). He recorded the first significant negative deflection starting at 20 milliseconds, a positive beginning at 25 milliseconds and peaking at 28 to 30 milliseconds and reported 1 later phase.

The general form of this response, recorded by Goff et al (1962[31]) was consistent among subjects. The initial evoked activity, component 1, based on median nerve stimulation, was a triphasic, positive, negative, positive complex with peak latencies at 16 ± 2 milliseconds, the negativity sharply peaked 3 to 4 milliseconds later, and a positive peak at 25 ± 4 milliseconds which was not consistently detectable in all subjects. Components 2 and 3 designated two positive deflections while component 4 had a negative peak at 65 ± 14 milliseconds and positive peaking at 85 ± 20 milliseconds. This component 4 showed the greatest intra- and inter-subject variability in waveform. In 1964 Giblin[27] reported that in some subjects as many as 7 distinct components could be identified in the first 100 milliseconds of the evoked potential. The time elapsing between the stimulus at the contralateral median nerve at the wrist and the peak of each of the potentials within the first 35 milliseconds could usually be measured with an accuracy of ± 1 millisecond but there was considerable difference between the responses recorded in different subjects. The initial negative was followed by a positive potential which differed as to whether one or two brief positive potentials were recorded within the first 35 milliseconds following the stimulus. The initial negative potential began 14-18 milliseconds after application of the stimulus and reached a peak at 17.5 to 21 milliseconds. In Group V, a single

brief early positive potential followed the initial negative one. The latency of its peak ranged from 23 to 31 milliseconds with a mean of 26.8 milliseconds. In the second type of response, Group W, the records from these subjects showed two brief positive potentials separated by a negative going deflection which varied in prominence in different subjects. The first positive potential at 22.4 ± .04 milliseconds (range 22-23 milliseconds), was evoked by relatively weaker shocks and attained its maximum amplitude with shocks which were below the motor threshold at a time when the second was still barely visible. The second at 30.71 ± 1.50 (29-33 milliseconds), attained its maximal amplitude in these subjects only when the shocks evoked a definite motor response. Furthermore, increasing the intensity of the stimulus never caused a decrease of more than 1 or 2 milliseconds in the latency of the first positive potential but occasionally decreased that of the second by as much as 6 milliseconds. The two positive potentials could also be shown to have different potential fields on the scalp. The first positive potential was clearly seen in records from electrodes anterior and medial to the standard (electrode over "hand" area of post central gyrus) while the second was the more prominent from more posterior and lateral electrodes. These brief early potentials were followed by a series of later waves which were longer in duration and often greater in amplitude than the earlier components of the response but showed greater individual differences in latency and

in their relative prominence. A later negative wave and still later positive wave could usually be identified with confidence. In eight subjects the positive wave was followed by a still later negative wave resembling the negative wave preceding it. Thereafter, the individual variations were so great that it was difficult to distinguish which waves were comparable. Allison[4] reported that at approximately 17 milliseconds following the stimulus a small positive wave sometimes appeared followed by a clearly repeatable sharp negativity with a peak latency of very nearly 20 milliseconds followed at 22 to 23 milliseconds by an equally positive wave designated 1. The positive peak was clearly defined in some subjects, in other subjects it appeared as a notch or inflection on the larger succeeding positivity. This was followed by a complex diphasic positive wave designated 2, and 3, a negative positive wave designated 4 and a negativity followed by a large positivity designated 5. S. J. Larson et al[43] reported a latency of the first portion of the response was usually between 15 and 20 milliseconds with median nerve stimulation and 30 to 35 millisecon ds when the siatic nerve was stimulated. Uttal and Cook[61] have divided the evoked potentials into M,N, and O waves with latencies of approximately 20, 30, and 50 milliseconds respectively. Dawson[17] first reported an initial positive wave at 22 to 23 milliseconds after stimulation of the ulnar nerve at the wrist but later (1954[20]) he reported that the first positive

might be preceded by a small negative potential. Giblin[27] in his series of experiments showed an initial negative potential to be a consistent feature of the response evoked by stimulation of any of the nerves of the upper extremity. Allison (1962[4]) and Goff et al (1962[31]) have in fact, reported that in addition to an early negative potential with a latency similar to that reported here they at times recorded a still earlier positive potential with a peak of 16 ± 2 milliseconds.

The distribution of the evoked potential over the skull was examined by a number of investigators. Goff et al (1962[31]) examined the waveform and distribution of evoked activity occurring within 500 milliseconds after peripheral stimulation. The general form of the response was consistent among subjects, different deflections having different distributions. Their components 1 and 2 were confined to the contralateral post-quadrant of the head from the post-Rolandic area back to the occiput with the focus of the activity posterior to the surface marking of the Rolandic sulcus. The distributions of components 1, 2, and 3 agreed with Dawson's[18] work in 1950 when he found a response maximum over the surface marking of the Rolandic sulcus but noted that the antero-posterior gradients were asymmetrical around the maximum. Goff et al [31] considered the focus of activity to be posterior to the surface marking of the Rolandic sulcus. Giblin's[27] initial negative potential has a relatively wide distribution and was recorded with much the same amplitude

and latency by electrodes placed anywhere over the contralateral hemisphere behind the central fissure and with a diminishing amplitude for about 3 centimetres in front of it. The early positive potential was slightly more localised over the scalp than the initial negative potential, largest in amplitude when recorded by an electrode in the standard position, ie. over the "hand" area of the post central gyrus. It decreased fairly abruptly when recorded by electrodes in front of the central fissure and decreased more gradually in records from electrodes either medial or lateral to the standard electrode in the same cortical plane. In all these locations it had the same latency but with electrodes three centimetres behind the standard, its peak was both smaller in amplitude and two milliseconds later. The responses evoked in the right and left hemispheres by shocks to the contralateral median nerves were compared in ten subjects. In 8, the early components of responses recorded from the two hemispheres were virtually indistinguishable. Responses evoked by stimulation of the medial or lateral popiteal nerves were largest in amplitude when recorded by scalp electrodes placed approximately over the upper end of the post central gyrus either on the midline or a few centimetres lateral to it. These responses never showed an initial negative component but consisted of a single early positive potential followed by negative and positives waves. S. J. Larson[43] reported that, for each individual, the

latency was not affected by changes in stimulus parameters but the amplitude and configuration of the evoked potential waveform were affected by the position of the recording electrodes and the distance between them. It was apparent that the responses of greatest amplitude were obtained from the electrodes in the vicinity of sensory and motor cortex on the side contralateral to the stimulus. The amplitudes of the potentials evoked by contralateral median nerve stimulation were not bilaterally symmetrical. The amplitudes were greater on the left side in 15 patients and on the right side in 4 patients, and were equal bilaterally in 1. In most of the subjects the amplitude difference was approximately 20 - 25%.

The initial specific response was abolished by concentration and unaltered at rates of stimulation up to 5 seconds. This was reported by L-E Larson[42] in 1953. The primary waves remained unaltered at stimulus rates up to 20 per second but this was tested by Larson only in one case. Dawson[21] reported that the later phase of the evoked potential varied more than the initial response with changes of attention or wakefulness. Giblin[27] showed that the cerebral response evoked by shocks to the median nerve at the wrist or elbow was greatly decreased in amplitude, if, during the time that they were being applied, various kinds of natural stimuli were repeatedly applied to the distal parts of the limb innervated by the same nerve. Repeatedly stroking the skin of the palm or fingers, intermittent pressure on the fingernails, and either passive or voluntary movements of the fingers were all

equally effective. While all components of the evoked response showed a reduction in amplitude roughly in proportion to the intensity of the natural stimulation, the initial negative potential was much less affected than either the early positive or late waves. In most subjects, when shocks of a constant voltage were regularly applied, the amplitude of the evoked cerebral response was observed to decrease with time. The shocks which just exceeded the threshold of some of the motor fibres in the nerve were applied uninterruptedly for 30 minutes. At two minute intervals, the response to 20 consecutive stimuli was averaged. The peak to peak amplitude of the initial negative and positive potentials changed during the experiment. By contrast, the late negative wave of the peak at 42 milliseconds showed an initial increase followed by an abrupt decrease in amplitude. It was likely that the initial increase in amplitude was due to a decrease in resistance to the skin and the stimulating cathode. The sensory and motor thresholds were frequently noted to decrease shortly after the beginning of an experiment and the skin was often noted to become reddened in this area. The decrease in the amplitude of the negative wave after 6 minutes of stimulation did not seem to be due to any subsequent decrease in the effectiveness of the stimulus, since it was not accompanied by any change in the motor response. In other subjects the afferent volley was also monitored and this also did not decrease with time as did the cerebral response. A decrease in the amplitude of evoked potentials

was also observed by Giblin[27], during sleep. The initial negative potential was but slightly reduced whereas the early positive was reduced. Once muscle contractions in response to the stimulus were observed further increases in intensity or duration did not affect the amplitude or configuration of Larson's (S.J.)[43] evoked potential. Increasing the stimulus frequency from 0.5 through 10 cycles per second produced slight reduction of amplitude without change in latency or major alteration of wave form. Although the waveforms were altered by anaesthetic agents, additional changes were not observed during paralysis. When Larson, S.J.[43] produced evoked potentials by stimulation of the dorsal columns during spino-thalamic tractotomy, the recordings obtained with the subject under nitrous oxide anaesthesia with succinlycholine immobilization appeared similar to those obtained with peripheral stimulation of unanaesthetized subjects with normal muscle function. Uttal and Cook[61] reported that the most significant difference between subjects detected during pilot studies was the fact that with certain subjects the M and N waves seemed to be partly or even wholly fused. Deliberate variations in electrode location did not substantially change the waveform. The latency of the M wave was fairly stable varying by only ± 1 millisecond for constant stimulus amplitude. The N wave, however, exhibited more jitter in the averaged responses, displaying recorded ranges as great as ± 4 milliseconds. The O wave variability was very large. The O wave seemed to be related to extremely complex

phenomenon such as awareness, attentiveness, and thought, as one would expect from a process associated with the alpha rhythm. They showed that the preceding stimulus had an inhibitory effect upon the succeeding evoked potential and that this lasted up to 200 milliseconds, and that the M wave was less seriously affected than the N wave. Strong interactions occurred when both the masking and test stimuli were presented to the same side of the body and there was little indication of interaction when they were presented to opposite sides of the body. When the subject was asleep a surprisingly small change occurred in the contralaterally generated M and N waveforms for it was only in the trailing edge of the N wave that any change could be detected. In ipsilateral records the O wave decreased in amplitude as the subject fell asleep. A comparison with the contralateral evoked potential confirmed this was also happening on the contralateral side of the body. There was no significant interaction between responses produced by independent stimuli applied simultaneously to each hand. In 1968, Nezlina and Vorob'eva[53] showed that evoked potentials in the sensorimotor cortex of different cats show individual differences which persist for a long period, that the differences in configuration of evoked potentials were independent of recording conditions (thickness of bone, position of electrode,etc.) and also strength of electrical stimulation of the skin, and that individual differences in evoked potentials probably reflected individual peculiarities in structural and functional organization

of the CNS of particular animals, especially of the cerebral cortex. During polygraphic investigation of monosynaptic reflexes in newborns and on infants, EEG deflections resembling evoked responses and time locked to the tapping of a tendon or muscle, were seen by Hrbek et al[37,38]. The responses, most clearly seen in the contralateral Rolandic area, consisted of a primary and an unspecific component. Latencies became progressively shorter during the neonatal period. The responses were dependent upon the behavioural state. In regular (NREM) sleep the unspecific component was larger. In irregular (REM) sleep it became smaller and the primary component achieved greater prominence. During quiet wakefulness the responses were similar to those in irregular sleep. It was suggested that these responses were probably of proprioceptive origin.

When a conditioning evoked response is followed after an interval by a test response the former will alter the latter. Relative test response amplitude plotted against interstimulus interval yields the recovery function. Recovery is expressed as the ratio of amplitude of test response to that of control response. At the briefest interval between the two stimuli, 3 milliseconds, the average amplitude of Allison's[4] component 1 was more than 60% of the control value. Minimum responsiveness occurred in different subjects at intervals from 5 to 20 milliseconds. Component 1 was approximately 90% recovered in 200 milliseconds. Latency measures obtained for the negative and positive phase of 1 showed little if any change in peak latency as a function of recovery. Several

characteristics of component 5 indicated that it was equivalent to the response previously named "vertex potential" of a "non-specific" nature. It may be evoked by auditory, somatic or visual stimuli. Schwartz and Shagass[59] showed a peak of recovery before 20 milliseconds followed by a decreased responsiveness and a subsequent return to full recovery at 100-200 milliseconds. They also reported decreased recovery with increased intensity.

Dawson[18] in 1950 suggested that the cerebral responses which may be detected in healthy man after electrical stimulation of nerve were probably produced by excitation of at least two kinds of nerve fibre. One type of fibre was that which carried afferent impulses from cutaneous receptors and the other was probably that carrying impulses from proprioceptors in muscle. Records made through skin by Dawson and Scott[22] showed that 70% of the nerve action potential which could be detected was due to activity in fibres with a lower threshold than motor fibres. It was suggested that the volley in these low threshold afferent fibres produced the greater part of the cerebral response. Records of nerve action potentials also indicated that variation in the size of the afferent volley was not the cause of the variation in size of the cerebral responses to successive stimuli of the same kind. Evidence was produced by Dawson[21] in 1956 which suggested that the afferent fibres from the fingers in man were as large as the largest motor or muscle afferent fibres from the small muscles of the hand and that the more excitable fibres from the fingers had an electrical

threshold at the wrist as low as that of the most excitable motor fibres at the same level in the nerve and lower than that of most of the motor fibres. He showed that the cerebral responses may be evoked by stimulation of fibres with a threshold lower than that of the motor fibres and that there was no evidence for the existence of any considerable number of afferent fibres with a threshold lower than that of those giving rise to a cerebral response. No cerebral responses were detected with stimuli just too small to produce paraesthesiae in the fingers, but stimuli which could just be felt produced detectable cerebral responses in some subjects. "If lower thresholds to electrical stimulation and higher conduction velocity can be accepted in any one species as being associated with larger fibres then conditions such as pressure and ischaemia which affect larger fibres first may be expected to affect sensory fibres from the fingers before they affect motor fibres to the hand." (Dawson, 1956[18]). Giblin's[27] initial negative potential at 14 to 18 milliseconds was evoked by relatively weak shocks and as the intensity of the stimulus was increased it reached a maximal amplitude, without decreasing in latency, slightly before the later components of the response. Increasing the intensity of the stimuli seldom decreased the latency of his early positive potential by more than 1 or 2 milliseconds, and a maximal amplitude was obtained in most subjects when the shocks evoked a barely visible motor response. Giblin pointed out that a close correlation was found in all healthy subjects between

the sensory threshold to electrical stimulation and the intensity of stimulus which evoked recordable cerebral potentials. At the sensory threshold (method of limits) subjects reported regularly applied shocks only intermittently. Detectable cerebral potentials were evoked by shocks of this intensity in some subjects but slightly stronger shocks, which just consistently evoked paraesthesiae in the peripheral distribution of the nerve being stimulated, evoked a recordable cerebral response in all subjects - either initial negative and positive potentials of small amplitude or the late positive wave with a peak latency of 40-60 milliseconds. Stronger shocks were required in order that an afferent volley be recordable from surface electrodes placed over the nerve trunk proximally. At the motor threshold the early components of the cerebral response were close to maximal in amplitude, and the afferent volley was usually between 50% and 75% of its maximum. By contrast to the median and ulnar nerves, the thresholds to electrical stimulation of motor fibres in the medial and lateral popliteal nerves were about the same as those of the afferent fibres responsible for the cerebral evoked response. Consistent with this, when stimulating the popliteal nerves, the sensory and motor thresholds were approximately equal. "If it is assumed that for each of the responses after stimulation at phalanges, wrist and elbow the central delay is the same, the difference in their latencies may be used to estimate the rate at which impulses, giving rise to evoked

cerebral potentials, are conducted by afferent fibres in the nerve." (Giblin, 1964[27]). Conduction velocities estimated in this way by Giblin[27] in one typical subject, were 72.5 metres per second from finger to wrist and 85 metres per second from wrist to elbow within the median nerve. It was interesting to note that the conduction times of the afferent volleys over the same stretches of nerve measured to the initial positive peaks of their triphasic action potentials were usually longer by about 0.5 milliseconds. Estimates of their conduction velocities were therefore somewhat lower. Thus, it was concluded that Giblin[27] found as previously reported by Dawson[20] (1954) that cerebral potentials were evoked by relatively weak shocks close to the threshold for evoking paraesthesiae, that the afferent fibres responsible for the cerebral response had thresholds to electrical stimulation below those of any other fibres in the nerve and the conduction velocity of these fibres was relatively high. Uttal and Cook[61] reported that the cortical response rather than being barely detectable at levels near psychophysical threshold and increasing with increasing stimulus magnitude was well over half of the maximum amplitude at threshold (approximately 1 ma) and is nearly 100%, long before full stimulus amplitudes are reached. They called this a saturation phenomenon. This saturation phenomenon in man was anticipated by a study of Mark and Steiner[48] which showed the relation between the cat's somatosensory cortical

response and the peripheral nerve response to be of this same form - nearly total evoked brain potential at low levels of peripheral nerve response. The intriguing comparisons made by Uttal and Cook[61] were 1) that the estimated of magnitude made by the subjects to randomly presented pulse stimuli amplitudes did show a monotonic increase in the perceived sensation of the full range of stimuli and 2) the peripheral nerve response has also been shown by Brown[14] to increase monotonically as a function of stimulus amplitude to levels well above 10 ma. Gotto et al[29] in 1968, reported that when the ulnar nerve is stimulated at an intensity below the threshold for muscle contraction in the innervated muscles, such as M. abductor digiti quinti in so far as determined by the electromyogram and visual observation, potentials may be recorded along the nerve attributable to the activity of the afferent fibres of the nerve . They illustrated the relationship between stimulus intensity and evoked responses from the ulnar nerve and from M. abductor digiti quinti. The muscle threshold was 1.8 x NT (Nerve Threshold). While nerve responses increased approximately linearlly with slight concavity, those of the muscle rose steeply showing a sigmoid curve. For both nerve and muscle the maximal responses were obtained at 3 x NT. The muscle potentials appeared invariably when the stimulus intensity was raided to 1.80 ± 0.33NT. Thus, the ulnar nerve contained, according to Gotto et al[32], afferent fibres of lower threshold than the efferent motor fibres. The conduction velocity of the afferent

fibres, the low threshold muscle afferents, was calculated to be 80.2 ± 8.4 metres per second. This was faster than the efferent motor fibres (56 metres per second), and the cutaneous afferent arising from the skin of the little finger (55 metres per second).

In 1963 Liberson and Kim[46] reported three negative components for the following latencies: 12 milliseconds, 18-25 milliseconds and 30-40 milliseconds. The two later components were predominently located in the contralateral central area confirming the original Dawson discovery. Their presence extended toward the occiput and the unexpectedly early component predominated at the inion. Further investigation showed that it originated from the midline of the posterior aspect of the cervical region (6-9 centimetres below the inion). The cervical early component presented the most stable latency and therefore according to Liberson and Kim could be used for the determination of conduction velocities in sensory nerve. This response was also reported by Cracco[16]. Bickford[12] and associates focused attention on the occurrence of evoked motor responses in association with more classical potentials of cortical origin. Widespread myogenic responses, first reported in 1963[11] below movement thresholds, to sound and photic stimulation have been described and have been named the sonomotor response and the photomotor response respectively. Myogenic potentials, resulting from median nerve stimulation, contaminated scalp recorded somatosensory evoked responses in 1968 (Cracco et al[16]). Responses were recorded at the posterior aspect of the neck

while the neck extensors were tensed, the lowest recorded latency of these responses being 14 milliseconds. The observation that responses to median nerve stimulation recorded from the neck and torso regions were accentuated by active muscle tension and virtually disappeared with relaxation seemed to establish the myogenic origin. Spinal cord potentials, another conceivable source of such responses, could not have been expected to show this property. Myogenic potentials were demonstrated with latencies similar to those of the classically described cortical response in all subjects. These may also be present in subjects who are apparently relaxed. Thus, it seemed probable that myogenic contamination was responsible for some of the disagreement in results published concerning these responses. The cortical response had an amplitude maximum on the contralateral somasthetic scalp recording region but the myogenic response commonly was symmetric and maximal in relation to temporal or neck muscle groups and underwent phase reversal in these locations. In a significant number of subjects, however, the myogenic response was asymmetric and was maximal contralateral to the side stimulated. Unlike somatosensory responses, in somatomotor (myogenic) responses the amplitude decreased directly with the amount of applied local muscle tension. Somatosensory and somatomotor responses appeared to be transmitted in peripheral nerve fibres having similar excitability properties. The somatomotor recovery cycle after

paired-shock stimuli was characterized by initial depression followed by facilitation and subsequent depression and eventual recovery in agreement with Schwartz and Shagass[59]. Somatosensory recovery was characterized by a similar pattern and the two responses could not be readily differentiated by this method. Unlike the primary evoked response of cortical origin the somatomotor responses interacted with auditory evoked responses. In paired sound-shock stimulation experiments no interaction could be demonstrated between auditory evoked responses and the primary (short latency) evoked somatosensory responses of cortical origin. This is in agreement with the work of Allison[4] who observed interaction only with long latency somatosensory evoked potentials when he used paired sound-shock stimulation.

Evoked potentials recorded by Giblin[27] from the cortex at operation resembled those recorded from the scalp in healthy subjects. A more direct comparison could be made in one of the patients since in this patient evoked potentials could be recorded under the usual conditions from the scalp and from previously implanted electrodes. Although not a healthy subject the patient's evoked potentials were apparently normal. In 1960 Jasper et al[39] conducted experiments on the exposed cortex of man with and without anaesthesia. The evoked potential received from the arm area of the post central gyrus after stimulation of the ulnar nerve showed a complex of 50 to 100 microvolts in amplitude with a latency of about 18 milliseconds (range 16-20 milliseconds). The initial rapid

complex was usually diphasic or triphasic. The peak latencies of the initial three responses being about 20 milliseconds for the positive, 22 milliseconds for the negative and 24 milliseconds for the positive. This initial complex was followed by larger slow wave responses reaching 200-500 microvolts in amplitude. The initial rapid complex appeared about the same with patients under light pentothal and nitrous oxide anaesthesia. Electrical stimulation gave satisfactory sensory responses from the central portion of the evoked potential area to stimulation of the contralateral ulnar nerve. The sensory responses were as follows: tingling in the ring finger, tingling in all fingers of the hand, numbness on the ulnar side of the hand, inflection of little finger, numbness of fingers and arm to elbow, a feeling in the ring and middle finger, electricity feeling in the ring and middle finger with some chronic movement. Sensory responses obtained from the post central gyrus just below the area of maximum evoked potential localization yielded responses in the other fingers of the hand and thumb, obviously adjacent to the ulnar distribution. The later slow components of the evoked potential were highly variable and not so well localised. Domino et al[23] indicated that scalp recordings did reflect the activity of the somatosensory cortex. As might be expected the scalp potentials were markedly attenuated. At the patient's sensory threshold, the scalp responses were of low amplitude while the epidural responses were higher in amplitude.

It was emphasised that scalp and epidural recordings were similar only if a sufficiently large area of cerebral cortex was involved. Discrete cortical electrical stimulation and simultaneous recordings with scalp and epidural electrodes did not show similarity indicating that scalp recordings did not reflect events of small populations of neurons. Presumably, much larger neuronal populations participated in somatosensory evoked responses so that scalp and epidural recordings showed qualitatively similar electrical activity. The scalp did not appear to distort the frequency in the range of 1 to a thousand cycles per second, but merely attenuated the voltage as determined by separate studied with a sine wave generator. The voltage attenuation was approximately 20, depending upon conditions. Kelly et al[40] reported evoked potentials from the cortex surface similar to those of Jasper et al[39]. When recording monopolarly (cortical surface to bone) in man, the largest responses were limited to the hand area of sensorimotor cortex. From here they fell off rapidly in amplitude in all directions. The same was true in animals but smaller potentials could be picked up from almost the entire area of the exposed hemisphere. In both animal and man evidence was presented to show that only in somatosensory hand area was the potential generated immediately beneath the cortical electrode. In all other areas, where potentials were recorded, the responses could not be generated locally beneath the cortical electrode but at some unknown distance.

Goff et al[31] extrapolating from animal data in light ofhomologies,

suggested that component 1 represented potentials in presynaptic thalamocortical fibres of the primary somatosensory projective pathway. Component 2 represented corresponding postsynaptic potentials and that component 3 probably reflected extralemniscal activity similar to the variously called "association area", "ascending reticular formation", "secondary" or "irradiation responses". Giblin[27] suggested that since the negative potential preceeded the positive wave of the primary it could only be due to presynaptic potentials in thalamo-cortical projection fibres. A negative potential might have been recorded from the surface of the cortex due to prolonged depolarization of the terminations of these fibres, acting as a current sink, before the development of the positive wave which is due to postsynaptic potentials in pyramidal neurons. It should have, in this case, been preceded by a positive potential as the impulses in these fibres approach their termination but this might have failed to be recorded due to temporal dispersion. Uttal and Cook[61] have suggested that the M wave was dependent upon the posterior columns, the N wave upon the spinothalamic tract and the O wave upon reticular pathways. Halliday and Wakefield[36] also have concluded from observations in patients with dissociated sensory loss that the average somatosensory potential in man was dependent upon impulses conducted over posterior columns and related fibres. Amplification by second order neurons in the dorsal columns activating a reverberating circuit may have caused the saturated response at the cortex. The coding of sensory intensity may have been by the degree of spread of the response of some subtle spatial coding in which either different

regions or different configurations of cells became active. Several criteria indicated to Allison[4] that the component designated 1 in his study was presynaptic in origin. Its negative and positive phases were each about 2 or 3 milliseconds in duration, briefer than and opposite in polarity to the classical primary postsynaptic response. The recovery function of component 1 also was consistent with the assumption that this potential was a radiation response of thalamocortical fibres presynaptic to the intracortical neurons. The recovery function of component 2 fitted the hypothesis that component 2 was postsynaptic, because its recovery depended upon the recovery both of presynaptic elements and of the cortical population yielding the response. Component 3 may have been analogous to the "association response" of Amassian (1954[6]) and others, or to the "ascending reticular system response" of Brazier (1954[13]). Uttal and Cook[61] proposed that conduction over different afferent pathways was responsible for the polyphasic evoked potentials recorded through scalp electrodes. The late response suggested to L-E Larson[42] a subcortical mechanism as it was decreased in opposite relation to the subject's degree of alertness at the time of stimulation. The explanation of the mechanism might then have been that the stimulus which produced the non-specific waves on arrival at the cortex also activated the reticular formation via collaterals from the classical sensory pathways and this, in its turn, inhibited them.

In contrast to potentials detected by scalp electrodes, responses from depth electrodes considered to be in the nucleus ventralis posterior lateralis of the thalamus could be obtained by S. J. Larson[43] only with contralateral stimulation. Marshall (1941[47]) examined several aspects of thalamic potentials evoked contralaterally by tactile stimulation, and by electrical shocks to the superficial radial nerve. The responses were recorded from the region of synaptic transfer in the lateral parts of the postero-ventral nucleus and he identified three components. The initial one was a sharp positive wave attributed to the summation of positive spikes representing activity in lemniscus axons and terminals. This was followed by a barrage of diphasic and negative spikes which he related to synaptic transfer. A subsequent slow positive wave was interpreted as an after-potential of thalamic neurons because its time relations correlated well with the excitability of the thalamo-cortical relay. Mountcastle and Henneman (1949[51]) studied the topical distribution of thalamic responses evoked by stimulating different parts of the body surface. The sensory figure they plotted within the contralateral postero-ventral nucleus (dorsal midline across the superior portion of the responsive area; tail, anterior laterally; face and mouth medially and extremities inferiorly) was distorted to allow greater volume of thalamic tissue for representation of the parts of the body surface most heavily innervated by sensory axons. In a circumscribed small thalamic area, two or three mm. behind the VL area, ~~that~~ Goto et al[32] reported that a positive phase appeared 13 milliseconds

after delivery of an electric shock to the ulnar at the elbow. A negative wave appeared at 16 milliseconds after the shock. A negative phase was never noted in a single sweep response, but positive deflections of the order of 10μv were recorded. The latency of the negative peaks was 12 to 16 milliseconds. Maximum amplitude of the negative peak was attained at 1.7 x NT, which was below the muscle threshold. Thus it was reasonable to consider the thalamic responses to be evoked by stimulstion of the low threshold muscle afferents. Negative potentials were recorded in only a small area extending 5 mm. vertically and 3 mm. laterally. In scalp leads, evoked positive potentials with a latency of 16 to 19 milliseconds appeared in the parietal region after the shock to the low threshold afferents of the ulnar nerve. In cases in which a small surgical lesion was made in this area of fast muscle afferent projection, no clinically detectable sensory deficit was produced. The large positive deflections recorded at maximum depth suggested that somewhere in the unsearched areas of the thalamus corresponding areas of large negative waves may be present.

Amassian et al[8] in 1964 stated, that on theoretical grounds it appeared unlikely that the surface primary response was due to algebraic summation of action potentials in either thalamo-cortical axons or in cortical neurons. The specific afferent component in the surface response was more readily identified when the stimulus was applied within or close to the specific thalamic relay nucleus. Several investigations (Li and Jasper 1953[45], Amassian 1953[5], Mountcastle et al 1957[52]) have noted that cortical unit activity is greatly reduced by factors such as deep anaesthesia or by the

Several investigations (Li and Jasper 1953[45], Amassian 1953[5], Mountcastle et al 1957[52]) have noted that cortical unit activity is greatly reduced by factors such as deep anaesthesia or by the arrest of artificial ventilation (Li and Jasper, 1953[45]). Because the surface primary response to somatic stimulation was extraordinarily resistant to deep anaesthesia (Marshall, Woolsey and Bard, 1941[50]), it was unlikely that cortical neuronal action potentials make a significant direct contribution to such responses. More promising candidates for the role of direct contributors to the surface primary response were the post-synaptic potentials (Eccles, 1951[24]) and the "after potentials". The critical requirement was that there existed a difference in membrane potentials between spatially separated portions of the neurons for a period of many milliseconds. Central axons had a very prominent negative after-potential (Rudin and Eisenman, 1954[56]) as compared with peripheral axons. If the terminal arborisations of specific thalamo-cortical afferent fibres had an after-potential which was different in amplitude or in time course from that of the parent axon, they might directly contribute to the surface primary response. However, depth-reversal of the peak positivity of the primary response may occur above the site of specific thalamo-cortical afferent termination. Any contribution by after-potentials in specific thalamo-cortical afferent fibres to the surface positive response, was overshadowed by the contributions made by cortical neurons. Thus by elimination, some function of the

cortical neuronal membrane appeared to be the most important contributor to the surface primary response. The limited information available about the response of cortical neurons to the specific thalamo-cortical afferent input included demonstration of post-synaptic potentials of several microvolts amplitude which lasted for many milliseconds (Albe-Fessard and Buser, 1955[2]; Amassian et,al, 1955[7]; Li, 1961[44]). There was general agreement that when a microelectrode was progressively inserted into sensory cortex, the positive component of the primary response reversed in sign, but there was little agreement on the depth at which reversal occurred. The negative-going potential gradients of early portions of the surface positive component commenced at greater depths than did those of the later portions. Although the complexity of the system of current generators was evident, the pattern of depth-reversal suggested that deep portions of cortical neurons acted as "sources" to still deeper "sinks" soon after the arrival of the specific afferent inflow, but more superficial portions of cortical neurons, e.g. superficial to 250 μ, became sources after a delay of several milliseconds. It was of interest that the pattern of reversal of the surface positive component, following stimulation of ipsilateral somatosensory area,was qualitatively similar to that of the primary response, because as Lorente de No (1938[47]) showed, specific thalamo-cortical and cortico-afferent fibres had a different pattern of termination in the cerebral cortex. The use of electrical stimulation probably obscured the differences in mode of action of the two sets of

afferent fibres. Amassian et al[8] concluded that the surface positive component of the primary response was mainly attributable to current flowing toward the deeper layers from post-synaptic membranes in the superficial one-third of the cortex. Indirect evidence was secured that activity started migrating towards the surface during the surface positive component. The amplitude of the surface response was presumably a complex function of synaptic potentials and after-potentials in many cortical elements. A situation may be readily envisaged in which the amplitude of the surface response may decrease when the number of discharging neurons increases. Suppose a change in state of consciousness occurred such that the background level of excitatory bombardment of cortical neurons was increased. A test volley arriving at the cortex would then act upon membranes which were closer to the equilibrium potential of the excitatory post-synaptic potential. The net effect might be to increase the number of neurons which discharge but to reduce the amplitude of synaptic potentials and consequently to reduce the amplitude of the surface response. Similarly, a paradoxical relationship between neuronal discharge and externally recorded potential could readily occur with mixed excitatory and inhibitory afferent volleys. Such events probably occur and add thereby to the difficulty of interpreting evoked potentials both in the animal and in the human. In some recent work Storozhuk (1968[60]) stated that variability of latencies of

single unit responses could be due to differences in conduction velocity of impulses from periphery to cortex, evidence of which was given, in particular, by indentation on the descending limb of the positive phase of the primary response appearing under light anaesthesia. However, this scatter did not appear over more than 5 milliseconds. The positive phase of the primary response was due to depolarization of neurons lying at a depth of 800-1400μ, and the negative phase to hyperpolarization of the same neurons at deep levels and also, perhaps, to depolarization of apical dendrites of neurons in an inactive state.

Proprioceptive loss in Uttal and Cook's[61] experiments was associated with marked changes in the evoked response, while apparently normal records were obtained from patients with loss of pain and temperature perception only. In two of Giblin's[27] patients with unilateral lesions involving peripheral nerves or spinal roots, the response evoked by shocks to the affected limbs were merely smaller in amplitude than those evoked by shocks to the unaffected limb. In patients with the most severe neuropathy, shocks evoked neither afferent volleys nor cerebral responses. Others, with less severe mixed motor and sensory polyneuropathy did not have evoked potentials comparable to those of the healthy subjects. Results in eight patients with lesions of the spinal cord showed evoked responses that were normal in three, abnormal in four, and asymmetrical in one patient and the results correlated well with the type of sensory loss which each

patient showed. If, in considering evoked responses recorded in the patient with cerebral lesions, a decrease in the amplitude of the response is taken as the criterion of abnormality, then evoked responses corresponded with the results of clinical sensory examination in 34 of the 42 patients who were studied. In the remaining eight patients, however, the results were markedly discrepant. Evoked potentials were virtually absent from one hemisphere in one patient without sensory abnormality while, conversely, normal evoked potentials were recorded in seven of 17 patients who showed definite sensory loss. The results in patients with peripheral neuropathy indicated that, even when sensory dysfunction was minimal or when sensory loss was confined to the distal parts of the extremities, the majority of the large afferent fibres in peripheral nerves were found to be abnormal. This was indicated by an increase in their thresholds to electrical stimulation and a decrease in the velocity with which they conducted impulses. A good correlation was found, in patients with lesions of the spinal cord, between changes in evoked cerebral response and the type of sensory loss. The correlation was best with the sense of position and passive movement at joints and was such that evoked potentials were abnormal if this modality was more than minimally impaired. This result has since been confirmed by Halliday and Wakefield (35) (1962). Alajouanine et al (1958) (1) recorded evoked responses in patients with cortical lesions, thalamic syndrome, brain stem or spinal cord lesions, and root lesions. Only amplitude was well determined because the limitations of their recording technique prevented a complete study on latencies, waveform and spatial display of the evoked responses. In none of these cases could an evoked ptential of normal amplitude be observed.

The greater the peripheral sensory deficit, the lower the amplitude of the evoked potential until in extreme deficits no response could be observed. Bergamini et al (1967[9]) noticed that when the lesion was in the medullary level, only posterior column damage induced modifications or disappearance of the somatosensory response evoked by electrical stimulation of the peripheral nerve. It was never abolished by even total lesions of the antero-lateral columns. It was evident from this that the impulses cuasing the somatosensory evoked potential in the cortex followed almost exclusively Goll and Burdach's columns in the spinal cord. The investigations of Halliday and Wakefield (1962[35],1963[36]) and Halliday (1965[34]) confirmed the results obtained by Giblin[27]: the cortical responses appeared to be only slightly modified in cases of tactile or thermal sensory loss, but they showed longer latencies and clear amplitude decrease when there was a sensory deficit of deep origin. "It may be concluded then that a sensory fibre lesion and damage of root and posterior columns are two pathological conditions which remarkably alter somatosensory potentials evoked by nerve electrical stimulation." (Bergamini et al, 1967[9]). These two conditions were differentiated by characteristics of their evoked responses. A lesion of the afferent peripheral fibre reduces its excitability and provokes a temporal dispersion of the afferent volley (Gilliatt and Sears, 1958[29]). This causes a decrease in the peripheral afferent conduction velocity, which expresses itself in the cortex by the increased evoked potential latencies. At the same time, the cortical evoked potential will have a longer duration

accompanying the temporal dispersion of the impulses arising from the periphery. The evoked potential behaviour, however, was different in posterior column and root lesions (Bergamini et al 1967[9]). In these lesions the evoked response was characterized by a profound morphology alteration; the earlier part, which very often could not be recorded, seemed to be constantly more and more modified. When a limb nerve was stimulated in cases which showed a clear clinical deficit of the proprioceptive sensitivity, no somatosensory cortical potential could be obtained. On this basis, the simultaneous determination of the peripheral afferent conduction velocity and of the somatosensory cortical evoked potential has made possible the electrophysiological determination of the elective level of lesions in different groups of abiotrophic-degenerative diseases of the peripheral nervous system (Bergamini et al 1966[10]) such as Charcot-Marie-Tooth-Hoffman disease, Friedreich's disease, tabes dorsalis and Adie's syndrome. In the first disease, although the afferent nervous conduction was greatly altered, the cortical evoked response appeared to be only slightly modified indicating a lesion of one peripheral afferent nerve. In Friedreich's disease both the nerve action potential and the cortical responses were greatly altered indicating a lesion of the peripheral afferent fibres and of the posterior medullary columns. In tabes dorsalis and in Adie's syndrome an almost normal peripheral afferent conduction accompanied pronounced modification of evoked responses, which very often could not be recorded, signifying only posterior column lesions. In the peripheral sensory "functional" lesions the somatosensory evoked potential was modified. Alajouanine et al (1958[1]) found no somatosensory evoked response modification in seven cases

of hysteric anaesthesia and in a few cases of congenital sensory loss. Giblin[27] concluded that, whatever may be the functional role of the operations of the nervous system which are manifest as a complex series of potentials recorded during the 100 msec. period following the application of a stimulus, they are clearly not sufficient for perception, even in an alert subject, although they may well be necessary.

INTRODUCTION

The preliminary work of finding and describing the somatosensory evoked potentials, having been accomplished, an attempt was made to bring the recent research in this area to a level such that it could be used to aid clinical diagnosis. As mentioned by many authors (1, 9, 10, 27, 34, 47 and 36) the configuration of the evoked potential could be used to help in the diagnosis of sensory fiber lesion and damage of root and posterior columns, However, no effort has been made so far to determine the sensory nerve conduction velocity using the latency of the evoked potential as the indicator of the arrival of a sensory stimulus at the cortex. If this could be accomplished, the results would be of value in testing the whole sensory pathway from the distal limb to the central terminations at the postcentral gyrus. Electromyographic standard tests only measure afferent nerve conduction velocities and distal antidromic or orthodromic sensory nerve latencies.

Consequently, the purpose of this study was to -:

(a) produce reliable, repeatable, and clear somatosensory evoked potentials.

(b) find a point on the beginning of the evoked potential from which to measure latencies.

(c) determine sensory nerve conduction velocities for a normal population.

(d) apply this test to abnormal patients with sensory nerve abnormalities and prove the import of the sensory nerve conduction velocity as a clinical aid to diagnosis.

METHOD

34 student nurses and 6 male university students were used as normal subjects. The patients were allowed to have their eyes open or closed, their mouth open and a rolled sheet was placed under the back of the neck in order to reduce scalp muscle activity.

On the head were placed five gold plated E.5.G. Grass electrodes, 9 mm. in diameter, filled with Redux electrode paste and fastened with Collodion as in Figure 1, such that the active electrodes were over the "hand" area of the postcentral gyrus. The inter electrode impedance was reduced to the order of 3KΩ by inserting a blunted hypodermic needle through the holes in the centres of the electrodes and scraping the surface of the skin lightly. The polarity was such that a potential recorded by the electrode over the postcentral gyrus, negative with respect to the references, caused an upward deflection in the final record.

The median and ulnar nerves were stimulated electrically at the wrist and elbow with the cathode 3 cm. proximal to the anode. Redux electrode paste was used to facilitate conduction of the stimulus. Xylocaine paste, a topical anesthetic was used to decrease the pain caused by the stimulus but was found ineffective. For stimulation of the median nerve, a pickup electrode, which could monitor the resulting compound action potential, was placed at the centre of the thenar eminence and the reference electrode was placed at the base of the first phalanx of the thumb. The ground electrode was placed on the palmar surface of the hand.

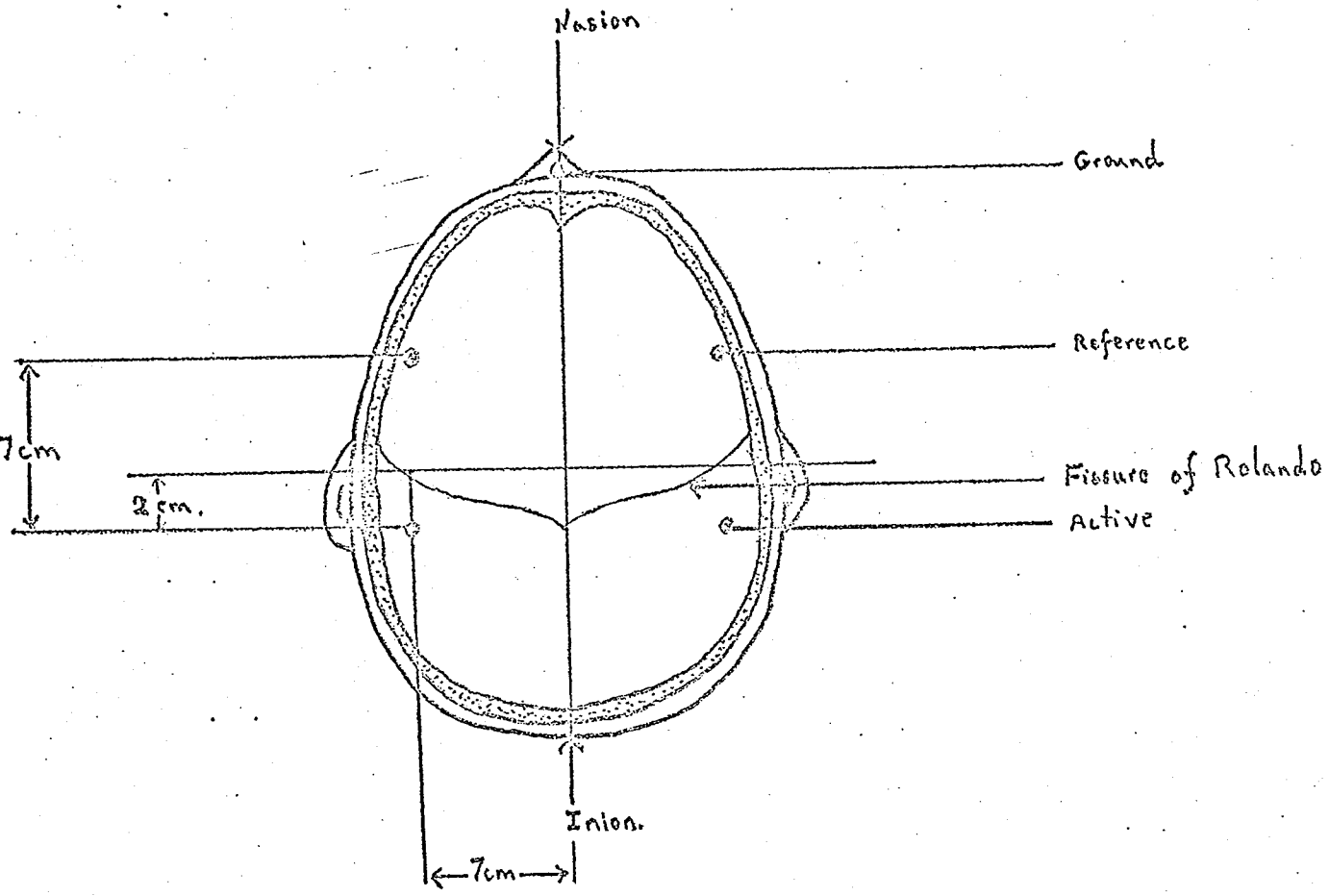


Figure 1.

Position of electrodes on the head for recording somatosensory evoked potentials after stimulation of the ulnar and median nerves.

To stimulate the median nerve, the stimulating electrode was placed 6 cm. above the elbow on the medial surface of the upper arm. The anode was placed in a position distal to the cathode. To stimulate the nerve at the wrist, the cathode was placed between the tendons of the palmaris longus and the flexor carpi radialis muscles, just proximal to the transverse carpal ligament. The anode was usually in a radial and distal position relative to the cathode in order to avoid simultaneous stimulation of the ulnar nerve. For stimulation of the ulnar nerve, the pickup electrode was placed midway along the lateral border of the hypothenar eminence and the reference electrode at the base of the proximal phalanx of the fifth finger. The ground electrode was placed on the dorsum of the wrist. For stimulation at the elbow, the stimulating cathode was placed 3 cm. above the ulnar notch and medical epicondyle. The anode was placed posterior and distal to the cathode. At the wrist the cathode was placed just medial to the tendon of the flexor carpi ulnaris muscle at about the same level as indicated for the median nerve. Here the anode was placed on the ulnar side of the nerve to avoid simultaneous stimulation of the median nerve.

The stimulus was of 100μsec duration and of sufficient strength to cause parasthesiae distally such that a compound action potential could be monitored from the electrodes on the hand. A Grass S5 stimulator was used, delivering 100 pulses, at the rate of 1 per 2 seconds, for each evoked potential recorded. Later, a stimulator of our own design was used which produced a stimulus delayed by 8 msec. after commencement of the averaging cycle.

The electrical activity from the scalp electrodes was fed through a Grass HIP 511 High Impedance Probe to a Grass P 511 Preamplifier set at a decay time of 100 msec. and a rise time of 0.3 msec. and amplified 100,000 times. One of the two outputs of the amplifier was monitored by one channel of a Type 502 Tektronix Dual Beam Oscilloscope set at a vertical sensitivity of 0.5 v/cm and sweep time of 10 msec./cm. This was done to ensure that the patient was relaxed and that there was no high amplitude muscle activity from the scalp. The other output of the amplifier was fed to a line driver, and then to an Analog-Digital Converter of a Computer.

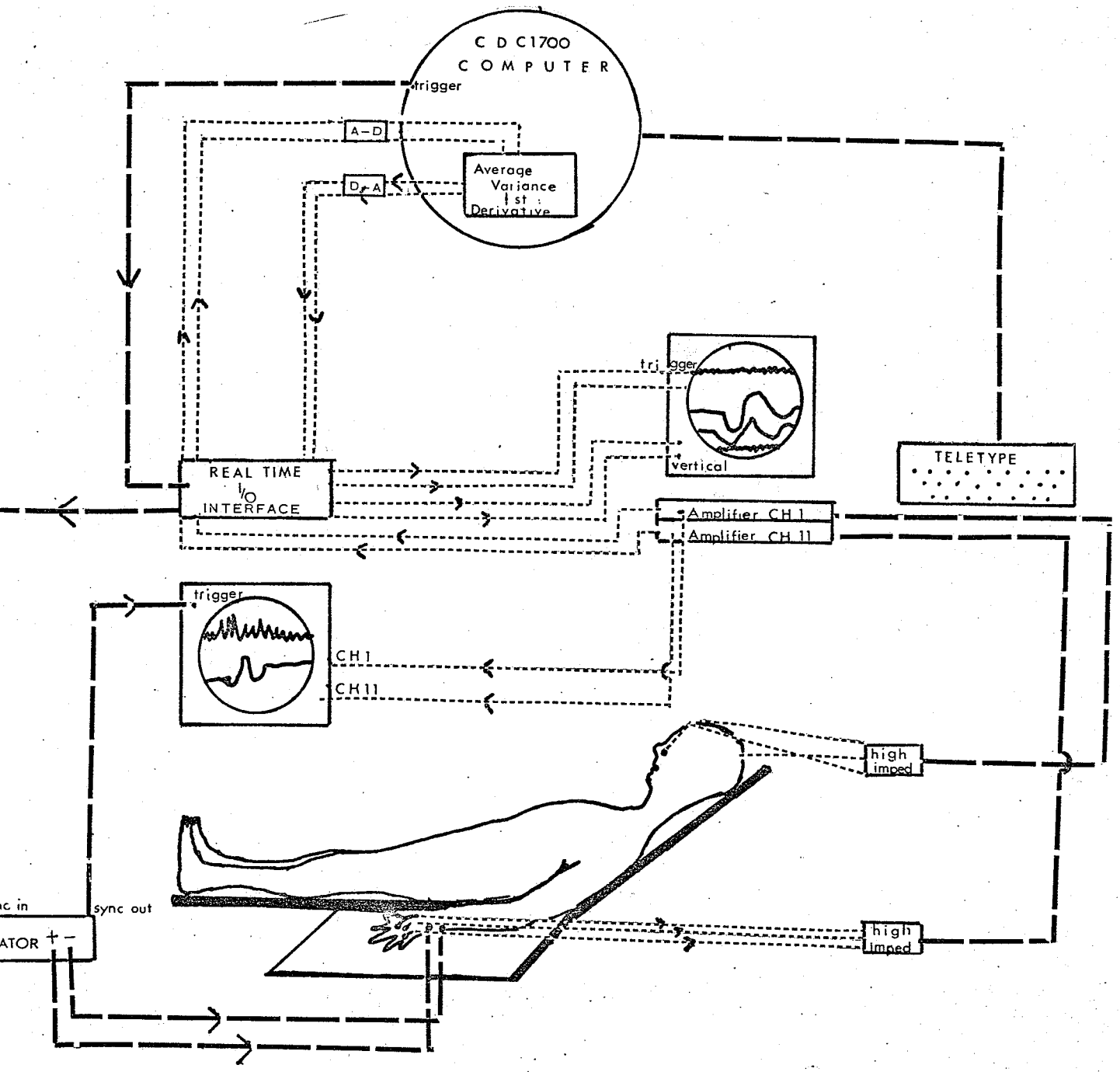
The compound action potential from stimulation of the nerve, monitored by the electrodes on the hand, was fed through another Grass HIP 511 High Impedance Probe to a Grass P 511 Preamplifier set at a decay time of 100 msec. and a rise time of 0.3 msec. and amplified 100,000 times. The out-put was monitored by the second channel of the Type 502 Tektronix Dual Beam Oscilloscope set at a vertical sensitivity of 0.2 v/cm. and sweep time of 10 msec./cm. The compound action potential was monitored to ensure that the stimulus was being delivered directly to the nerve.

The two Grass P 511 Preamplifiers were powered by a Grass RPS 106 Regulated Power Supply.

The amplified signals from the scalp were passed through the line driver along a 1300 foot cable to an analog to digital converter of a Control Data Corporation 1700 Computer which averaged the information for 30 msec. every 2 seconds for 100 events. The computer also calculated the variance of the background noise,

FIGURE 3

Line Diagram of Equipment Used for Averaging of Somatosensory Evoked Potentials, Calculation of Variance of Background Noise and Calculation of First Derivatives of the Somatosensory Evoked Potentials.



calculated the first derivative of the averaged somatosensory evoked potential, and provided the trigger for the stimulator. Further explanation of averaging and calculation of the variance and first derivative will be found in the discussion. The information was passed from the digital to analog converter of the computer, through the cable to the Real Time Output Interface and displayed upon a Type 503 Tektronix single Beam Oscilloscope set at a vertical sensitivity of 1 v/cm.

Two channels of information could be processed together or consecutively and displayed together. The program for the averaging of evoked potentials was called by means of a Teletype. The stimulus interval, sweep time, number of averaging cycles, channel identification number (channel 1) and commencement were typed on the teletype. The stimulus was then delivered to the wrist and the somatosensory evoked potential on the contralateral "hand" area of the postcentral gyrus was averaged. At the same time the variance of the background noise was calculated and displayed on the single beam oscilloscope. At the end of 100 stimuli, the computer ceased averaging and ceased triggering the stimulator. The stimulating electrode was moved to the elbow and the parameters were again typed into the computer using the second channel (channel II) of the computer program. The final display was photographed by a Tektronix Oscilloscope Camera C-12 using Polaroid 3000 Speed/Type 107 Black and White film. The command for first derivatives of the evoked potentials was given to the computer and the first derivatives were photographed (see Fig.4-7 for variances, averages, and derivatives). The distance between the two stimulating cathode positions was measured by a "Map Measurer" following the path of the nerve as much as possible.

The difference in latencies of the two evoked potentials was measured and divided by the distance between the two stimulating positions to give the nerve conduction velocity in meters per second.

The above method was repeated for the peroneal and posterior tibial nerves with the following differences. 3 electrodes were attached to the head (Figure 2). The active electrode was placed 2 cm. posterior to the vertex, the reference electrode was placed 7 cm. anterior to the active electrode and the ground electrode was attached to the bridge of the nose. This positioning of the electrode enabled the evoked potential to be recorded when either leg was stimulated. The response of the peroneal nerve was recorded from the extensor digitonum brevis muscle. A pickup electrode, which could monitor the compound action potential resulting from stimulation, was placed over the most prominent portion of the muscle with the reference electrode near the outer edge of the foot. The ground electrode was placed over the dorsum of the foot. To stimulate the peroneal nerve at the knee, the cathode was placed inside the lateral border of the popliteal fossa medial to the head of the fibual. The position of the anode, was distal to that of the cathode. Stimulation of the peroneal nerve at the ankle was just lateral to the tendon of the long toe extensor, slightly below the level of the ankle (lateral maleolus). For stimulation of the posterior tibial nerve at the knee, the cathode of the stimulator was placed in the central portion of the popliteal space with the anode distal in position. Stimulation of the tibial nerve at the ankle was just behind the medial malleolus. The pickup electrode was placed at the base of the fifth metatarsal bone on the plantar surface of the foot with the reference electrode on the lateral aspect of the fifth toe. The

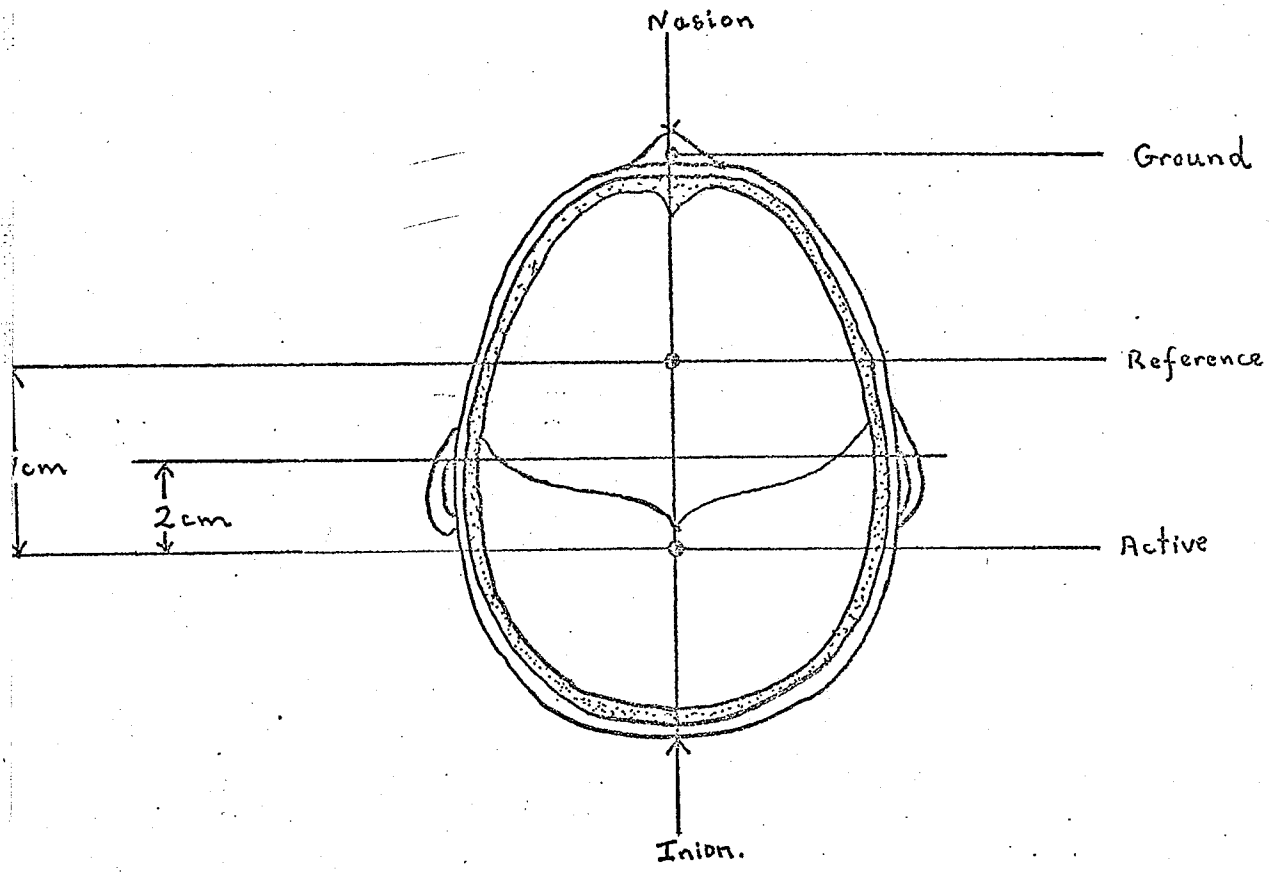


Figure 2.

Position of electrodes on the head for recording somatosensory evoked potentials after stimulation of the peroneal and posterior tibial nerves.

ground was placed on the plantar surface of the foot.

To stimulate the nerves in the leg a higher voltage than that produced by the Grass S5 was required, so that this stimulator was replaced by a current regulated stimulator of our own design producing 120 v maximum. This stimulator produced a stimulus delayed by 8 milliseconds after commencement of the averaging cycle. All other parameters were the same as for the median and ulnar nerve except that a 50 millisecond sweep and an amplification of 200,000 times was used.

Skin temperature was measured by means of a Yellow Springs Instrument Co., Inc. Tele-Thermometer with a thermistor taped to the surface of the arm or leg halfway between the two stimulating positions.

Calibration was performed by passing a calibration signal through the High Impedance probes from a Grass SWCIB Square Wave Calibrator.

To determine whether the initial waves of the evoked potential could be recorded elsewhere on the head, electrodes were placed at the inion (active) with one on the mastoid as a reference, and in the standard positions for stimulation of the legs and arms. While one of the nerves was being stimulated the responses from two pairs of electrodes were amplified by both amplifiers 100,000 times and monitored by the two channels of the Dual Beam Oscilloscope. The computer averaged the responses on both channels together. This was done for three patients using the left median nerve at the wrist, the left peroneal nerve at the ankle, and the right ulnar at the wrist as stimulus positions.

For calibration of the variance an Automation Laboratories Inc. Low Frequency Gaussian Noise Generator was set at an output of

approximately 1.73 v rms. and connected to the external sine wave input of the Grass SWCIB Square Wave Calibrator such that the square wave calibrator produced Gaussian Noise at approximately 1μv to 50μv depending upon the setting of the voltage control on the calibrator. A graph of the Gaussian Noise Level against variance in mm at 1v/cm sensitivity on the Single Beam Oscilloscope was plotted for amplifications of 100,000 times and 200,000 times (Figure 8). From this graph it was possible to estimate the background noise level.

It was originally hoped that the variance would show peaks of jitter of the evoked potential across time and would thus provide an accurate marker for the beginning or early parts of the evoked potential. However, what was produced was approximately a straight line rising above the baseline in accordance with the amplitude of noises. An attempt to introduce a false jitter was made by passing the stimulus through a flip-flop circuit to two pairs of electrodes and consequently stimulating at two positions about 3 cm. apart alternately. It was thought that this would jitter the evoked potential across time. However the variance failed to show any peaks and the method of using the variance for a marker was abandoned.

The peak of the first derivative of the first down sweep of the first positive wave of the evoked potential was found to be a good marker for commencement of the evoked potential. Where

possible, the beginning of the evoked potential or the first negative peak were used as markers for latency measurements.

In one test, the distance between the stimulating positions was measured ten times in order to estimate the standard deviation of the distance measurement. Similarly, the differences in latencies of the evoked potentials and first derivatives were measured ten times. This was done for each nerve and the results were outlined in Table X.

The tests for estimating nerve conduction velocities was applied to one patient eight times to determine the consistency of the results.

Three statistical tests were performed on the results in order to determine whether the velocities were independent, random, and normally distributed. Tests of skewness and kurtosis were conducted. The data was plotted on probability paper (3) and the resulting line was tested for linearity by regression analysis. This was done for the data from the left side and from the right side and for the accumulated data of right and left sides together. A chi-square test was performed on the frequency distribution of the accumulated data also.

Finally, three patients, A, B, and C, were tested for nerve conduction velocities by the above method. Patient A had a lesion of the spinal cord at T_{10-11}. Patient B was suffering from a brain stem vascular lesion and patient C was a case of diffuse polyneuropathy involving all four limbs.

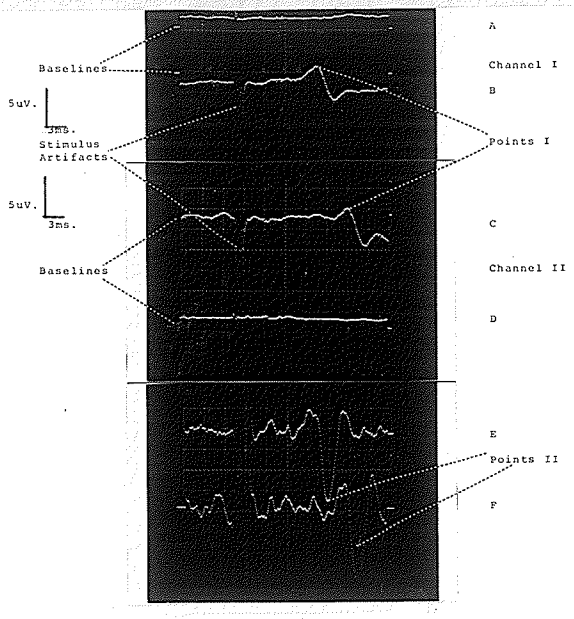


Figure 4

The variance A, evoked potential B, after stimulation of the right median nerve above the elbow (Channel I), the evoked potential C, and variance D, after stimulation of the right median nerve at the wrist (Channel II), and the first derivatives, E and F, of the evoked potentials B and C respectively.

Noise level Channel I ... 3 mm = 20.5uV
Noise level Channel II .. 2 mm = 15.8uV

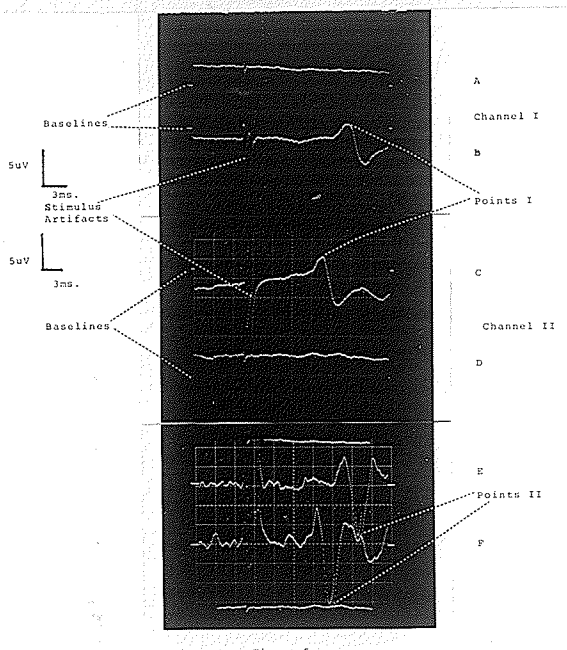


Figure 5.

The variance,A, evoked potential,B, after stimulation of the right ulnar nerve at the wrist (Channel I), the evoked potential,C, and variance, D, after stimulation of the right ulnar nerve above the elbow (Channel II), and the first derivatives, E and F, ofthe evoked potentials B and C respective -ly.

Noise level Channel I....4mm = 25uV.
Noise level Channel II...5mm = 28.5uV.

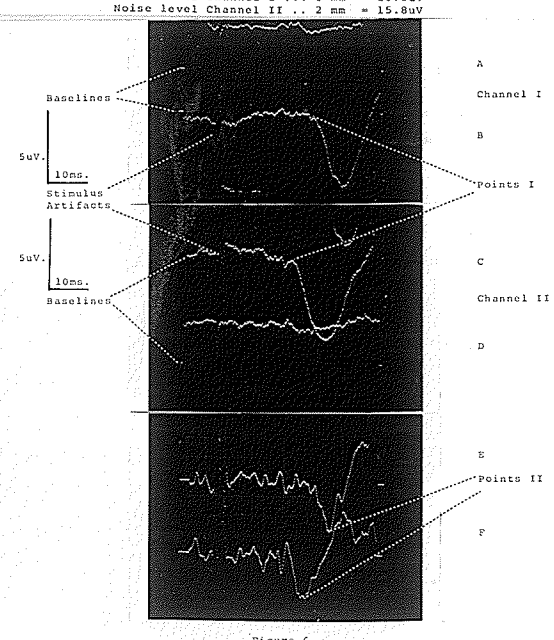


Figure 6.

The variance,A, evoked potential,B, after stimulation of the left peroneal nerve at the ankle (Channel I), the evoked potential,C, and variance,D, after stimulation of the left peroneal nerve at the knee(Channel II), and the first derivatives,E and F, of the evoked potentials,B and C, respectively.

Noise level Channel I....11mm =22.5uV.
Noise level Channel II...10mm =21.0uV.

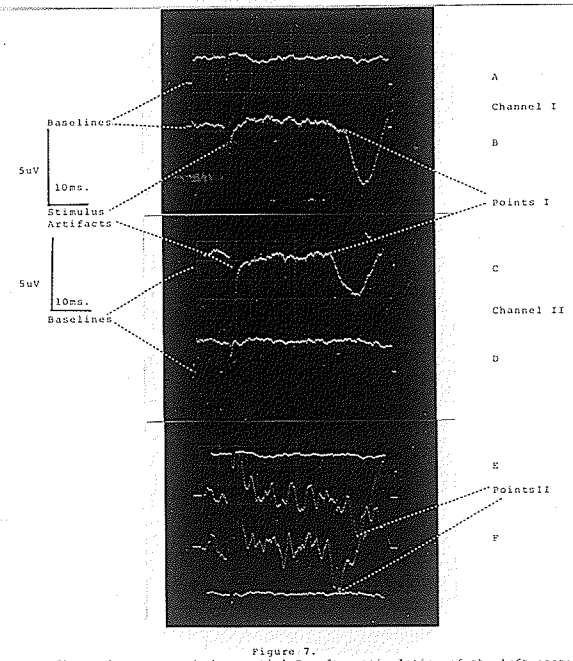


Figure 7.

The variance,A, evoked potential,B, after stimulation of the left posterior tibial nerve at the ankle (Channel I), the evoked potential,C, and variance,D, after stimulation of the left posterior tibial nerve at the knee (Channel II), and the first derivatives, E and F, of the evoked potentials, B and C, respective-ly.

Noise level Channel I....8mm = 27.8uV.
Noise level Channel II...8mm = 27.8uV.

Figure 8.

Graph of Gaussian noise level in uv. versus the variance measured in mms.

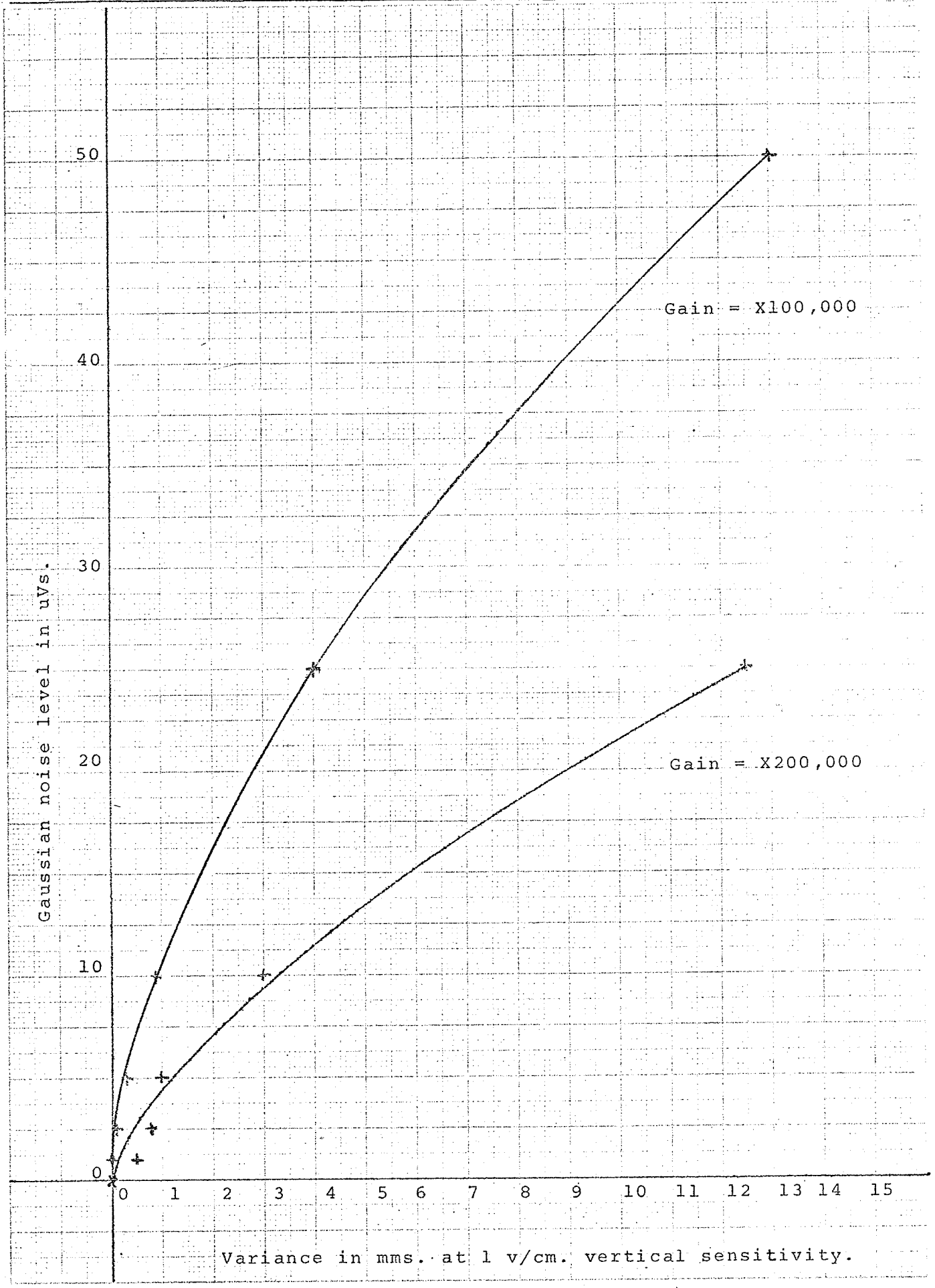


TABLE I

LEFT ULNAR NERVE

Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
3	25.0	3.6	3.6	69.4	69.4
4	28.0	3.3	3.9	84.8	71.8
5	22.0	3.3	3.3	66.7	66.7
6	32.0	4.5	4.5	71.1	71.1
7	28.0	4.2	4.2	66.7	66.7
9	30.0	4.5	4.8	66.7	62.5
10	28.0	4.5	4.5	62.2	62.2
12	30.0	3.3	3.3	90.9	90.9
13	29.0	3.6	3.9	80.6	74.4
14	25.0	3.3	3.6	75.8	69.4
15	27.0	3.6	4.2	75.0	64.3
20	31.0	4.2	4.2	73.8	73.8
21	34.0	4.2	4.8	81.0	70.8
22	28.0	4.5	4.5	62.2	62.2
42	29.0	4.2	3.6	69.0	80.6
43	29.5	4.2	4.2	70.2	70.2

RIGHT ULNAR NERVE

Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
1	23.0	4.2	-	54.8	-
2	32.0	4.5	4.5	71.1	71.1
3	26.0	3.3	3.6	78.8	72.2
4	25.0	3.6	3.6	69.4	69.4
5	24.0	3.0	3.3	80.0	72.7
6	32.0	4.2	4.8	76.2	66.7
7	29.0	3.6	4.2	80.6	69.0
8	27.0	3.0	-	90.0	-
9	30.0	4.2	4.2	71.4	71.4
10	27.5	3.9	3.9	70.5	70.5
12	28.0	3.3	3.3	84.8	84.8
13	30.0	4.2	4.2	71.4	71.4
14	25.0	3.9	3.9	64.1	64.1
15	27.0	3.0	4.5	90.0	60.0
20	31.0	4.2	4.5	73.8	68.9
21	30.0	4.8	4.8	62.5	62.5
22	27.0	3.0	4.2	90.0	64.3
42	30.0	3.9	3.9	76.9	76.9
43	28.0	3.9	3.9	71.8	71.8

ble I showing the sex of the patient (M=male, F=female), the number of the tient, the distance between the two points of stimulation, the difference latencies measured from Points I (Latency I) and from Points II (Latency II) d the velocities determined by dividing the distance by the latency.

TABLE II

LEFT MEDIAN NERVE

Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
9	30.0	3.6	3.6	83.3	83.3
11	31.0	4.5	4.8	68.9	64.6
12	30.0	3.3	3.6	90.9	83.3
13	34.0	3.7	4.2	91.9	81.0
14	26.5	3.0	3.6	88.3	73.6
15	27.0	3.6	3.9	75.0	69.2
20	38.0	4.2	4.2	90.5	90.5
21	36.0	4.8	5.4	75.0	66.7
22	26.0	3.3	3.6	78.8	72.2
36	30.5	3.6	3.6	84.7	84.7
37	33.5	4.2	4.2	79.8	79.8
38	32.0	4.8	3.9	66.7	82.1
39	34.5	3.6	3.9	95.8	88.5
42	30.0	3.6	3.9	83.3	76.9
43	33.0	3.6	4.5	91.7	73.3

RIGHT MEDIAN NERVE

Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
1	23.0	2.4	-	95.8	-
8	22.0	3.0	-	73.3	-
9	30.0	3.0	3.3	100.0	90.9
11	33.0	4.2	4.8	78.6	68.8
12	31.5	3.6	3.6	87.5	87.5
13	34.0	4.0	4.2	85.0	81.0
14	26.5	3.0	3.6	88.3	73.6
15	28.0	3.3	3.9	84.8	71.8
17	30.5	3.6	3.6	84.7	84.7
20	33.0	3.6	3.6	91.7	91.7
21	33.0	4.5	5.4	73.3	61.1
22	27.0	4.2	4.2	64.3	64.3
36	30.0	3.6	3.3	83.3	90.9
37	34.0	4.5	4.2	75.6	81.0
38	32.0	4.5	4.2	71.1	76.2
39	36.5	4.2	4.2	86.9	86.9
42	32.0	3.6	3.9	88.9	82.1
43	31.5	3.9	4.2	80.8	75.0

le II showing the sex of the patient (M=male, F=female), the number of the ient, the distance between the two points of stimulation, the difference latencies measured from Points I (Latency I) and from Points II (Latency II), the velocities determined by dividing the distance by the latency.

TABLE III

LEFT PERONEAL NERVE

Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
12	42.0	8.0	7.0	52.5	60.0
15	42.0	7.8	8.0	53.8	52.5
16	37.0	7.2	6.0	51.4	61.7
17	44.0	4.5	5.5	97.8	80.0
18	45.0	7.0	8.0	54.3	56.5
23	39.0	-	4.0	-	97.5
24	40.5	-	7.5	-	54.0
25	43.0	5.5	4.5	78.2	95.6
26	41.0	5.5	8.1	74.5	50.6
27	40.0	6.5	7.3	61.5	54.8
28	43.0	9.0	7.0	31.1	61.4
29	43.0	12.5	8.0	34.4	53.8
30	39.0	8.0	8.9	48.8	43.8
31	45.0	9.0	8.9	50.0	50.6
32	41.0	6.0	7.0	68.3	58.6
33	43.0	7.0	7.8	61.4	55.1
34	38.0	5.5	8.0	69.1	47.5
35	40.0	7.2	7.5	55.6	53.3
40	41.0	6.8	8.5	60.3	48.2
41	43.0	7.0	8.0	61.4	53.8

RIGHT PERONEAL NERVE

Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
12	40.0	7.0	7.5	57.1	53.3
15	42.0	6.5	7.0	64.6	60.0
16	38.0	7.1	7.0	53.5	52.9
17	43.0	5.0	5.0	86.0	86.0
18	41.0	5.0	8.0	82.0	51.3
24	41.5	-	7.5	-	55.3
25	43.0	5.0	4.5	86.0	95.6
26	41.0	8.5	8.0	48.2	51.3
27	39.0	7.8	7.0	50.0	55.7
28	41.0	9.0	6.0	45.6	68.3
29	46.0	10.3	8.0	44.7	57.5
30	40.0	8.0	8.0	50.0	50.0
31	45.0	8.0	8.0	56.3	56.3
32	41.0	6.0	8.0	68.3	51.3
33	40.0	9.0	8.3	44.4	48.2
34	37.0	5.0	8.0	74.0	46.3
35	41.0	7.7	8.5	53.2	48.2
40	39.0	6.0	8.5	65.0	45.9
41	44.0	-	8.0	-	55.0

ble III showing the sex of the patient (M=male, F=female), the number of the tient, the distance between the two points of stimulation, the difference in tencies measured from Points I (Latency I) and from Points II(Latency II), d the velocities determined by dividing the distance by the latency,

TABLE IV

LEFT POSTERIOR TIBIAL NERVE

x	Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
	12	34.0	5.0	5.5	68.0	61.8
	15	37.0	11.0	11.0	33.6	33.6
	17	38.0	6.0	6.5	63.3	58.5
	18	38.0	7.0	6.0	54.3	63.3
	30	37.0	5.0	6.0	74.0	61.7
	32	41.0	7.0	7.0	58.6	58.6
	34	36.0	6.0	7.0	60.0	51.4
	35	35.0	7.3	7.0	47.9	50.0
	37	38.5	7.0	8.0	55.0	48.1
	38	36.0	4.0	5.0	90.0	72.0
	39	43.0	4.8	5.2	89.6	82.7
	40	37.0	8.2	8.0	45.1	46.3
	41	40.0	7.5	6.0	53.3	66.7

RIGHT POSTERIOR TIBIAL NERVE

x	Patient No.	Distance in cms.	Latency I in msecs.	Latency II in msecs.	Velocity I in m/sec.	Velocity II in m/sec.
	12	35.0	4.0	5.0	87.5	70.0
	15	35.0	9.0	9.0	38.9	38.9
	17	37.0	7.0	7.0	52.9	52.9
	18	38.0	5.0	6.0	76.0	63.3
	25	42.0	4.5	4.1	93.3	102.4
	30	38.0	6.5	6.5	58.5	58.5
	31	41.0	7.0	7.8	58.6	52.6
	32	39.0	9.0	6.0	35.6	65.0
	34	36.0	7.0	8.0	51.4	45.0
	35	37.0	5.0	6.5	74.0	56.9
	36	35.0	6.0	6.5	58.3	52.8
	37	38.0	7.0	8.0	54.3	47.5
	38	36.0	4.5	6.0	80.0	60.0
	39	43.0	4.2	5.2	102.4	82.7
	40	37.0	61.	8.0	60.7	46.3
	41	39.5	6.0	5.5	49.4	71.8

le IV showing the sex of the patient (M=male, F=female), the number of the
ient, the distance between the two points of stimulation, the difference
latencies from Points I(Latency I) and from Points II(Latency II), and
velocities determined by dividing the distance by the latency.

TABLE V

NERVE	MEAN	S.D.	SKEWNESS	KURTOSIS	LINEAR REGRESSION			FREQUENCY DISTRIBUTION		
					T	R	R^2	χ^2	D.F.	$\chi^2_{0.05}$
L. Ulnar 1	72.9	8.1	0.621	2.376	8.9	0.95	0.91			
L. Ulnar 2	70.4	7.4	1.205	4.232	20.1	0.99	0.98			
R. Ulnar 1	75.2	9.5	-0.080	2.368	5.3	0.88	0.78			
R. Ulnar 2	69.9	5.7	0.620	3.667	15.0	0.98	0.97			
Ulnar 1	74.1	8.9	0.223	2.432	22.2	0.99	0.98	2.01	3	7.815
Ulnar 2	70.1	6.5	1.115	4.734	8.2	0.95	0.89	4.39	2	5.991
L.Median 1	83.0	8.8	-0.355	1.819	4.5	0.85	0.72			
L.Median 2	78.0	7.8	-0.146	1.723	5.1	0.87	0.76			
R.Median 1	83.0	9.1	-0.172	2.331	6.9	0.93	0.86			
R.Median 2	79.2	9.6	-0.338	1.819	4.2	0.83	0.69			
Median 1	82.9	8.9	-0.243	2.175	27.1	0.99	0.99	1.70	3	7.815
Median 2	78.6	8.6	-0.248	1.948	23.1	0.99	0.99	4.22	3	7.815
L. Peroneal 1	58.7	15.4	0.356	3.327	13.0	0.98	0.96			
L. Peroneal 2	59.5	14.7	1.604	4.423	6.1	0.91	0.82			
R. Peroneal 1	60.5	14.3	0.592	1.859	5.1	0.88	0.77			
R. Peroneal 2	57.3	13.0	1.813	5.338	8.9	0.95	0.91			
Peroneal 1	60.1	14.7	0.473	2.915	19.7	0.99	0.98	6.71	7	14.067
Peroneal 2	58.4	13.7	0.776	5.129	4.9	0.87	0.75	29.78	4	9.448

CONT'D

TABLE V (CONT'd)

NERVE	MEAN	S.D.	SKEWNESS	KURTOSIS	LINEAR REGRESSION			FREQUENCY DISTRIBUTION		
					T	R	R^2	χ^2	D.F.	$\chi^2_{0.05}$
L. Tibial 1	61.0	16.3	0.401	2.237	6.5	0.92	0.84			
L. Tibial 2	58.1	12.5	0.020	2.536	10.0	0.96	0.93			
R. Tibial 1	64.5	19.2	0.416	2.019	6.9	0.92	0.86			
R. Tibial 2	60.4	15.9	0.061	3.963	18.9	0.99	0.98			
Tibial 1	63.1	18.0	0.444	2.247	20.5	0.99	0.98	9.60	7	14.067
Tibial 2	58.4	14.2	0.967	4.068	16.3	0.99	0.97	3.77	6	12.592

Table V showing the means and standard deviations of sensory nerve conduction velocities measured using points I and II for the left and right sides, separately and together (i.e. Ulnar 1 - l. Ulnar 1 + R. Ulnar 1), and the statistics indicating the normal distribution of the data. S.D. = standard deviation ' D.F. = degrees of freedom.

TABLE VI

LEFT ULNAR-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
4	29.0	4.2	3.6	69.0	80.6
5	29.5	3.9	3.9	75.6	75.6
6	29.0	4.5	3.6	64.4	80.6
7	29.0	4.5	4.5	64.4	64.4
8	29.0	4.2	4.8	69.0	60.4
9	29.0	4.8	4.8	60.4	60.4
10	28.0	4.5	4.5	62.2	62.2
11	30.0	4.8	4.8	62.5	62.5
				Mean= 65.9	Mean= 68.3
				S.D.= 5.0	S.D.= 9.0

RIGHT ULNAR-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
4	30.0	4.2	4.2	71.4	71.4
5	30.0	4.2	4.2	71.4	71.4
6	29.0	3.9	3.6	74.4	80.6
7	29.0	3.9	3.9	74.3	74.3
8	28.5	3.6	3.9	79.2	73.1
9	29.0	3.6	3.9	80.6	74.4
10	29.0	4.2	4.2	69.0	69.0
11	28.0	3.6	4.2	77.8	66.7
				Mean= 74.8	Mean= 72.6
				S.D.= 4.1	S.D.= 4.2
			Acculumated Means =	70.4	= 70.5
			Accumulated S.Ds. =	6.4	= 7.1

Table VI showing the trial number, the distance between the two points of stimulation, the difference in latencies from Points I (Latency I) and from Point II (Latency II), the velocities determined by dividing the distance by the latency, the means and standard deviations (S.D.) of the velocities for right and left ulnar nerves and for the accumulated data from both sides. All data was collected from one subject (Patient Number 34.).

TABLE VII

LEFT MEDIAN-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
3	31.5	3.9	3.9	80.8	80.8
4	29.0	4.2	3.6	69.0	80.6
5	33.0	4.5	3.9	73.3	84.6
7	32.0	4.8	5.1	66.7	62.7
8	32.0	4.8	4.8	66.7	66.7
9	34.0	4.8	4.9	70.8	69.4
11	32.0	4.8	4.8	66.7	66.7
				Mean= 70.6	Mean= 73.1
				S.D.= 5.2	S.D.= 8.7

RIGHT MEDIAN-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
3	31.5	3.9	3.9	80.8	80.8
4	31.0	3.9	3.9	79.5	79.5
5	35.0	4.8	4.8	72.9	72.9
7	32.0	3.9	4.2	82.1	76.2
8	32.0	3.9	4.2	82.1	76.2
9	34.0	4.5	4.5	75.6	75.6
11	33.0	4.2	3.9	78.6	84.6
				Mean= 78.8	Mean= 79.0
				S.D.= 3.5	S.D.= 3.9
			Accumulated Means=	74.7	= 75.5
			Accumulated S.Ds.=	6.0	= 7.0

Table VII showing the trial number, the distance between the two points of stimulation, the difference in latencies from Points I (Latency I) and from Point II (Latency II), the velocities determined by dividing the distance by the latency, the means and standard deviations (S.D.) of the velocities for right and left median nerves and for the accumulated data from both sides. All data was collected from one subject (Patient Number 34.).

TABLE VIII

LEFT PERONEAL-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
1	38.0	5.5	8.0	69.1	47.5
2	39.5	5.0	5.0	79.1	79.0
3	37.0	5.0	5.5	76.0	67.3
4	38.0	5.0	5.0	76.0	76.0
5	36.0	7.0	7.0	51.4	51.4
6	37.0	6.0	5.0	61.7	74.0
9	37.0	7.0	9.0	52.9	41.1
10	38.0	5.5	7.0	69.1	54.3
				Mean= 66.7	Mean= 61.3
				S.D.= 10.4	S.D.= 14.5

RIGHT PERONEAL-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
1	37.0	5.0	8.0	74.0	46.3
2	38.0	5.0	5.0	76.0	76.0
3	38.0	5.0	6.0	76.0	63.3
4	37.0	5.0	5.0	74.0	74.0
5	36.0	6.8	6.5	52.9	55.4
6	38.0	5.5	6.5	69.1	58.5
9	37.0	6.5	6.0	56.9	61.7
10	37.5	7.0	7.5	53.6	50.0
				Mean= 66.6	Mean= 66.7
				S.D.= 10.3	S.D.= 10.5
			Accumulated Mean=	66.6	= 61.0
			Accumulated S.D.=	10.0	= 12.2

Table VIII showing the trial number, the distance between the two points of stimulation, the difference in latencies from Points I (Latency I) and fromPoint II (Latency II), the velocities determined by dividing the distance by the latency, the means and standard deviations (S.D.) of the velocities for right and left peroneal nerves and for the accumulated data from both sides. All data was collected from one subject (Patient Number 34.).

TABLE IX

LEFT POSTERIOR TIBIAL-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	Latency II in msec.	Velocity I in m/sec.	Velocity II in m/sec.
1	36.0	6.0	7.0	60.0	51.4
2	34.5	6.0	7.0	57.5	49.3
3	34.0	5.0	8.0	68.0	42.5
6	35.0	4.5	5.5	77.8	63.6
7	34.0	6.5	5.0	52.3	68.0
8	35.0	6.5	7.5	53.8	46.7
10	34.0	5.0	5.0	68.0	68.0
11	35.0	6.0	6.0	58.3	58.3
				Mean= 62.0	Mean= 56.0
				S.D.= 8.6	S.D.= 9.9

RIGHT POSTERIOR TIBIAL-CONSISTENCY

Trial	Distance in cms.	Latency I in msec.	LatencyII in msec.	Velocity I in m/sec.	Velocity II in m/sec.
1	36.0	7.0	8.0	51.4	45.0
2	34.0	7.0	6.5	48.6	52.3
3	35.0	6.0	8.0	58.3	43.8
6	34.5	8.0	6.5	43.1	53.1
7	35.0	6.0	7.0	58.3	50.0
8	35.0	5.5	8.0	63.6	43.8
10	35.0	65.	7.0	58.8	50.0
11	34.0	6.0	6.0	55.8	55.8
				Mean= 54.7	Mean= 49.2
				S.D.= 6.6	S.D.= 4.6
			Accumulated	Mean= 58.4	= 52.6
			Accumulated	S.D.= 8.3	= 8.2

Table IX showing the trial number, the distance between the two points of stimulation, the difference in latencies from Points I (Latency I) and from Point II(Latency II), the velocities determined by dividing the distance by the latency, the means and standard deviations (S.D.) of the velocities for right and left posterior tibial nerves and for the accumulated data from both sides. All data was collected from one subject (Patient Number 34.).

TABLE X

ERROR ESTIMATE OF MEASUREMENT

ULNAR NERVE			MEDIAN NERVE		
Latency I in msec.	Latency II in msec.	Distance in cms.	Latency I in msec.	Latency II in msec.	Distance in cms.
4.4	4.5	29.0	4.2	4.2	32.0
3.9	4.4	30.0	4.8	3.9	34.0
3.6	4.4	30.5	4.2	4.2	32.0
4.2	4.4	29.0	4.3	4.6	32.0
4.3	4.2	29.0	4.2	4.2	35.0
4.0	4.3	29.0	4.0	4.3	32.0
3.9	4.2	29.0	4.7	4.3	32.0
3.9	4.5	32.0	4.5	4.2	31.5
3.7	4.6	31.0	4.3	4.2	32.0
3.6	4.2	29.0	4.5	4.3	32.5
ean 3.95	4.37	29.75	4.37	4.24	32.5
.D. 0.28	0.14	1.09	0.25	0.17	1.11
.D. 0.56	0.28	2.18	0.50	0.34	2.22

PERONEAL NERVE			TIBIAL NERVE		
Latency I in msec.	Latency II in msec.	Distance in cms.	Latency I in msec.	Latency II in msec.	Distance in cms.
5.7	6.0	42.5	5.3	5.4	38.5
7.1	7.0	44.0	5.0	5.2	39.0
7.0	7.0	42.0	4.0	5.1	39.0
6.7	7.5	42.0	5.0	5.5	40.0
6.0	6.7	42.0	4.9	5.0	39.0
7.1	7.0	43.0	5.1	5.2	40.0
6.9	7.2	42.5	5.9	5.1	39.0
6.8	7.5	45.0	6.0	5.3	39.0
5.8	7.0	42.0	5.0	5.5	40.5
5.2	6.9	42.5	4.5	5.1	39.5
ean 6.43	6.98	42.75	5.07	5.24	39.35
.D. 0.69	0.43	1.01	0.59	0.18	0.63
.D. 1.38	0.86	2.02	1.18	0.36	1.26

Table X showing the difference in latency of the evoked potential (Latency I) the difference in latency of the first derivatives (Latency II), and the distance between the two stimulating points measured ten times for each nerve. The mean, standard deviation and 2x standard deviation are shown beneath each column.

Abnormal Subjects:

Patient A was a 57 year old female who on admission complained of numbness, tingling, pain, twitching and weakness in the legs. The legs were markedly paretic, with diminished tendon reflexes and bilateral Babinski signs. There was parasthesia and absent vibration sense in both legs. There was good movement in the left leg but only fair movement with weakness in the right leg. Eighteen months previously she had a CVA with right sided weakness and since then the legs had remained numb. She was operated upon for decompressive laminectomy and dural graft. The laminae in T10, T11, and T12 were greatly hypertrophied. There was also severe arachnoiditis secondary to compression of the spinal cord by osteophyte and thickened bone. Both left and right sensory nerve conduction velocities of the peroneal were slowed with a slightly slower velocity on the right side. The evoked potentials resulting from stimulation of these nerves were low in amplitude. Velocities were 37 m/sec. on the left and 32.2 m/sec. on the right. Sensory nerve conduction velocities of the left and right ulnar nerves were normal (75.8 m/sec.left, and 77.8 m/sec.right) but the amplitude of the somatosensory evoked potential resulting from stimulation of the right ulnar was markedly higher in amplitude than the amplitude of the evoked potential resulting from stimulation of the left ulnar nerve. These results suggest that damage was done to the spinal cord interrupting the sensory pathways from the legs but that these pathways still exist although they are delayed.

Patient B, was a 52 year old female with a brainstem vascular lesion and right hemiplegia. There was numbness of the right side of the face, tingling in the right arm, loss of power of the right arm and leg, right hemianesthesia, dense paralysis of the right arm and marked paresis of the right leg. The left side was normal. She recovered power on the right side within 24 hours of admission but opthalmoplegia remained with inability to move the left eye medially or laterally and deviation of the right eye to a lateral position. Total right hemianesthesia persisted. The blink reflex test, a test of the central connections of the trigeminal to facial reflex (Kimura, 1969[41]) was abnormal implicating brainstem involvement which appeared to be very irregular in distribution. The early as well as the late reflexes were absent when the right side was stimulated but the masseter reflex was absent on the left side. This suggested a mesencephalic lesion on the left side. Stimulation of the left ulnar produced a low amplitude somatosensory evoked potential with a sensory conduction velocity of 54 m/sec. but the evoked potential was so small that no certain determination of conduction velocity could be made. The evoked potential after stimulation of the right ulnar was again small in amplitude with conduction velocity of 93 m/sec. Again the evoked potential was so small that certainty of determination of conduction velocity was difficult. The midbrain lesion, irregular in distribution and interrupting the sensory pathways from the arms probably was the cause of the low amplitude evoked potentials and the widely differing conduction velocities.

Patient C, a 61 year old female, was suffering from a diffuse polyneuropathy involving all four limbs. Schilling's test and the severe loss of vibration sense were compatible with a cord lesion as in subacute combined degeneration of the spinal cord. EMG showed partial denervation in distal muscles of the left leg and slowed motornerve conduction velocities of the left and right peroneal and median nerves. Two point discrimination of the left hand was not reliable although the right hand was normal. The legs were sore to touch and the position sense of the left leg was lost while that of the right leg was probably impaired. She was very ataxic while standing. The left leg appeared worse with tingling and numbness and no vibration sense. Thirteen years before the patient had suffered a stroke involving the left side of the body. Stimulation of the left and right median nerves gave normal potentials but indicated a delayed conduction velocity of the left side (left median 56.3 m/sec., and right median 81.8 m/sec). Stimulation of the right peroneal gave an evoked potential of low amplitude from which it was not possible to calculate a nerve conduction velocity. The left peroneal nerve gave no evoked potential. The results are compatible with a polyneuropathy involving the left leg more than the right leg and the left arm more than the right arm.

DISCUSSION

The Stimulus:

It was found that clear evoked potentials could only be recorded if the stimulus was applied directly to the nerve such as to cause peripheral parasthesiae. Consequently, a high voltage had to be used which occasionally caused a stinging pain over the area of stimulation. This was very rare except when stimulating the posterior tibial nerve at the knee. The topical anaesthetic, xylocaine, had no effect on alleviating the pain but provided the nerve was close to the surface and the patient had a high pain threshold, a sufficient compound action potential could be produced.

A two second interval was used as any smaller interval would occasionally involve interference of the evoked potential upon the succeeding evoked potential.

It is probable from the evidence of Bergamini et al (1967)(8) that the stimulus passes up the nerve to the posterior columns along type A α fibres which have a fibre diameter of 12-20μ and a conduction velocity of 70-120 m/sec. (Ganong, 1967[26]). From the dorsal columns the fibres synapse in the gracile and cuneate nuclei. The second order neurons from the gracile and cuneate nuclei cross the midline and ascend in the medial lemniscus to end in the cortical relay nuclei of the thalamus (specifically, in the ventral posterolateral and posteromedial nuclei). There the nerve fibres synapse on the neurons whose axons form the thalamic radiation to the postcentral gyrus.

Recording from the Scalp:

The arrangement of the thalamic radiation fibers is such that the parts of the body are represented in order along the postcentral gyrus, with the feet on top and the head at the foot of the gyrus. The location of these points was as described in the method.

During the recording of the evoked potential it was important that the patient did not move, clench the jaws or cause any muscle activity as the electrical activity from muscle contraction masked the evoked potential. Consequently, the patient must be cooperative and relaxed. Only the first negative and positive waves of the evoked potential were recorded as these have the most constant latencies (Giblin, 1964)(24).

Averaging:

The process of averaging is designed to separate a constant time locked signal from random noise having negative and positive values. The activity recorded from the scalp has an amplitude of approximately 30μV and the somatosensory evoked potential primary waves have amplitudes of approximately 5μV. By averaging, the general random electroencephalographic noise was reduced to zero, while the constant amplitude, time locked evoked potential of 5μV remained. One problem with averaging is that the process can also "average in" any constant signal such as 60 c/s activity from the main power supply. This was eliminated by jittering the stimulus interval randomly across the time of 1 cycle of 60 c/s.

frequency, i.e. 16.67 msec. so that in fact the stimulus interval was not exactly two seconds but varied by 16.67 msec. Consequently the 60 c/s frequency waves of the previous averaging cycles were never in phase with the succeeding averaging cycle. The evoked potential, however, was time locked with the stimulus which was time locked with the commencement of the averaging cycle and consequently the previous evoked potential was always in phase with the succeeding evoked potentials.

The computer sampled input signals at 500 fixed time intervals, converted the samples to digital form, and stored the sample values at 500 separate locations in a memory. The sampling process was continued for 100 repetitions of the stimulus - evoked potential cycle. During the first repetition, sample values were stored in memory with each memory location corresponding to a definite sample time. Then, during subsequent repetitions, the new sample values were added algebraically to the values accumulated at the corresponding memory locations and divided by the number of repetitions.

To tell the computer where the beginning of each signal repetition was, a synchronizing signal, the stimulus, was triggered by the computer. To be averaged, the evoked potential had to repeat exactly following each sync. pulse.

This simple averaging process tends to enhance the signal with respect to the noise. On the other hand, the noise - which was random and not time-locked to the signal - made both positive and

negative contributions at any sample point during successive repetitions. Therefore, the noise portion of the stored sum decreased with averaging.

More formally, the averaging or summation process can be described as follows:

Let the input be f(t), composed of a repetitive signal portion s(t) and a noise portion n(t).

Say the kth repetition of s(t) begins at time t_k (and let $t_1 = 0$).

Finally, let samples be taken every T seconds. We then have:

$$f(t) = s(t) + n(t)$$

This signal is sampled, and the sample values are:

$$f(t_k + iT) = s(t + iT) + n(t_k + iT)$$

$$= s(iT) + n(t_k + iT)$$

For a given i and k, $n(t_k + iT)$ is a random variable. It is reasonable to assume that in a real situation, where the noise is 1/f noise, all the $n(t_k + iT)$ have a mean value of zero and the same RMS value, say σ. For different k's, the noise samples are statistically independent.

Now consider the ith sample point. A measure of the noise masking signal is the signal-to-noise voltage ratio, S/N. On any particular repetition,

$$S/N = \frac{s(iT)}{\sigma}$$

After m repetitions, the value stored at the ith memory location is:

$$\sum_{k=1}^{m} f(t_k + iT) = \sum_{k=1}^{m} s(iT) + \Sigma\, n(t_k + iT)$$

$$= ms(iT) + \Sigma\, n(t_k + iT)$$

Since the noise is random and the m samples are independent, the mean square value of the sum of the m noise samples is $m\sigma^2$, and the RMS value is $\sqrt{m}\,\sigma$. Therefore, the signal-to-noise ratio after summation is:

$$(S/N)_m = \frac{ms(iT)}{\sqrt{m}\;\sigma} = \sqrt{m}\;(S/N)$$

Thus summing m repetitions improves the signal-to noise ratio by a factor of $\sqrt{m}$. In this case m was 100 and the improvement in S/N was 10 times.

The average was calculated from the simple formula:

$$M_m^i = \frac{1}{m} \sum_{k=1}^{m} f_k^i$$

However, as the averaging process proceeded, the computer did not memorize the raw data. Consequently at even f_m^i the only information available was the original average M_{m-1}^i and the number of averaging cycles m.

Therefore the formula $M_m^i = M_{m-1}^i + \dfrac{f_m^i - M_{m-1}}{m}$ was used to determine the average.

The Variance:

The variance of background noise was measured by measuring the variance of each individual point of 500 points used in the averaging process. The formulu used originated from the standard formula for the variance:

$$V_m^i = \frac{1}{m} \sum_{k=1}^{m} (f_k^i)^2 - (M_m^i)^2$$

However, the only information available in the computer as the program progressed was the old variance, V_{m-1}^i, the old average M_{m-1}^i, the present value f_m^i and the new average. Thus the formula:

$$V_m^i = V_{m-1}^i + (M_{m-1}^i)^2 + \frac{(f_m^i)^2 - V_{m-1}^i - (M_{m-1}^i)^2}{m} - (M_m^i)^2$$

was used and simplified as follows:

$$\text{Let } S_{m-1}^i = V_{m-1}^i + (M_{m-1}^i)^2$$

$$\therefore \quad V_m^i = S_{m-1}^i + \frac{(f_m^i)^2 - S_{m-1}^i}{m} - (M_m^i)^2$$

S_{m-1}^i was introduced to reduce the number of calculations within the computer.

The variance only showed a straight line probably because the background EEG noise was very high in comparison with the evoked potential and thus the system was not sufficiently sensitive to detect the variance due to jitter of the evoked potential across time.

However, this straight line did give an estimate of the background noise if it is assumed that Gaussian noise is comparable to background noise,(57,58).

Intuitively, it seems that, if a constant of 20μV or 3 mm was subtracted from the variance, and if more jitter was given to the evoked potential, as described in the method, with a flip-flop arrangement, a peak or drop in variance may occur. If the first derivative of this result was taken, then, it is probable that another point for measurement of latency would materialize. However, this point has to be investigated further.

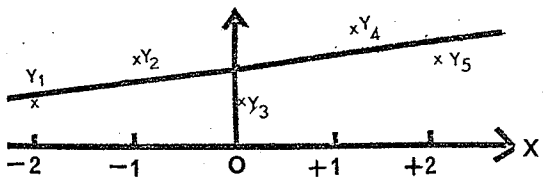
The First Derivative:

After the averaging of the evoked potential, the first derivative of the evoked potential was calculated by the computer using the least square approximation of first order. The final equations for the least square line are:

$$y = a_0 + a_1 x \qquad \text{where} \qquad a_0 = \frac{(\Sigma Y)\ (\Sigma X^2) - (\Sigma X)\ (\Sigma XY)}{N\Sigma X^2 - (\Sigma X)^2}$$

$$\text{and} \qquad a_1 = \frac{N\Sigma XY - (\Sigma X)\ (\Sigma Y)}{N\Sigma X^2 - (\Sigma X)^2}$$

The constant a_1 represents the slope, and a_0 is not needed. If the coordinate system is selected in the following way,



The calculations are simplified:

$$N\Sigma X^2 = 10(N) \qquad (\Sigma X) = 0 \qquad \therefore \quad a_1 = \frac{\sum_{i=1}^{5} X_i\ Y_i}{10}$$

It was found that 19 points for each regression line was a suitable number to deal with such that there were X_i values from -9 to +9

$$\therefore \quad a_1 = \frac{\sum_{i=1}^{19} X_i Y_i}{570}$$

$$\therefore \quad a_1 = \text{const } (-9Y_1 - 8Y_2 \ldots\ldots 0Y_{10} \ldots\ldots + 9Y)$$

$$= \text{const } (-9Y_1 \ldots\ldots -Y_9 + Y_{11} \ldots\ldots +9Y_{19})$$

a_1 was calculated for the first 19 points, then for points 2 to 20, 3 to 21, et seq. The result was 500 points with values of a_1. These were displayed on the oscilloscope screen to give a display of the first derivative of the evoked potential. If a less number of points were used for determination of a_1, the display had too many peaks and if a greater number of points were used, the display was too smoothed.

The Evoked Potential

The potential evoked by stimulation of the ulnar or median nerves, at times, showed an initial negative peak followed by a positive wave (Fig. 4 and 5) which was always present. When the initial negative wave was not present, the positive wave usually had an abrupt beginning (Fig. 6 and 7). The peak of the negative wave or the beginning of the positive wave were found to be good points from which to measure latencies. The presence or absence of the negative wave was probably determined by the position of the active electrode and the synchrony of the compound action potentials arriving at the brain. A nerve that could easily be stimulated, resulted in the production of an evoked potential with an initial negative wave. The latencies of the waves of the evoked potential were in agreement with those determined by Giblin (27).

The potential evoked by stimulation of the posterior tibial and peroneal nerves rarely produced the initial negative wave probably because of the longer pathway that the action potential had to travel and the consequent loss of synchrony of the action potential on arrival at the brain. However, the positive wave was always present.

The first derivative of the initial downsweep of the evoked potential provided a good peak from which to measure latencies. Occassionally, with the tibial and peroneal nerves, a double peak would arrise. In this situation the beginning of the downsweep of the first derivative peak was used as the point for measurement.

Evoked potentials could also be recorded occassionally from other areas of the head,as described in the method, but these potentials were very much lower in amplitude and not consistent. Liberson and Kim (44) reported that an evoked potential with a latency of 12 msec could be recorded at the inion but this was not recorded in the three experiments performed. Any evoked potentials recorded outside the "hand" area of the postcentral gyrus would probably be due to muscle activity. No evoked potential was recorded when there was no stimulus.

Statistical Analysis

Tests for skewness and kurtosis were done to test whether the data was normally distributed, and that if it was not a normal distribution, in what direction it was distributed. Skewness indicates whether the data is positively skewed or skewed to the right or whether it is negatively skewed or skewed to the left. Zero indicates a perfect normal distribution. Kurtosis indicates the peakedness of the distribution, 3being the figure for a perfect normal distribution. Any figure above 3 indicates a peaked distribution or a leptokurtic distribution, 3 indicates a mesokurtic distribution and any figure below 3 indicates a platikurtic or flattened distribution.

The data was also plotted on probability paper, as described by Alger (3), which has the horizontal coordinates expanded on both sides of the center in such a way that points following the normal error function will lie along a straight line. To test whether the data actually did follow a straight line and to what degree it did so, a linear regression and analysis of

variance was performed. The value T was a test of the hypothesis that B, the slope of the line, was equal to zero and consequently whether the distribution was linear. Any T value greater than 3.355 or smaller than -3.355 (the theoretical T value at its corresponding 8 degrees of freedom) would mean that $B \neq 0$ at the 0.5% level and consequently that the data was linear or normally distributed. R indicates to what degree the X and Y values are correlated linearly. Ideally the R value should be 1 or -1. All the data showed a high degree of correlation in a positive direction. R^2 is a measure of the variance of the X values associated with the variation of the Y values. R^2 gives the amount of variation in the X variables which may be explained by the linear association with the Y variables. Again the ideal value is 1.

The chi-square distribution test is another test of normality. If the χ^2 value is smaller than χ_α^2, then the hypothesis that the data is normally distributed can be accepted.

The data appears normally distributed with one exception, peroneal 2, in which the χ^2 value is greater than the $\chi^2_{0.05}$. The peroneal nerve however shows a wide range of values and in the analysis of variance, the residuals show that the higher values are the more deviated from the straight line. More values for the peroneal nerve are needed before a conclusion that the data is not normally distributed could be made.

As there appeared to be little difference in the mean velocities and standard deviations from the left and right sides, it was assumed that the velocities on the left and right are equal. Consequently the velocities from the left and right for each nerve were added together to give more data and thus to give the statistical tests more validity.

Sources of Error:

Table V shows, that, assuming the velocities on the right side are the same as the velocities on the left, the mean sensory nerve conduction velocities are as follows:

	Method I	Method 2
Ulnar Nerve.....	74.1 ± 8.9 m/sec.	70.1 ± 6.5 m/sec.
Median Nerve.....	82.9 ± 8.9 m/sec.	78.6 ± 8.6 m/sec.
Peroneal Nerve.....	60.1 ± 14.7m/sec.	58.4 ± 13.7m/sec.
Tibial Nerve.....	63.1 _ 18.0m/sec.	59.4 ± 14.2m/sec.

Method I denotes that the velocities were determined from the evoked potential latencies, and Method 2, denotes that the velocities were determined from the latencies of the first derivatives. As the standard deviations of Method 2 are not significantly different from the standard deviations of Method I, it may be assumed that, across a population, Method 2 is as good as Method I. Consistency tests showed the same result (Tables VI-IX).

Consider, now, the two worst possible situations, which may arise because of error due to measurement of latencies, and distance and because of error caused by a change of temperature. At the 95% level or 2 standard deviation (S.D.) level of error, the

highest velocity of the ulnar nerve (Table X) would be, from Latency I:

$$\frac{31.93}{3.39} = 94.2 \text{ m/sec.}$$

and the lowest velocity of the ulnar nerve from Latency I would be:

$$\frac{27.57}{4.51} = 61.1 \text{ m/sec.}$$

This would give a possible range of 33.1 m/sec. due to measurement error. Similarly, using Latency II, the range would be 18.8 m/sec. skin temperature varied by 1°C. Thus, assuming that a rise of 1°C increases the velocity of conduction by 2 m/sec. (Buchthal and Rosenfalck[15]), the theoretical range of velocity due to error would increase by 4 m/sec. to 37.1 m/sec. for Latency I and 22.8 m/sec. for Latency II.

However, considering the accumulated data from consistency experiments (Tables VI-IX), the velocity range from Method I for the ulnar nerve at the 95% level or 2 S.D. level of error would be 70.4 ± 12.8 m/sec. or a range of 25.6 m/sec. Similarly, from Method 2 the range would be 28.4 m/sec.

This seems to indicate that the error of measuring nerve conduction velocities was attributable to change in temperature and to error in measurement of latencies and distances. The other three nerves, the median, peroneal and tibial, fall into line with this hypothesis.

There was little range in age, thus this factor can not be considered as a source of error. No difference in conduction velocities between the sexes has been reported and there were not enough male subjects in this study to come to any conclusion on

whether a difference exists.

Evaluation of error indicates that the wide range of values is probably a result of error due to temperature change and error in measurement of latencies and distance. The standard deviations from Methods I and 2 show that each method is as good as the other - a clear evoked potential will produce a distinct first derivative. The advantage is that the latencies can be measured by two methods.

Inferences:

A somatosensory evoked potential of about 5μV in amplitude can be recorded from a background noise of about 30μV in amplitude. From this evoked potential the sensory nerve conduction velocity may be determined by two methods; one, using the evoked potential (Method I) and the other using the first derivative of the evoked potentials (Method 2). If it is assumed that the velocities recorded were normally distributed, as statistical tests infer, and that the velocities on the right side are the same as the velocities on the left, then it can be stated that the nerves have the following sensory nerve conduction velocities:

	Method I	Method 2
Ulnar Nerve.....	74.1 ± 8.9 m/sec.	70.1 ± 6.5 m/sec.
Median Nerve.....	82.9 ± 8.9 m/sec.	78.6 ± 8.6 m/sec.
Peroneal Nerve.....	60.1 ± 14.7m/sec.	58.4 ± 13.7m/sec.
Tibial Nerve.....	63.1 ± 18.0m/sec.	59.4 ± 14.2m/sec.

The peroneal and tibial nerves showed a greater standard deviation because of the longer pathway for the stimulus to travel and thus a more diffuse evoked potential with its concomitant error in measurements of latency.

Tests on abnormal subjects showed the applicability of nerve

conduction tests to clinical practice if it is assumed that all values of nerve conduction velocity within 2 standard deviations of the mean are normal. If one side is markedly delayed in comparison with the other side (i.e. above 10 m/sec. in difference), then this could be a measurement of the abnormality of one side. The amplitude or presence of evoked potentials can also give an indication of abnormality. However, the patient must be cooperative and muscle activity of the scalp must be minimal. It must be realised that if there is a lot of muscle activity, or that if the evoked potential is low in amplitude or diffuse, then, it is possible to arrive at a nerve conduction velocity lower than normal as it is to arrive at a nerve conduction velocity above normal. An example of this may occur in the following situation. Stimulation of the nerve at the wrist is easier than above the elbow because the nerve is closer to the surface at the wrist. In a difficult situation it may be possible to produce a good evoked potential from stimulation of the wrist with a normal latency but a poor evoked potential with a longer than normal latency may be produced from stimulation above the elbow. The result may be a difference in latency of zero and therefore, theoretically a nerve conduction velocity of infinity. Therefore, to make a statement concerning nerve conduction velocities, a good evoked potential must be the first premise.

SUMMARY

(1) The sensory nerve conduction velocities of the ulnar, median peroneal and posterior tibial nerves were determined from the latencies of somatosensory evoked potentials and the first derivatives of the somatosensory evoked potentials.

(2) Tests for the distribution of the evoked potential over the scalp were conducted.

(3) The background noise level was determined from the variance.

(4) Consistency of the nerve conduction velocities within one individual was determined.

(5) Statistical tests were performed upon the velocities to determine whether the data was normally distributed.

(6) Three clinical patients were tested for nerve conduction velocities.

(7) The processes of stimulating, recording and averaging, the variance, the first derivative, the evoked potential, statistical analysis and sources of error were discussed.

(8) It was inferred that there are two methods of recording sensory conduction velocities within one nerve, method I using the evoked potential, method 2 using the first derivative, which were equally accurate.

(9) Conduction velocities were as follows:

	Method I	Method 2
Ulnar Nerve.....	74.1 ± 8.9 m/sec.	70.1 ± 6.5 m/sec.
Median Nerve.....	82.9 ± 8.9 m/sec.	78.6 ± 8.6 m/sec.
Peroneal Nerve....	60.1 ± 14.7m/sec.	58.4 ± 13.7m/sec.
Tibial Nerve.....	63.1 ± 18.0m/sec.	59.4 ± 14.2m/sec.

(10) It was inferred that this study could be put to clinical use.

BIBLIOGRAPHY

1. Alajouanine, T., Scherrer, J., Barbizet, J., Calvet, J., and Verley, R.: Potentiels évoqués corticaux chez des sujets atteints de troubles somesthésiques. Rev. Neurol. (Paris), 98: 757-761, 1958.

2. Albe-Fessard, D., and Buser, P.: Activites intracellulaires receuillies dans le cortex sigmoide du chat: participation des neurones pyramidaux au "potentiel évoqué" somesthésique, J. Physiol. Paris, France 47 (1955) 67-69.

3. Alger, P.L.: Mathematics for Science and Engineering, McGraw Hill Book Company, Inc. 1957, pp. 305-306.

4. Allison, T.: Recovery functions of somatosensory evoked responses in man. Electroenceph. Clin. Neurophysiol., 14, (1962) 331-343.

5. Amassian, V.E.: Evoked single cortical unit activity of the somatic sensory areas. Electroenceph. Clin. Neurophysiol., 5 (1953) 415-438.

6. Amassian, V.E.: Studies on organization of a somesthetic association area, including a single unit analysis, J. Neurophysiol., 17 (1954) 39-58.

7. Amassian, V.E., Patton, H.D., Woodbury, J.W., Towe, A., and Schlag, J.E.: An interpretation of the surface response in somatosensory cortex to peripheral and interareal afferent stimulation. Electroenceph. Clin. Neurophysiol., 7 (1955) 480-482.

8. Amassian, V.E., Waller, H. J., and Macy,Jr. J.: Neural mechanism of the primary somatosensory evoked potential. Ann. N.Y. Acad. Sci., 25 (1964) 5-32.

9. Bergmanini, L., Bergamasco, B.: Cortical evoked potentials in man. Charles C. Thomas, Illinois, U.S.A., 1967 pp. 67-75.

10. Bergamini, L., Bergamasco, B., Fra, L., Gandiglio, G., Mombelli, A.M., and Mutani, R.: Réponses corticales et périphériques évoquées par stimulation du nerf dans la pathologie des cordons posterieurs. Rev. Neurol (Paris) 155: 99-112, 1966e.

11. Bickford, R.G., Galbraith, R.F., and Jacobson, J.L.: The nature of average evoked potentials recorded from the human scalp. Electroenceph. Clin. Neurophysiol., 15 (1963) 720-725.

12. Bickford, R.G., Jacobson, J.L, and Cody, D.T.R.: Nature of average evoked potentials to sound and other stimuli in man. Ann. N.Y. Acad. Sci., 112 (1964) 204-223.

13. Brazier, M. A. B.: Studies of electrical activity of the brain in relation to anaesthesia. Neuropharmacology, Trans. First Conf. Josiah Macy Jr. Foundation, New York (1954) 107-144.

14. Brown, J.E.: A parametric study of neuroelectric responses to skin stimuli. Unpublished Master's Thesis, M.I.T. Cambridge, Mass. (1960).

15. Buchthal, F., and Rosenfalck, A.: Evoked action potentials and conduction velocity in human sensory nerve. Brain Research, 3 Vol. 1966.

16. Cracco, R.Q., and Bickford, R.G.: Somatomotor and somatosensory evoked responses, Arch. Neurol. 18 (1968) 52-68.

17. Dawson, G.D.: Cerebral responses to electrical stimulation of peripheral nerve in man. J. Neurol. Neurosurg. Psychiat. 10 (1947) 137-140.

18. Dawson, G.D.: Cerebral responses to nerve stimulation in man. Brit. Med. Bull., 6 (1950) 326-329.

19. Dawson, G.D.: Autocorrelation and automatic integration. Symposia of IIIrd International EEG Congress, Electroenceph. Clin. Neurophysiol. (Suppl) 4 (1953) 26-37.

20. Dawson, G.D.: A summation technique for the detection of small evoked potentials. Electroenceph. Clin. Neurophysiol. 6 (1954) 65-84.

21. Dawson, G.D.: The relative excitability and conduction velocity of sensory and motor nerve fibres in man. J. Physiol. 131.(1956) 436-451.

22. Dawson, G.D., and Scott, J.W.: The recording of nerve action potentials through skin in man. J. Neurol. Neurosurg. Psychiat. 12 (1949) 259-267.

23. Domino, E.F., Matsuoka, S., Waltz, J., and Cooper, I.: Simultaneous recordings of scalp and epidural somatosensory-evoked responses in man. Science 145 (1964) 1199-1200.

24. Eccles, J.C.: Interpretation of action potentials evoked in the cerebral cortex, Electroenceph. Clin. Neurophysiol., 3 (1951) 449-464.

25. Eichler, W.: Uber die Abteilung der Aktionspotentiale vom menschlichen Nerven in situ. Z. Biol. 98 (1938) 182-214.

26. Ganong, W.F.: Review of medical physiology, Lange Medical Publications, 1967 pp. 32.

27. Giblin, D.R.: Somatosensory evoked potentials in healthy subjects and in patients with lesions of nervous system. Ann. N.Y. Acad. Sci. 112 (1964) 93-142.

28. Gilliatt, R.W., Goodman, H.V., and Willison, R.G.: The recording of lateral popliteal nerve action potentials in man. J. Neurol. Neurosurg. Psychiat. 24 (1961) 305-318.

29. Gilliatt, R.W., and Sears, T.A.: Sensory nerve action potentials in patients with peripheral nerve lesions. J. Neurol. Neurosurg. Psychiat. 21 (1958) 109-118.

30. Gilliatt, R.W., and Willison, R.G.: Peripheral nerve conduction in diabetic neuropathy. J. Neurol. Neurosurg. Psychiat. 25 (1962) 11-18.

31. Goff, W.R., Rosner, B.S., and Allison, T.: Distribution of somatosensory evoked responses in normal man. Electroenceph. Clin. Neurophysiol. 14 (1962) 697-713.

32. Goto, A., Kosaka,K., Kubota, K., Nakamura, R., and Narabayushi, H.: Thalamic potentials from muscle afferents in the human. Arch. Neurol. 19 (1968) 302-309.

33. Haller, A.von,: Elementa physiologiae corporis humani, Grasset, Lansanne, 4 (1762) 596 pp.

34. Halliday, A.M.: Changes in the form of cerebral evoked responses in man associated with various lesions of the nervous system. Sixth International Congress of Electroencephalography and Clinical Neurophysiology. Vienna, 1965. EEG-Abstracta-EMG, 9, 1965.

35. Halliday, A.M., and Wakefield, G.S.: Cerebral evoked responses in patients with dissociated sensory loss. Electroenceph. Clin. Neurophysiol. 14 (1962) 786.

36. Halliday, A.M. , and Wakefield, G.S.: Cerebral evoked potentials in patients with dissociated sensory loss. J. Neurol. Neurosurg. Psychiat. 26 (1963) 211-219.

37. Hrbek, A., Hrbkova, M., and Lenard, H.: Somatosensory evoked responses in newborn infants. Electroenceph. Clin. Neurophysiol. 25 (1968) 443-448.

38. Hrbek, A., Hrbkova, M., and Lenard, H.: Somatosensory auditory and visual evoked responses in newborn infants during sleep and wakefulness. Electroenceph. Clin. Neurophysiol. 26 (1969) 597-603.

39. Jasper, H., Lende, R., and Rasmussen, T.: Evoked potentials from the exposed somatosensory cortex in man. J. Nerv. Ment. Dis. 130 (1960) 526-537.

40. Kelly, D.L., Goldring, S., and O'Leary, J.L.: Average evoked somatosensory responses from exposed cortex in man. Arch. Neurol. 13 (1965) 1-9.

41. Kimura, J., Powers, J.M., and Van Allen, M.W.: Reflex response of the orbicularis oculi muscle to supraorbital nerve stimulation: Study in normal subjects and in peripheral facial paresis. Arch. Neurol. 21: 193-199 Aug. 1969.

42. Larson, L-E.: Electroencephalographic responses to peripheral nerve stimulation in man. Electroenceph. Clin. Neurophysiol. 5 (1953) 377-384.

43. Larson, S.J., Sances, A., and Christenson, P.C.: Evoked somatosensory potential in man. Arch. Neurol. (Chic) 15 (1966) 88-93.

44. Li, C-L: Cortical intracellular synaptic potentials J. Cell. Comp. Physiol. 58 (1961) 153-167.

45. Li, C-L, and Jasper, H.H.: Microelectrode studies of the electrical activity of the cerebral cortex in the cat. J. Physiol. 121 (1953) 117-140.

46. Liberson, W.T., and Kim, K.C.: The mapping of evoked potentials elicited by stimulation of the median and peroneal nerves. Electroenceph. Clin. Neurophysiol. 15 (1963) 720-725.

47. Lorente de No, R.: The cerebral cortex architecure, intracortical connections and motor projections. Physiology of the Nervous System by J. F. Fulton, Chap. 15, p.p. 291-325, Oxford University Press, New York, N.Y. (1938).

48. Mark, R.F., and Steiner , J.: Cortical projection of impulses in myelinated cutaneous afferent nerve fibres of the cat. J. Physiol. 142 (1958) 544-562.

49. Marshall, W.H.: Observations on subcortical somatic sensory mechanism of cats under nembutal anaesthesia. J. Neurophysiol. 4 (1941) 25-43.

50. Marshall, W. H., Woolsey, C.N. and Bard, P.: Observations on cortical somatic sensory mechanism of cat and donkey. J. Neurophysiol. 4 (1941) 1-24.

51. Mountcastle, V.B., and Henneman, E.: Pattern of tactile representation in thalamus of cat. J. Neurophysiol. 12 (1949) 85-99.

52. Mountcastle, V.B., Davies, P.W., and Berman, A.L.: Response properties of neurons of cat's somatic cortex to peripheral stimuli. J. Neurophysiol. 20 (1958) 374-407.

53. Nezlina, N. I., and Vorob'eva, A.D.: Differences in configuration of evoked responses of the cat sensorimotor cortex. Neuroscience Translation 1969, Zhurnal Vysshei Nervnoi Deyatel'nosti imeni I.P. Pavolva. 18, no.3 (1968) 500-506.

54. Piper, H.: Uber die leitungsgeschwindigkeit in den markhalfigen menschlichen Nerven, Pflugers, Arch. ges. Physiol. 124 (1908) 591-600.

55. Piper, H.: Weitere Mitteilungen uber die Geschwindigkeit der Erregunsgsleitung im mark haltigen menschlichen Nerven, Pflugers Arch. ges. Physiol., 127 (1909) 474-480.

56. Rudin, D.O., and Eisenman, G.: On the role of the spinal afferent neuron as a generator of the extracellular current. J. Gen Physiol. 37 (1954) 795-812.

57. Saunders, M.G.: Amplitude probability density studies of alpha activity in the electroencephalogram, In: Mathematics and computer science in biology and medicine. London, Her Majesty's Stationery Office, p. 163-175, (1965).

58. Saunders, M.D.: Amplitude probability density studies on alpha and alpha-like patterns. Electroenceph. Clin. Neurophysiol. 15, 761 (1963).

59. Schwartz, M., and Shagass, C.: Recovery functions of human somatosensory and visual evoked potentials. Ann. N.Y. Acad. Sci. 25 (1964) 510-525.

60. Storozhuk, V.M.: Unit activity of primary somatosensory cortex during primary responses, Neuroscience Translations 1968, Zhumal Yysshei Nervnoi Deyatel'nosti imeni I.P. Pavolva, 18 no. 3(1968) 469-477.

61. Uttal, W.R., and Cook, L.: Systematics of the evoked somatosensory cortical potential: a psychophysical-electrophysiological comparison. Ann. N.Y. Acad. Sci. 112 (1964) 60-80.