

THE NEURAL EFFECT OF ANOREXIA NERVOSA AS EXAMINED  
THROUGH SOMATOSENSORY EVOKED POTENTIALS

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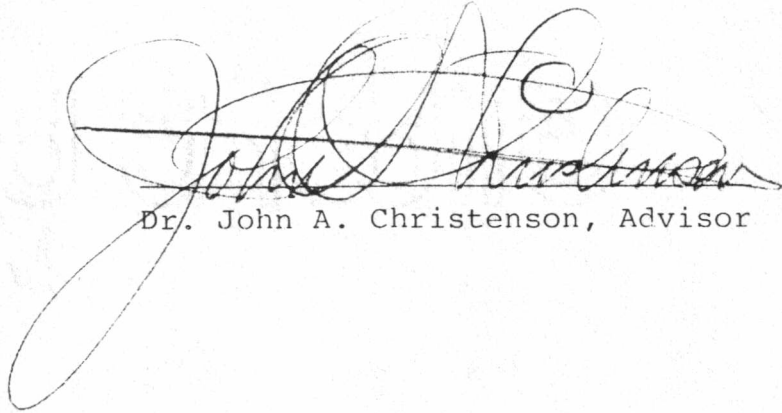
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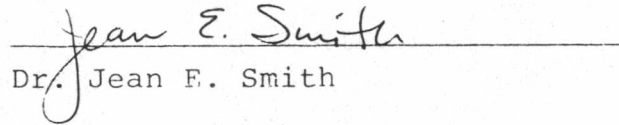
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The Rev. J. Eugene Peoples

April 2, 1984

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## ABSTRACT

Four patients diagnosed as having anorexia nervosa were compared to eight normal patients using somatosensory evoked potentials in looking for a symptomatic neural component in the metabolic dysfunction. Central conduction time along the spinal cord and peripheral nerve velocity were measured and calculated in each group of patients. Lower means were found in the anorexic in comparison to the normal patients, but the difference was not significant. More testing needs to be done on more patients to insure conclusiveness of the test.

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## INTRODUCTION

Medical experts believe anorexia nervosa and bulimia are rapidly reaching epidemic proportions (6). It is estimated that anorexia nervosa and or bulimia strike more than one in every 200 teenagers (6), nearly 1,000,000 or more victims in this country alone (2). These disorders are curable, but their long term affects on one's health are generally unknown (6).

The purpose of the study is to determine whether or not anorexia has a symptomatic neural component. Anorexia involves a rapid loss of 25% or more of body weight brought about through starvation, vomiting, laxative abuse, and excessive exercise or a combination of these methods. Studies have shown that specific metabolite deficiencies have a neural effect (1,10,11,13). This study examines the neural effect of a general metabolite deficiency, using somatosensory evoked potentials.

## LITERATURE REVIEW

Anorexia can end in death. Authorities estimate that between 5 and 20% of all anorexics die (2,6,15). However, prior to this death, should it occur, anorexia exhibits a wide range of physical and physiological changes seen in most victims. Occasionally anorexic symptoms are produced by brain tumors, lesions of the CNS, glandular disorders, or other physical and biological causes (2,6,9). This condition is known clinically and secondary anorexia. Most of the time though, anorexia is produced through voluntary excessive weight loss which produces dire health consequences for the victim (9).

Symptoms produced by eating disorders vary according to the methods used to produce the anorexic tendencies. Laxative abuse produces dehydration of the skin, constipation, edema from electrolyte imbalance (mainly potassium and sodium), and bloating (3,6). These are common symptoms linked to the method of self imposed starvation.

As a result of excess weight loss, other problems are produced within the victim. Anorexia characteristically produces hypothermia in the victim (5). Here the victim feels chilled and growth of fine body hair, lanugo, is produced. Also, the heart and metabolic rate slow, generally hampering the blood pressure, pulse rate, and general circulation (5).

One of the major concerns is the effect on the endocrine system. Through the loss of body fat, the anorexic becomes

unable to produce hormones necessary for sexual development (3). If this occurs during the growth spurt, the victim eventually stops growing and further starvation arrests the adolescent's sexual development, thus eliminating their reproductive capacity (9). In older female anorexics, the endocrine disorder causes the cessation of the menstrual cycle - secondary amenorrhea (3,6,9). Anorexia also produces loss of hair and nail quality (due to protein deficiencies), insomnia, ulcers, acrocyanosis, petechiae, leukopenia, and anemia (3,6,15). These are the general dysfunctions commonly associated with anorexia. They can be lifelong or even fatal.

Much more investigation has been done on the neural component of specific nutritional deficiencies. These studies revolve mostly around vitamin deficiencies; deficiencies of vitamins A, E, and B12. One study, which examined nervous and ocular disorders in children with cholestasis and vitamin A and E deficiencies, found four types of clinical manifestations: abnormalities of eye movement, retinal degenerative changes, peripheral neuropathy, and cerebellar dysfunction (1). A similar study on vitamin E deficiency and chronic intestinal malabsorption, discovered clinical manifestations of dysarthria, cerebellar ataxia, and prominent proprioceptive loss with depressed or absent tendon reflexes (11).

A substantial amount of research has been done on vitamin B12 deficiency. A deficiency in B12 leads to the demyelination of myelinated nerves, slowing the conduction time. One study

demonstrated this with visual, auditory, and somatosensory evoked potentials. It showed that the evoked response delay correlated directly with the degree of neurological dysfunction. They also found that abnormalities were present in sensory systems without clinical evidence of involvement (13). A similar study supported these findings with the evoked response studies, and also showed with peripheral nerve conduction tests, an axonal degeneration peripheral neuropathy (10).

The effect of anorexia on the nervous system is not as well known. One investigator, using visual evoked potentials (VERs), concluded that the typical cases of anorexia nervosa can be classified as psychosomatic diseases, and the atypical cases of eating disorders as neuroses (5). They noticed that the amplitudes of the VERs increased markedly during hyperventilation as did those of anxiety neurotic patients. Another set of investigators working with rats using VERs, found the conduction velocity to be slower than in normal rats (14).

The schema of my experiment was to test for a neurologic change in the anorexic patient using somatosensory evoked potentials. I could find nothing written on this subject, using this specific procedure.

Sensory evoked potentials offer a unique tool to the neurologist, they allow a noninvasive physiologic assessment of the brain and nervous system (16,17). An evoked potential is produced as an electrical manifestation of the brain's

response to an external stimulus. Evoked potentials have very low amplitudes that normally are masked by background brain wave activity and artifact. Because of this they are not recorded on EEG recordings. Since they are coupled to a specific stimulus, and the background activity is not, it is possible to extract them by use of a computer signal averager. The presence or absence of appropriate evoked potential waveforms and their latencies are the principle components used in interpretations (7,8), once they are extracted. the contribution of sensory evoked potentials is that they allow earlier and more accurate diagnoses than previously possible (17).

From these concepts, three evoked potential tests have evolved into clinical use: pattern-shift visual (VEP), brain stem auditory (BAEP) and somatosensory evoked potentials (SSEP). Pattern-shift evoked potentials are produced by altering a pattern on a T.V. screen, as the patient watches, and monitoring the brain's response to the changing stimulus. Brain-stem auditory evoked potentials are produced by delivering "clicks" through headphones directly to the ears, and again monitoring the brain's response. These two tests monitor the response in just the brain, somatosensory evoked potentials assess also the entire length of the somatosensory pathway (12). SSEPs are produced by a slight electrical shock to a peripheral nerve, usually either the median nerve at the wrist, or the tibial nerve at the ankle. This test allows you to measure and calculate the peripheral conduction velocity, the central latency, and also assess normal brain function (12,16).

These three tests are reliable diagnostic aids that constitute objective measures of functions in their respective sensory systems and tracts (7,8).

Evoked potentials provide sensitive quantitative extensions of the clinical neurologic examination. These become important when the neurologic examination and or the patient history prove equivocal (7,8). The potential value of evoked potentials is many fold. They demonstrate abnormal sensory system functions, clinically unsuspected lesions, and demyelination. They aid in defining the anatomic distribution of a disease process, and as an objective measure of changes in a patient's status over a period of time (7,8). Because of the diagnostic benefits, the use of evoked potentials is increasing and is becoming more important as a diagnostic tool for the neurologist.

Somatosensory evoked potentials may prove to be the most clinically useful of the three procedures, because of the more extensive portion of the CNS traversed by the SSEPs (17). This allows for monitoring along peripheral nerve tracks, the spinal cord, and then also the brain. This becomes of diagnostic value in evaluation of disorders causing proximal lesions, reduced sensory conduction in peripheral nerves due to demyelination, and subclinical lesions which could be useful for diagnosis of a disease in its early stages (12). SSEPs can be used to evaluate the entire length of the somatosensory pathway, and by itself is a powerful diagnostic tool. This makes it useful to study disorders with an unsuspected neural component.

## MATERIALS AND METHODS

### Patients

Four patients were obtained through permission of Bruce Chessen, Ph.D., Clinical Psychologist, South Central Regional Mental Health Center in Billings, Montana; William Hague, M.D., Psychiatrist; and Mona Sumner, Board of Directors at the Rimrock Foundation, also in Billings. The criteria on patient selection was that they had been diagnosed as presently having anorexia nervosa. Consent was received from both the patient and the attending physician, and both were informed as to the procedure and were allowed to ask questions.

Upon receiving the consent from both the physician and the patient, the patient was examined by Dale Peterson, M.D., a neurologist from the Billings Clinic. This examination was a primary neurological examination, concentrating on clinical signs of neurological dysfunction. It consisted of:

- 1) Past neurological history.
- 2) Testing for normal deep tendon reflexes on both upper and lower extremities.
- 3) Appreciation of vibratory senses.
- 4) Proprioception.
- 5) Pinprick pain sensation.

This examination evaluated the somatosensory pathway in its various components and the reception of the stimulus in the different brain centers of the patient. If dysfunction was detected then the patient was rejected. However, this did not occur.

### Normal Subjects

Eight normal subjects were obtained at random from the hospital staff. These subjects were asked if they presently had a diagnosed condition or if they had a past history of neurological problems. If these were negative then they were informed on the procedure, allowed to ask questions, and allowed to decide on their participation. Once their informed consent was given, they were tested in the same manner as the anorexics.

### Somatosensory Evoked Potentials (SSEPs)

The Nicolet CA1000 signal averager, X-Y plotter, and floppy disk memory storage unit were utilized in this experiment. Stimulus and acquisition parameters used were the generally accepted parameters for SSEP recordings:

#### Stimulus:

Rate: 4.1/sec.

Duration: 300 microsec.

Intensity: 5.9-10.4 milliamps

#### Acquisition

Sensitivity: 25-50 micro volts

Filter, low: 5 hz

high: 250 hz

Analysis time: 100 millisecc.

No. of Repetitions: 500

Conscious patients were tested in the supine position in the EEG room at the Deaconess Hospital in Billings, Montana. These patients were asked to relax or even sleep if they wanted. Surface electrodes were attached to positions L1 and L5 on the lumbar segment of the spinal cord, and to positions Cz

and Pz on the top of the skull and middle of the forehead respectively, by placing them in electrode cream and taping them in position. These leads were then connected into the signal averager via a lead box. A ground lead was placed on the belly of the gastrocnemius and was also connected into the lead box. The electrical stimulator was directly attached to the signal averager. The signal averager then monitored each lead for 100 millisecc. after the electrical impulse of duration 300 microsec. was initiated from the stimulator. It then averaged these through 500 repetitions of the stimulus as it was initiated and traveled along the neural pathway to the cortex.

The stimulus was applied to the tibial nerve just below the ankle bone. The intensity of the stimulus was increased or decreased accordingly, to produce a noticeable toe twitch without discomfort. This stimulus at the tibial nerve was enhanced by thorough cleaning of the skin directly over the nerve, and by applying a light coat of electrolyte gel. Once the toe twitch was achieved, the averaging process began. This was done twice on both legs to insure reproducibility of the results. The results were then plotted by computer readout onto a standardized graph printout, the latencies measured by the computer, and then the data were stored on floppy disk.

After the recording was completed, several measurements were taken. A measurement from the point of stimulus to L5 was taken, which represents the length of the peripheral

pathway. The height and age of the patient were also obtained. All the information regarding the anorexic condition of each patient was obtained directly from their attending physician.

## RESULTS

From the positions of the positive deflections in electrode recordings, and the latencies between them, it was possible to measure the Central Conduction Time. In the spinal segment of the SSEP recording, a positive deflection was recorded at the L5 electrode, and usually occurred around 20 msec. In the cerebral portion of the recording, a similar phenomenon occurred around 40 msec. The difference between these gave an estimate of the Central Conduction Time for the stimulus, as it moves up the spinal cord to the cerebral hemispheres.

The mean Central Conduction Time (CCT) for the standard normal subjects was 20.00 msec., with a standard deviation of 2.14. The CCT mean for the anorexic patients was 18.4 msec., which is within one standard deviation of the normal. The Student t value of significance was 0.4089 with a degree of freedom of 22. The t value for this degree of freedom at a .02 level of significance, was 2.508. Therefore, since 0.4089 was less than 2.508, there was no significant level of difference between the CCT times in the anorexic and normal subjects.

The peripheral conduction velocity can be calculated knowing the time of the first positive deflection and the distance from the stimulus to the position of the positive deflection. The first positive deflection occurred at the

L5 electrode, and with the distance between this electrode and the stimulus at the tibial nerve measured, it was possible to calculate the peripheral conduction velocity.

The velocity was obtained in m/sec by dividing the distance in centimeters (cm) by the latency in msec. The normal mean obtained for both legs was 50.2 m/sec, with a standard deviation of 3.54. The mean velocity for the anorexic patients was 47.4 m/sec, which was within one standard deviation of the normal. The Student t value of significance was 0.6416 with a degree of freedom of 22. The t value for this degree of freedom at a .02 level of significance was 2.508. Therefore, since 0.6416 is less than 2.508, there was no significant level of difference between the means of conduction velocities in normal and anorexic patients.

Table 1

Anorexic Patient Profile  
as obtained from their attending physician

PATIENT	SEX	AGE	HEIGHT (cm)	AGE OF ONSET	TREATMENT DURATION	HIGHEST WEIGHT	LOWEST	PRESENT	VITAMINS Y/N	DIAGNOSIS
							% OF HIGHEST	% OF HIGHEST		
A1	F	14	176.5	<1 yr	<1 yr	125	$\frac{91}{73}$	$\frac{105}{84}$	?	Anorexia
A2	M	17	170.2	15	2 yr	165	$\frac{70}{42}$	$\frac{100}{61}$	IV*	Anorexia Bulimia
A3	F	21	174.0	17	Outpat. 6 mon. Inpat. 4 wks.	170	$\frac{100}{59}$	$\frac{113.5}{67}$	?	Anorexia Bulimia #
A4	F	24	166.4	17	1 yr	140	$\frac{95}{68}$	$\frac{95}{68}$	?	Anorexia Bulimia

\*. This patient was hospitalized at the time of the testing and had been receiving vitamins intravenously.

#. This patient was also diagnosed as chemically dependent-EtOH.

Table 2

Conduction Velocity and Central Conduction Time in anorexic patients, recorded and measured by the Nicolet CA1000 signal averager using somatosensory evoked potentials.

PATIENT	SEX	AGE	HEIGHT	L	CONDUCTION VELOCITY		CCT P40-P20 mSEC		
						AVE	L	R	AVE
A1	F	14	176.5	52.1	48.7	50.4	20.0	19.6	19.8
A2	M	17	170.2	46.4	42.1	44.3	16.4	17.2	16.8
A3	F	21	174.0	50.3	44.6	47.5	18.8	19.6	19.2
A4	F	24	166.4	47.2	48.1	47.4*	18.0	17.6	17.8#

\*. The total average for conduction velocity for all the patients was 47.4 m/sec.

#. The total average for CCT for all the patients was 18.4 msec.

Table 3

Conduction Velocity and Central Conduction Times in anorexic patients, recorded and measured by the Nicolet CA1000 signal averager using somatosensory evoked potentials.

PATIENT	SEX	AGE	HEIGHT (cm)	CONDUCTION VELOCITY M/SEC			CCT P40-P20 mSEC		
				L	R	AVE	L	R	AVE
N1	M	32	180.3	49.0	51.5	50.3	20.4	23.2	21.8
N2	F	24	175.3	46.7	48.8	47.8	21.0	17.6	19.3
N3	F	35	157.5	47.3	48.9	48.1	21.0	19.6	20.3
N4	F	28	166.4	52.9	51.6	52.3	19.4	18.8	19.1
N5	M	30	190.5	43.3	44.1	43.7	20.0	19.8	19.9
N6	M	41	190.5	53.1	52.5	52.8	24.0	23.2	23.6
N7	F	21	162.6	53.3	55.8	54.5	18.0	16.8	17.4
N8	F	20	166.4	53.2	51.6	52.4*	20.4	19.5	18.8#

\*. The total average for conduction velocity for all patients was 50.2 m/sec.

#. The total average for CCT for all patients was 20.0 msec.

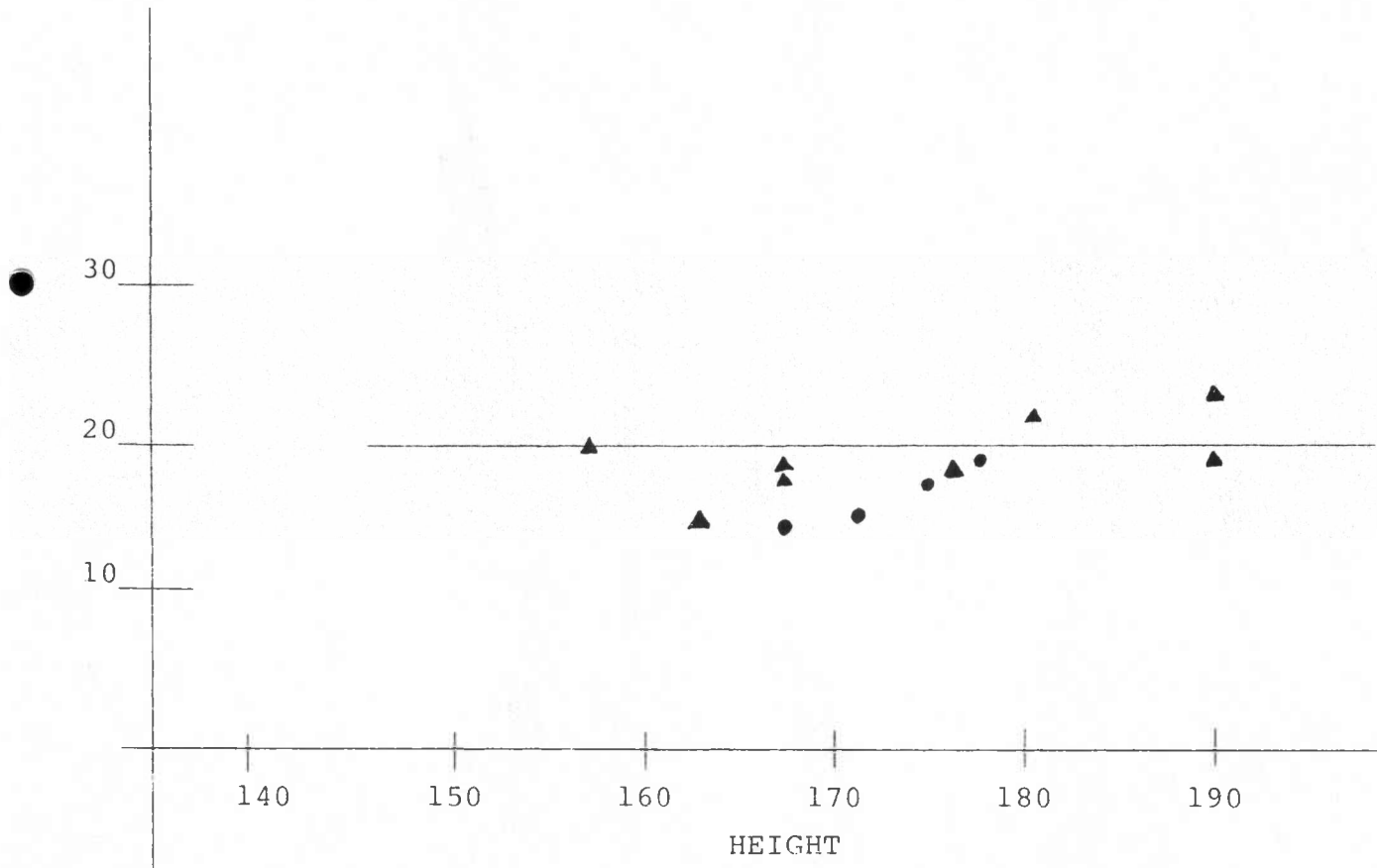


Fig. 1. Comparison of anorexic patients and normal patients with respect to the average Central Conduction Time in relation to overall height.

The Central Conduction Time was obtained by subtracting the latencies between the first positive deflections in the spinal cord and cerebral hemispheres as measured by SSEP recording. Normal patients (●); anorexic patients (▲); average for normal patients (—).

## DISCUSSION AND CONCLUSION

The statistics indicate that a significant difference does not exist between the means of CCT and conduction velocity in anorexics and normal subjects. This study was limited, however, in the number of patients tested so it is cautioned not to extend this statement to say that no difference exists at all. The Student t statistic simply states that no significant difference exists between the group means involved. The .02 level of significance indicates that with 98% confidence this finding did not occur by chance.

This is further supported by the standard deviation statistic. Most evoked potential measurements have a normal distribution so that the bounds of the normal range can be expressed in multiples of the standard deviation, (SD), (7,8). A standard deviation range of  $\pm 2.5$  SD, provides a probability of error less than 2%. The individual and group means of all the anorexics fall well within this range.

As a group, the anorexics were within the normal range, but examining the patients individually, interesting phenomena appear. A1, the patient most recently affected and judged strictly on weight gain, no longer anorexic (less than 25% depletion in original body mass), exhibited nearly normal means. The conduction velocity was 0.2 m/sec faster than normal, whereas the CCT was only 0.2 msec less than normal.

This is contrasted by the patient which was most severely affected, A2. This patient had been as low as 42% of original

body weight, and still exhibited the lowest percentage of original body weight in the test group, 61%. This was reflected in the largest deviation from the normal means in the anorexic test group. A2 exhibited a conduction velocity which was lower than normal by 5.9 m/sec and a CCT 3.2 msec slower than normal. It would have been interesting to see if a major difference existed prior to the administration of vitamins through IV.

The data on A3 and A4 are not as trendish. Patient A4 was tested at her lowest weight, and exhibited deviations from the normal means as well. The changes here were not as dramatic as in the case of patient A2, but they still represented a considerable deviation from the normal. Her conduction velocity was lower by 2.8 m/sec, while the CCT was less by 2.2 msec. Patient A3 exhibited a large change in conduction velocity, 2.7 m/sec slower than the normal, but a very slight change in CCT, only 0.8 msec.

It would be interesting to follow these patients in their treatment. By following the patients, one might be able to detect whether or not subsequent weight loss or gain affected the SSEP recording in a significant manner.

The data on the normals exhibit several interesting results. Patients N7 and N8 both had low CCT times, 17.4 msec and 18.8 msec respectively, while patient N5 exhibited a very low conduction velocity, 43.7 m/sec. These deviations within the normal test group make generalization on the effect of anorexia on the nervous system very difficult to make on an individual basis.

In comparison of the normal and anorexic test groups, it should

be noted that the age and sex of the patients contribute little to the variability. In this population of anorexic patients, the male may have been represented in higher percentage than which is normally found. Male anorexics represent 5% to 10% of all cases of anorexia, however, it is accepted that anorexia is underdiagnosed in males making it difficult to judge the actual occurrence of it in anorexic populations (4). Age plays even less of a role than does the sex. After age 2, there is little change in the latency of the nerve conduction until the fifth decade of life when it increases by about 2 msec per decade thereafter (8). Therefore, these factors contributed little to the difference between anorexic and normal patients, and may be ignored in interpreting the results.

This experiment has its limitations in linking a general metabolite deficiency to neural dysfunction. More information is needed on the anorexic's vitamin intake and general diet trend. The lack of information on vitamin intake is a serious gap in the data, judging from the literature written on the subject of vitamin deficiencies (1,10,11,13). Without more information of the actual diet, the variability here makes it difficult to assess the meaning behind a dysfunction and to reach a general conclusion.

Another difficulty in this experiment, was the limited population tested. Each laboratory must test their own normal subjects, for the results are not transferable (7). This creates a problem in the size of the population tested in regards to randomness and representativeness. I used very small populations for both the normals and the anorexics, and therefore may have inadvertently

incurred an error in lack of representativeness and randomness. This may have been avoided if more patients had been made available.

More testing is needed in order to make this study more conclusive. A greater population of anorexic patients should be tested against a greater population of normal subjects, and the background dietary information on the anorexics made more complete. The anorexic means in both the CTT and the conduction velocity were both below the normal mean values; this may be coincidence, but more work needs to be done in order to clarify this phenomenon and to insure conclusiveness.

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