EFFECTS OF PROTEIN ENERGY MALNUTRITION ON PERIPHERAL NERVE CONDUCTION AND AUDITORY EVOKED POTENTIAL RESPONSES IN CHILDREN

Dissertation submitted to

THE TAMILNADU Dr.M.G.R MEDICAL UNIVERSITY

In partial fulfilment of the

regulations for the award of the degree of

M.D. (PHYSIOLOGY)

BRANCH – V

Thanjavur Medical College and Hospital

The Tamilnadu Dr. M.G.R Medical University

Chennai, India

April 2015

CERTIFICATE

This is to certify that this Dissertation entitled "**Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction and Auditory Evoked Potential Responses in Children**" is a bonafied work done by Dr.S.Rubha, under my guidance and supervision in the Department of Physiology, Thanjavur Medical College, Thanjavur doing her Postgraduate course from 2012 -2015

Dr.K.MAHADEVAN , M.S.,
The Dean,
Thanjavur Medical College,
Thanjavur – 613004.

Dr.R.VINODHA, M.D.,
Professor and HOD,
Department of Physiology,
Thanjavur Medical College,
Thanjavur – 613004.

CERTIFICATE

This is to certify that this Dissertation entitled " **Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction and Auditory Evoked Potential Responses in Children**" is a bonafied work done by Dr.S.Rubha, under my guidance and supervision in the Department of Physiology, Thanjavur Medical College, Thanjavur doing her Postgraduate course from 2012 -2015

Dr.R.VINODHA,MD,

Professor and HOD,

Department of Physiology,

Thanjavur Medical College,

Thanjavur – 613004.

DECLARATION

I solemnly declare that this Dissertation " **Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction and Auditory Evoked Potential Responses in Children"** was done by me in the Department of Physiology, Thanjavur Medical College, and Hospital , Thanjavur under the Guidance and Supervision of my Professor **Dr.R.VINODHA, M.D.,** Department of Physiology, Thanjavur Medical College, Thanjavur between 2012 and 2015.

This Dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University , Chennai in partial fulfilment of University requirements for the award of M.D Degree (Branch – V) in Physiology.

Dr.S.Rubha
Postgraduate Student
Thanjavur Medical College
Thanjavur.

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA-613 001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

INSTITUTIONAL ETHICAL COMMITTEE

CERTIFICATE

Approval No. : 003

This is to certify that The Research Proposal / Project titled

EFFECTS OF PROTEIN ENERGY MALNUTRITION ON PERIPHERAL NERVE CONDUCTION AND AUDITORY EVOKED POTENTIAL RESPONSES IN CHILDREN

submitted by Dr. S. RUBHA **of**

Dept. of PHYSIOLOGY **Thanjavur Medical College, Thanjavur**

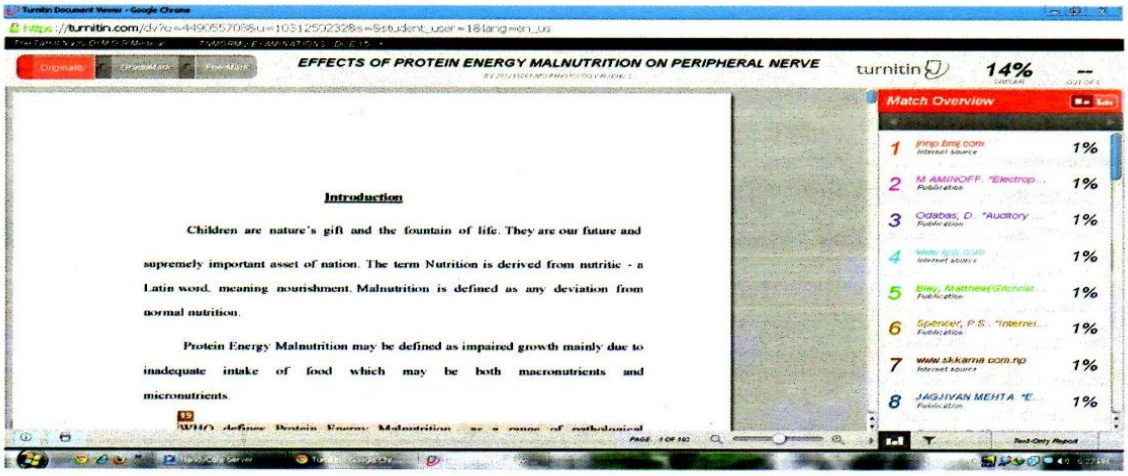
was approved by the Ethical Committee.

THANJAVUR MEDICAL COLLEGE THANJAVUR

Thanjavur
Dated : 06.12.2013

Secretary
Ethical Committee
TMC, Thanjavur.

ANTI PLAGIARISM – ORIGINALITY REPORT



ACKNOWLEDGEMENT

I thank the all mighty God for the abundant grace & blessings and making me successful in every field to complete the dissertation work.

I express my sincere gratitude to my Professor & HOD **Dr.R.VINODHA,M.D.,** for her motivation & meticulous guidance towards the successful completion of my Dissertation work.

I would like to thank the Dean, Thanjavur Medical College , Thanjavur for granting me permission to conduct this study at Thanjavur Medical College , Thanjavur

I would like to thank the Professor & HOD , Department of Pediatrics for permitting me to do this study.

I would like to thank the Professor & HOD , Department of Biochemistry for granting permission to use the laboratory.

I thank all the children , Parents / Guardians for their participation & cooperation to conduct the study.

Finally I thank all my family members for their encouragement & moral support throughout the period of study.

CONTENTS

ABSTRACT

TOPIC : Effects of Protein energy malnutrition on Peripheral nerve conduction & Auditory Evoked Potential Responses in children.

Aim :

Protein energy malnutrition affects the myelination and growth of the nervous system. The aim of this study is to determine the effects of protein energy malnutrition on Peripheral nerve conduction & Auditory Evoked Potential Responses in children.

Materials & Methods :

40 study group & 40 control group children were selected for this study. Study group includes 40 malnourished children of 5 – 10 years of age from Raja Mirasudar Hospital , Thanjavur based on anthropometric measurements like weight for age & height for age.

Control group consists of 40 normal children of same age group. Genetic & endocrine causes for short stature , children with ear pathology were excluded from the study. Informed written consent from the parents / guardians was obtained. Ethical committee approval was obtained before commensing the study. A detailed history & complete clinical examination were performed . Recording of BAER & NCS was done. Results were analysed statistically using student's 't' test.

Result :

There was a significant prolongation in wave I, II, III, IV latency , IPL I-III ,III-V & nerve conduction study showed reduced motor and sensory nerve conduction velocity ($p < 0.05$) in children with Grade III malnutrition. Children with Grade I, II malnutrition showed significant prolongation in IPL I – III and reduced sensory nerve conduction velocity ($p < 0.05$).

Keywords : Malnutrition Peripheral Nerve Conduction Interpeak latency Auditory Evoked Potential Response

INTRODUCTION

Introduction

Children are nature's gift and the fountain of life. They are our future and supremely important asset of nation. The term Nutrition is derived from nutritic - a Latin word, meaning nourishment. Malnutrition is defined as any deviation from normal nutrition.

Protein energy malnutrition may be defined as impaired growth mainly due to inadequate intake of food which may be both macronutrients and micronutrients.

WHO defines protein energy malnutrition as a range of pathological conditions arising from coincidental lack in varying proportions of proteins and calories, frequently occurring in infants and young children usually associated with infection. About 60 – 70% of children have mild to moderate malnutrition and the remaining are severely malnourished.[1]

Protein energy malnutrition is known to be a major health and nutrition problem in India.[2] Children having birth order greater than or equal to 3 and those not immunized had higher prevalence of Protein Energy Malnutrition.[3,4]

Protein energy malnutrition encompasses a wide spectrum of conditions, with Kwashiorkor the result of a greater deficiency of protein than energy intake at one end of the spectrum and Marasmus, resulting primarily from an inadequate energy intake.[5]

Dietary proteins are the source of brain enzymes and neurotransmitters. The quality of dietary proteins determines the quantity of cerebral proteins and neurotransmitters. Thus the amino acid profile of cerebral extracellular milieu is a function of dietary proteins.[6]

A marginally adequate diet, as weaning diets in developing countries does not meet these increased needs. Protein energy malnutrition is observed even in industrialized countries, associated with the presence of clinical conditions that decrease food intake or absorption of food. PEM is basically an exaggeration in physiological adaptation to inadequate nutrients.[5]

Malnutrition does not only risk the population for anemia and repeated infection, but it affects the developmental milestones and intellectual development. This persistent influence will lead to devastating effects in future. This burden continues in generations, as malnourished young girls become mother, deliver a malnourished young offspring.[7]

Undernutrition can cause developmental delays among the children and adolescents, leads to poor school performance and cause school dropouts.[8] Nervous system involvement in Protein Energy Malnutrition results not only from deficiencies of protein and energy alone but also from deficiency of micronutrient needed for brain growth and development.[9]

Iron deficiency alters myelination, Neurotransmitter synthesis, Hippocampal energy metabolism during neonatal period. Zinc deficiency alters autonomic nervous system regulation , Hippocampal & Cerebellar development. Long chain fatty acids are essential for synaptogenesis , membrane function and myelination.[10]

Malnutrition causes structural and functional pathology of brain. Effect of chronic Protein Energy Malnutrition causes stunting and wasting in children. It can also affect higher cognitive processes during childhood (> 5 yrs of age).[11]

Malnutrition leads to permanent suboptimal physical and mental development results in mental retardation.[12,13] Kinds of behaviours & cognitive functions are impaired by malnutrition which is related to altered emotional response to a stressful events.[13]

CNS injuries caused by severe malnutrition can be shown clinically and electrophysiologically.[14] Auditory Evoked Potentials are sensitive measures related to brain functions. Protein energy malnutrition and Iron deficiency anemia affect myelination & neural maturation of Auditory brainstem pathway[6]

Early onset malnutrition causes more abnormalities in AEP. These could be due to defect in myelination that ends in decreased synaptic efficiency in the auditory system. PEM slows the process of myelination, thus preventing the increase in caliber of myelinated nerve fibers.[15]

Measuring Nerve Conduction Velocity is a method of evaluation of status of peripheral nerves. Sachdev et al. & Kumar et al observed delayed motor nerve conduction velocities in children with protein energy malnutrition.[16] Delayed nerve conduction velocity in malnutrition is due to slowing or arrest of myelination that results from deprivation of nutrition.

Hence with the help of electrophysiological study we can find the nervous system alteration in children with malnutrition.

AIM & OBJECTIVES

AIM & OBJECTIVES

- Protein energy malnutrition affects the myelination and growth of the nervous system
- The aim of the study is to determine the effects of Protein energy malnutrition on peripheral nerve conduction and Auditory Evoked Potential Responses in children.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HEALTH

Health is a common theme in most cultures. Oldest definition for health is the absence of disease. Health may be defined as the condition of being sound in body, mind, or spirit, free from physical disease or pain.

WHO defines Health as , a state of complete physical, mental, and social well being not merely an absence of disease or illness. [17]

DIMENSIONS OF HEALTH : [18]

Health is multifactorial in nature. The dimensions of health are

1. **Physical dimension**

   It refers to perfect functioning of body. Signs of physical health are good complexion, clean skin, bright eyes etc.

2. **Mental dimension**

   Good mental health indicates the ability of an individual to respond to various experiences of life with relative flexibility.

3. **Social dimension**

   Social dimension means the quality and quantity of an individual's interpersonal ties and the extent of involvement with the community.

4. **Spiritual dimension**

   Spiritual dimension helps a person to reaches out and strives for meaning of life.

Determinants Of Health :

1. **Biological determinant**

   Genetic make – up is unique to an individual which cannot be altered after conception. A number of diseases are well known to be of genetic origin.

2. **Behavioural & Sociocultural conditions**

   The term life style denotes the way people live that reflects social values, attitude and activities. Health requires promotion of healthy lifestyle.

3. **Environmental conditions**

   - Internal environment : Body tissue, organ system with their perfect functioning.
   - External environment : The environment which is external to human host.

4. **Socioeconomic conditions**

   - Economic status
   - Education
   - Occupation
   - Political system

5. **Nutrition**

   Nutrients are the substances obtained from the food to provide energy.

They are

- Water
- Carbohydrate
- Fat
- Protein
- Vitamins
- Minerals

} Organic nutrients

Organic Nutrients :

Carbohydrate, Fat and Protein provide energy for body functions. Carbohydrate and fat are the major energy providing nutrients. Vitamins releases energy from carbohydrate & fat.

Inorganic Nutrients:

Minerals are inorganic nutrients. They yield no energy. They help in release of energy.

Unit of Energy :

Energy released from food is measured as kcal. It is the energy available in food.

- Carbohydrate - provides 4kcal in 1g
- Fat - provides 9kcal in 1g
- Protein - provides 4kcal in 1g

Proper Diet - Importance:

Basic purpose of giving nutrition is to achieve

(i) Physical Development

(ii) Immunity Development

(iii) Prevention of chronic disease in later life (eg) : Diabetes , Cancer etc

(iv) Prevention of Iron deficiency, undernutrition & Intrauterine growth retardation[19]

(v) **Nervous system development**

Development of central nervous system occurs by following process : After Conception three primary brain vesicles – the Prosencephalon, Mesencephalon and Rhombencephalon are visible as broadenings in the neural plate.

- During 5th week the prosencephalon subdivides into Telencephalon & Diencephalon. At the same time rhombencephalon subdivides into Metencephalon and Myelencephalon.[20]
- Hindbrain subdivides into Rhombomeres which are small repetitive segments. The extension of Neural tube caudal to rhombomeres forms spinal cord.
- Neural tube undergoes flexion at three points,
  - Mesencephalic flexure
  - Cervical flexure
  - Pontine flexure.

Of the three , mesencephalic and cervical flexure bends vertically and pontine flexure directs dorsally.[21]

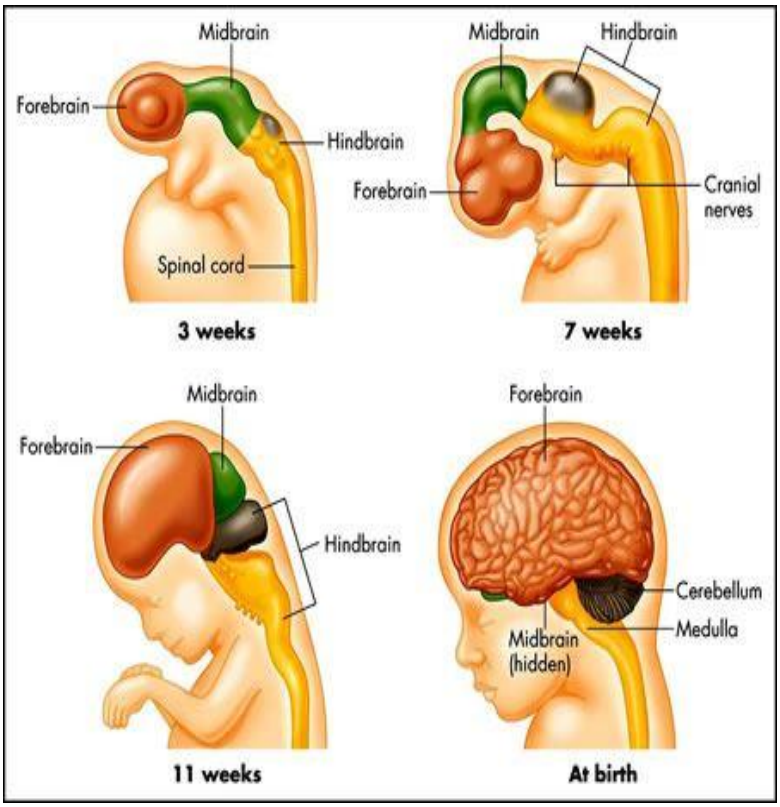
- At the end of 4th week cytodifferentiation of neural tube begins. During this process the neuroepithelium proliferates to produce neurons, glia and ependymal cells of central nervous system.
- Mantle zone is formed by young neurons in ventricular zone surrounding the central lumen. Mantle zone is the precursor of gray matter where majority of mature neurons are present.
- Axons extending from mantle layer produces marginal Zone (future white matter) surrounding the mantle layer.
- Mantle zone of spinal cord and brainstem is organized into a pair of ventral plates and a pair of dorsal plates. Laterally they abut at a groove called Sulcus limitans.
- Association neurons form in dorsal plates. One or two cell columns form in the ventral plates ; the somatic motor column and Visceral motor column.[22]
- Nuclei of 3 – 12 cranial nerves are located in the brainstem. Some of these cranial nerves are motor , some are sensory and some are mixed arising from more than one nucleus.
- Myelencephalon gives rise to medulla oblongata. Metencephalon gives rise to Pons ,a bulbous expansion that consists of massive white matter.
- Superior & inferior colliculus are visible round protuberance on dorsal surface of midbrain. Superior colliculus controls ocular reflexes & inferior colliculus serve as relays in auditory pathway.

- A special process of neurogenesis occurs in cerebellum gives rise to gray matter of cerebellar cortex as well as deep nuclei.

- The alar plate of diencephalon is divided into a dorsal & ventral portion by hypothalamic sulcus during 5th week. Hypothalamic swelling ventral to this groove differentiates into hypothalamus , which has function of controlling visceral activities like heart rate & pituitary secretion.

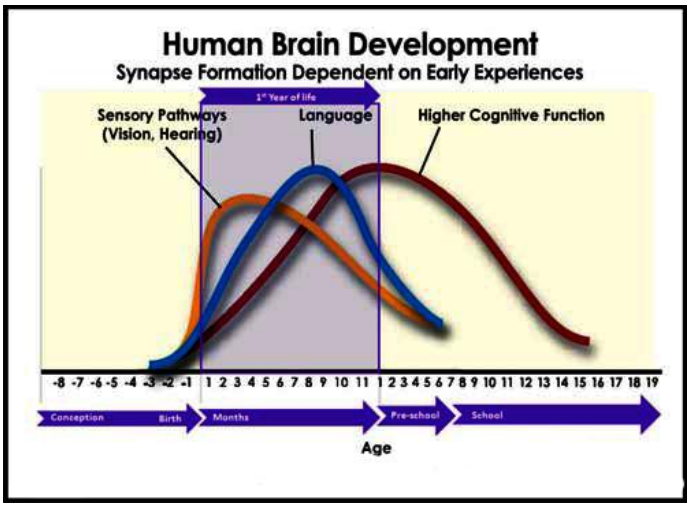
Figure 1 :

Development of Brain



- Dorsal to hypothalamic sulcus gives rise to thalamus which serve as a relay station, Information processor of subcortical signals before reaching cortex.
- During 6^{th} week epithalamus gives rise to pineal gland
- Infundibulum differentiates to posterior pituitary during 3^{rd} week. Rathke's pouch grows to meet infundibulum and become anterior pituitary.
- Telencephalon is subdivided into pallium and subpallium , where the latter forms basal ganglia (Corpus striatum , Globus pallidum)
- During 4^{th} month Cerebral hemispheres are initially smooth surfaced. Then it folds into sulci and gyri as the hemisphere grows. Two hemispheres are joined by corpus callosum and lamina terminalis.
- During 6^{th} week Olfactory bulb and tract arises from telencephalon and receives input from primary olfactory neurosensory cells
- The expanded primitive ventricles formed by neuralcanal in the secondary brain vesicles give rise to ventricular system on the Brain.
- The CSF fills this ventricular system which is mainly produced by choroid plexus in the lateral, third and fourth ventricle that are formed by ependymal and vascular piamatter.

Figure : 2



The above picture shows the time of brain development . Sensory pathways for hearing & vision , language development , higher cognitive function all starts developing prenatally. Development of language & sensory pathways continues upto 6yrs of life. Development of higher cognitive function occurs till 16 years.

So any nutritional deprivation during this period will cause marked functional changes.

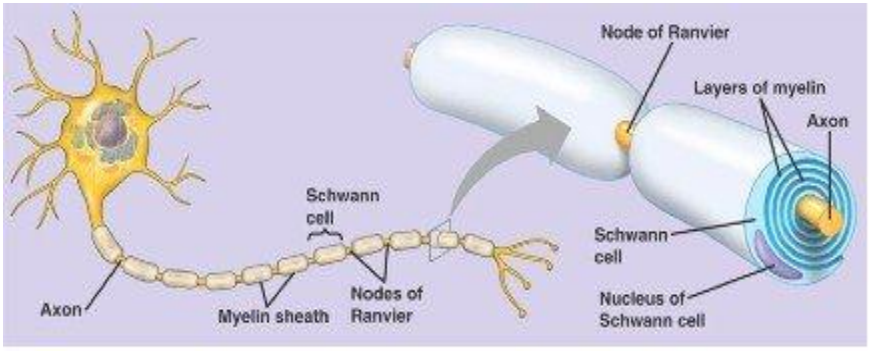
GROWTH OF BRAIN : [23,24]

Brain at birth is of 25 % of adult volume. Postnatal growth of brain results in increase in size of neuronal cell bodies and proliferation of neuronal processes. Brain reaches its final size at around 7 years of age. Most of these growth results from myelination of nerve fibers.

Myelinogenesis :

Myelinogenesis is a process of sequential myelination or development of myelin sheaths around the nerve fibres of central nervous system.

Figure : 3



Function of myelin :

Myelination allows neural signals to propagate with less signal loss. Thus they produces better connectivity between various brain regions. It has been found that myelination is the key process for speed of conduction.

History :

Paul Flechsig studied and published the details of process of myelination in cerebral cortex of human beings.

- The first cortical region to myelinate is the motor cortex (Part of Broadmann's area 4)
- The second part to myelinate is the olfactory cortex
- The third part to myelinate is Somatosensory cortex (Brodmann's area 3, 1, 2)

- The last areas to myelinate are anterior cingulate gyrus, inferior temporal cortex & dorsolateral prefrontal cortex.

Stages of myelination :

1. Axon contact
2. Glial cell gene production
3. Axonal ensheathment
4. Maturation

Myelination is an important process for conduction of nerve impulses as it determines the speed of conduction . Any factors that delays myelination will affect nerve conduction. In children most important factor is nutritional status. Malnutrition will adversely affects myelination and central nervous system development.

Malnutrition:

Malnutrition results from a diet that does not satisfy nutritional requirements.

Primary Malnutrition :

Caused by social and economic factors like

- Poverty
- Inadequate food
- Recurrent Infections
- Lack of health care

It is common affecting quarter of world's children because many live in poverty in the developing world. It contributes to about 60% of all child deaths.

Secondary Malnutrition :

Malnutrition resulting from disease like Inflammatory bowel disease.[12,25]

Pathophysiology of PEM :[1]

Malnutrition occurs because of deficiency of protein and calorie intake.

There are two different entities

(i) Edematous Malnutrition - Kwashiorkor & Marasmic kwashiorkor

(ii) Nonedematous Malnutrition - Marasmus

The cause for the different manifestations of these diseases are still not clear. However the following theories have been proposed.

1. **Classical Theory :**

Proposed by Williams, which describes

- Marasmus occurs due to protein and calorie deficiency
- Kwashiorkor occurs due to high carbohydrate, low protein diet, associated with late gradual weaning, starchy family diet & repeated infections contributing to edematous malnutrition occurs due to early abrupt weaning followed by starvation & infection contributing to wasting.

2. Dysadaptation Theory :

- Proposed by Gopalan.

This theory suggests that children with marasmus adapted to deficient protein calorie intake. There occurs loss of fat and degradation of muscle protein secondary to high cortisol and low insulin levels. The protein thus derived was used for production of proteins including albumin. So, no edema and hypoalbuminemia.

In Kwashiorkor, this adaptation did not occur. The dietary protein was used for energy production and for production of acute phase reactants in response to repeated infections. There was no muscle protein available - hence hypoalbuminemia occurs.

3.Free Radical Changes :

This theory is based on an imbalance between free radical production and elimination by antioxidants and oxygen radical scavengers.

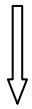
Free radical induced injury to the cells cause membranes to leak and results in edema.

Infection increase the oxidant stress in kwashiorkor where the proteins are used for production of acute phase reactants and there is inadequate substrate for albumin production, results in hypoalbuminemia and edema.

HOMEOSTASIS IN UNDERNUTRITION [19]

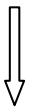
Simple Undernutrition : (Adaptation)

Glycogen stores and proteins convert to glucose

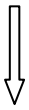


Later fat stores are drawn , provides ketones to fuel metabolism

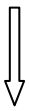
Conservation of enzymes



Utilization of proteins to form glucose and protein needs



Body minimises glucose needs.



Conservation of enzymes and protein stores

Severe undernutrition :

Glycogen stores and protein forms glucose. Stress results in increased ketone production. Body continues to produce glucose and stress factors by depleting protein stores. Metabolic rate rises and body's total energy need remains high leading to accelerated loss of energy and protein stores.

Coexistence of undernutrition and stress :

Infection / Injury

⇩

Potentiating nutrition depletion rate

⇩

Reduction in body weight

⇩

Weakened immunity - Additional Stress

⇩

Compromised recovery

⇩

Worsening of malnutrition

Immunological Changes in severe malnutrition :

Reduced lymphocyte number, cell mediated immunity and phagocytosis occurs in severe malnutrition. The levels of other immune mediators including complement also decreases in PEM. Low protein intake results in a decline in both cell mediated and antibody based immunity.

Prevalence of malnutrition : [26]

Malnutrition is like an iceberg. Most people in the developing countries live under the burden of malnutrition.[17] Despite improvement in life expectancy , adult literacy an estimated 780 million people in low income countries lack sufficient food. One of every 5 persons in developing world is chronically undernourished , 192 million children suffers Protein energy malnutrition & around 2000 million is deficient in micronutrients.

Etiology : [27]

1.Poverty : [28]

A poor cannot purchase adequate amount of food of desired quality for nutritional requirements. This deprivation affects their physical work resulting in low earning and poverty.

2.Low Birth Weight :

Malnourished mothers gives birth to low birth weight and growth retarded babies. In India and Bangladesh 30% of total live births are low birth weight babies, because of malnourished mothers and poor lactational performance.[29]

3.Infections :

- Malnutrition makes the child susceptible to infection, recovery becomes slower with high mortality.[17]

- Infections such as diarrhea, pneumonia, malaria, measles precipitate acute malnutrition and aggravate existing nutritional deficit.
- Metabolic demands for protein during infections are higher. Malnutrition may affect immune status and make the children vulnerable to infections. Thus forming malnutrition – infection – malnutrition cycle. This cycle shows that malnourished children are prone to develop infections like diarrhea and pneumonia which may adversely affect immunity which inturn precipitate malnutrition.

4. Population Growth :

Large families and higher birth order results in malnutrition. Rapid succession of pregnancies affects nutritional status of mother. Preoccupied care of a large sibship prone to neglect her own health and antenatal checkup during pregnancy. Her baby tend to be small resulting in higher incidence of low birth weight.[30]

5. Feeding Habits :

- Lack of exclusive breast feeding for first 6 months makes a child prone to get early development of malnutrition.
- Poor quality of the substitute milk, excessive dilution and use of unhygienic feeding bottles cause disastrous effect on baby.
- Some foods are mistaken to be hot or cold in nature or likely to produce liver disease. Irrational beliefs about nutritional needs of

infants, cultural taboos on food of certain types leads to malnutrition.

Shaheen Banu et al found that there is significant decrease in serum Iron, ferritin and total iron binding capacity in children with malnutrition when compared with normal healthy children[31]

6.Advertising Baby Foods :

High pressure advertising by food manufacturers and social demands on urban educated working women causes early discontinuation of breast feeding.

Unhygienic feeding practices in preparation of milk formula results in high incidence of diarrhoea and diminished absorption of food.

7.Social Factors :

- Repeated pregnancies, food taboos, broken homes, separation of a child from parents are important social factors that plays a role in development of protein energy malnutrition.[32]
- Natural disasters like floods, earthquakes shifts the nutritional balance towards the negative side.[27]
- The major associated risk factors for acute malnutrition were older age of child, undernourished mother, jobless father / father with a low paying job, low total family income.[28,30,33]

8.Intrauterine growth retardation & fetal malnutrition :

Malnutrition does not start after birth but it is a continuation of intrauterine malnutrition. 20 – 40 % of babies born in India have low birth

weight because the mothers are malnourished often presenting with severe anemia and chronic diseases . Continuing malnutrition in postnatal period produces severe damage to development of Brain which has its maximum growth spurt in last 3 months of pregnancy and in the first 2 years of life.[34,35]

EFFECTS OF MALNUTRITION :

- Reduced protein synthesis
- Lower neuronal number
- Decease in cellularity[36] & hypomyelination[10,14]
- Reduced oxidative metabolism in hippocampus & frontal cortex
- Impairment of synaptogenesis[13]
- Change in cortical glial cell density & reduced number of cortical dendrites[26]
- Reduction in number of norepinephrine receptors causes reduced down regulation, a functional consequences that diminishes the ability to adapt to stressful situations.[13]
- **Hippocampus :**

Reduction in the degree of dendritic branching & the number of granule cells. The number of synapses per neuron shows completely different pattern.[37]

- Delayed neurointegration of special senses.[38]

- **Cerebellum :**

  Increase in density of purkinje cells & granule cells. Abnormal electrophysiological activity in purkinje cells and suppression of synapse neuron ratio also occurs in malnutrition.[14]

CLINICAL MANIFESTATION OF PEM

The effect of protein energy malnutrition on community is graded by anthropometric measurements like weight for height / age into mild , moderate and severe forms and the individual cases with PEM are defined by Clinical forms (Kwashiorkor, Marasmus & Intermediate forms) [26]

Clinical manifestation Depends on

- Severity
- Duration
- Age of onset of nutritional lack of different food
- Presence or absence of associated infections

Mild to Moderate undernutrition :

- If the dietary intake is deficient for short period, a child adapts its metabolism in order to compensate for the deficit. Energy in the diet are expended efficiently and nitrogen excretion in the urine and stool is reduced.
- If the food deficit persists for a longer period a child conserves its energy by curtailing physical activity. Moderate malnourished children appear slow and less energetic.
- Growth lag is more pronounced in weight than length. With prolonged deprivation , length is also reduced. As nutritional deficit exaggerates with onset of infections, a child may become marasmic or develops kwashiorkor.
- Nutritional marasmus and kwashiorkor are two extreme forms of malnutrition.

MARASMUS :

Features :

- Emaciation (Body weight < 60 % of expected weight for age)
- Marked stunting
- No edema
- Depletion of adipose tissue fat because it is used up for energy production

- Dry, Scaly inelastic skin prone to be infected

- Hypopigmented hair

- Distended abdomen due to wasting & hypotonia of abdominal muscles

KWASHIORKOR :

Kwashiorkor is an african word which means , " the disease that occurs when the child is displaced from the breast by another child "

Features :

- Retarded growth
- Psychomotor changes
- Edema due to prerenal diversion of fluid from capillary bed to exteacellular space. Other causes are increased capillary permeability, hypoalbuminemia, free radical induced damage to cell membranes.
- Lethargic, listless, apathetic child
- Hepatomegaly – Liver is enlarged due to fatty infiltration with soft in consistency
- **Hair changes** : Thin, dry, easily pluckable hypopigmented hair

Alternate appearance of bands of hypopigmented & normal pigmented hair produces Flag sign

- **Skin changes** : Areas of erythema followed by hypopigmentation. They may desquamate to expose raw, hypopigmented skin that gives appearance

of old paint flaking off the surface of the wood (Flaky paint dermatosis). Underlying raw skin is easily infected.

- **Recurrent infections** : Diarrhoea , respiratory and skin infections are common.

CLASSIFICATION OF PEM :

The following classifications have been proposed based on clinical, anthropometric & biochemical parameters.

GOMEZ's classification : First classification to be proposed

Weight of the child	Grade of malnutrition
90 – 110 % of standard	Normal
75 – 90 % of standard	I degree malnutrition (Mild)
60 – 74 % of standard	II degree malnutrition (Moderate)
< 60 % of standard	III degree malnutrition (Severe)

Standard weight for age used here is Harvard growth standard , 50^{th} centile being 100% [(2)]

IAP CLASSIFICATION [39]

Grade	% of standard weight for age
Normal	>80 %
Grade I	71 – 80 %
Grade II	61 – 70 %
Grade III	51 – 60 %
Grade IV	≤ 50 %

Standard value : 50th percentile of Harvard growth standard

Waterlow Classification : [40]

Anthropometry	Normal	Wasted	Stunted
Weight / Age (%)	100	70	70
Weight / Height (%)	100	70	100
Height / Age (%)	100	100	84

WHO Classification :[41]

	Moderate undernutrition	Severe undernutrition
Symmetrical edema	No	Yes
Weight for height (measure of wasting)	SD -2 to -3 (70 – 79% of expected weight)	SD score < -3 (<70 % of expected)
Height for age (Measure of stunting)	SD -2 to -3 (85 – 89% of expected)	< 85% of expected

$$\text{SD (Z) Score} = \frac{\text{Observed value} - \text{Expected value}}{\text{Standard deviation of reference population}}$$

Median (50^{th} percentile of NCHS standard)

Arnold's classification

Nutritional status **Midarm circumference (cm)**

Normal ---------- ≥ 13.5

Mild to moderate ---------- 12.5 – 13.4

Severe PEM ---------- ≤ 12.4

Jellife's Classification :

Grade	% of standard weight for age (50^{th} centile of Harvard standard)
Normal	More than 90%
Grade I	80 – 90 %
Grade II	70 – 79 %
Grade III	60 – 69 %
Grade IV	Less than 60 %

ASSESSMENT Of NUTRITIONAL STATUS : [19,42]

1. **Dietary history**

   - Vegetarian / Non vegetarian
   - Recent intake of food & fruits
   - Usual diet before the current illness

2. **Clinical evaluation**

   - Weight loss

- Presence of edema
- Duration & frequency of any illness
- Features of infection
- Specific nutrient deficiency e.g. Anemia, rash, stomatitis, glossitis

3.Anthropometry

- Height
- Body weight
- Body mass index
- Mid arm circumference

<u>Age dependent criteria</u>

1.Weight for age :

Commonly used & most sensitive method for supervising growth. Weighing is an easy method for a medical as well as for a paramedical worker . It can be expressed by median & standard deviation values or as percentage of reference value (median value = 50^{th} percentile).

2.Height for age :

A child's height is compared with expected height of a healthy child of same age group. Changes in height occurs slowly, so it is not useful in early diagnosis of malnutrition.

AGE INDEPENDENT CRITERIA :

1.Midarm circumference (MAC)

MAC is relatively constant from 1 to 5 years around 16.5 to 17.5cm. In this age group MAC < 12.5cm is considered as undernutrition. It is a useful method in screening large number of children in nutritional emergencies.

2.Weight for Height

It can be used to distinguish different types of malnutrition. In chronic malnutrition, the child is stunted. In acute malnutrition his height is appropriate but he is wasted (low weight for height & age). It is a useful method in differentiating current malnutrition & longterm malnutrition.

3.Skinfold thickness

It is an indicator of caloric stores availability in subcutaneous fat.

Sites for measurement :

- Triceps
- Subscapular region

Skin fold thickness measurement below 90 % of standard is subnormal.

80 – 90 % ---------- Mild malnutrition

60 – 80 % ---------- Moderate

< 60 % ---------- Severe malnutrition

Management :

Complete management of malnutrition means complete catch up growth (Gaining weight greater than 50th percentile for age , which may require 150 % or more of calorie recommended for an age matched, well nourished child) [25] followed by sustained normal growth , health & developments.

Management of initial phase (1 – 7 days)

- Assessment
- Management of complication

1. Hypothermia (Axillary temperature < 35°C) – Wrap especially at night
2. Hypoglycemia (Blood sugar < 54mg / dl) that occurs due to reduced glycogen stores & increased utilisation.

3. Dehydration occurs if the child has diarrhoea. Rehydration solution of choice is RESOMOL (with less sodium , more potassium, magnesium, Zinc & Copper)[12]

   - **Nutritional management :**

1. **Energy / Calories :** Initial intake should be 80 – 100 kcal /kg /day that should be gradually increased to about 10 – 12 kcal /kg /day.
2. **Protein :** 6 – 12 % of total calories should be from proteins. Start at 1 g/kg/ day & increase to 2 – 3 g / kg / day
3. **Fat :** upto 50 % of total energy.
4. **Carbohydrate :** 40 – 45 % of total intake

Management of recovery / rehabilitation phase : (2 – 6 weeks)

- Nutritional management – Mainstay of treatment
- Physical & emotional stimulation
- Educating family

The goal is to achieve > 10g / kg / day

Followup phase : (6 weeks – 6 months)

- Nutritional rehabilitation & continued care at home

- Monitoring & home visits which should be continued till the child reaches > 90 % of weight which usually takes 6 – 8 months.

Role of nutritional rehabilitation centre :

" Give a malnourished child food , it will relieve child's hunger , but teaches a mother how to feed & it will have longterm benefit "

Nutritional rehabilitation centres are practical training centres where the mother is taught to feed the child using nutritional diet from locally available & cheap food. These centres can be residential or with day care facilities where the former being ideal.

Nutritional recovery syndromes :

Some children manifests recovery syndromes if the nutritional status is rapidly corrected.

They are

- Pseudotumour cerebri
- Gomez syndrome - Ascites, Hepatomegaly, Parotid swelling, gynaecomastia
- Kahn syndrome - CNS manifestation such as tremor , rigidity & myoclonus.

PREVENTION

1. Early contact between mother & child
2. Support & promote breast feeding practices
3. Growth & development monitoring
4. Early intervention if there is any deviation from normal growth
5. Social & health support to malnourished child
6. Parent education of norms of growth & nutrition
7. Basic health care facilities for children to prevent infection & to provide immunisation
8. Improved socioeconomic condition
9. Enforcement of National programmes to improve nutrition.

Undernutrition & Learning abilities :

- Active brain growth extends from 30^{th} week of gestation to around the end of second year of life.
- Malnutrition during first 6months of life adversely affect the development of brain. They may show poor intersensory organisation among Visual & Kinesthetic sensation for geometric forms recognition.
- Severely malnourished children shows significant lag in IQ compared to siblings & peers and the lag persists even after recovery.[27]

- **Dilek Dilli et al** showed low developmental index in infants of very low birth weight born with sepsis.[43]

- Severely malnourished children revealed that they had IQ of 60 because they went through malnutrition in early childhood.[28]

- Follow – up studies on survivors of 6 – 8 years after recovery from overt malnutrition shown abnormal neurointegrative function and lower intelligence compared with controls.[44]

Electrophysiological assessment :

Evoked potential study is used to evaluate the functional integrity of afferent pathways in central nervous system physiology. They are noninvasive and have excellent temporal resolution.

Evoked potentials :

Evoked potentials are small electrical potentials arising from neural tissue along sensory pathways in response to stimuli.[45]

Brainstem auditory evoked potential response :

Brainstem auditory evoked potentials are the potentials recorded from ear and vertex in response to a brief auditory stimulus to assess the conduction in auditory pathway upto midbrain.[46]

It comprises of five or more waves within 10ms of the stimulus, which for clinical purposes is a monoaural click. This is usually set about 65db above the hearing threshold which is delivered by headphone and the opposite ear is masked by white noise. BAEP is of very low voltage, around 1000 & 2000 responses are recorded so that BAEP can be extracted by averaging from background noise.

If the electrical square causes the headphone diaphragm to move towards the patient's ear, a condensation click is produced. Reversing the polarity of square pulse produces a rarefaction click. Rarefaction clicks are generally preferred as they yield BAEPs with better waveform components.[47]

Early auditory evoked potentials :

They are short latency auditory evoked potentials which are recorded within first 10 – 12msec after an auditory stimulus.

Middle latency auditory evoked potentials :

These potentials are recorded between 10 – 50msec after an auditory stimulus. It can be recorded from transient or high frequency stimuli.

Late auditory evoked potentials :

Evoked potentials occurring 50msec after the auditory stimulation are called late auditory evoked potentials. They can be subdivided into exogenous components N1, P1, and P2 that depends on external stimuli and endogenous components such as P300 and N400.[48]

Brainstem electrical activity & its correlation with BAEP :

The acoustic nerve and brainstem auditory potentials are volume conduction to surface recording electrodes. As the vertex and earlobe, they form vertex positive and vertex negative waves known as BAEPs.

Wave I originates from peripheral portion of VIII cranial nerve adjacent to cochlea.

Wave II originates from cochlear nucleus.

Wave III originates from superior olivary nucleus.

Wave IV originates from lateral lemniscus.

Wave V from inferior colliculi.[46]

POSTNATAL MATURATION :

Brainstem auditory evoked potential response recorded from infants within few hours after birth differs from those recorded after a day of birth. BAEP can be recorded from premature infants as young as 25 weeks of gestational age. Amplitude increases and at term they approach adult values.[48]

Wave I is delayed just after birth by 0.8msec, due to residual fluid in middle ear. Significant difference is observed in I – V interval of 0.2msec indicates central changes during first day of life.[49]

In normal children BAEP to monoaural stimulation matures to adult pattern by 3years of age.[50]

Wave I latency reaches adult values by 2months. Wave III & V shows a rapid decrease in latency over first several months & reaches adult values at the end of third year.

Amplitude of Wave V markedly increases after 6months but does not reach adult values until 5years of age.

Binaural interaction is present at birth with latencies and amplitudes that are relatively same to monaural response in adults.

Threshold for detecting BAEP decreases by about 10dB in first 3months of life & by 5dB further by the end of first year.[49,51]

Maturation of BAEP peak latency (msec)[48]

Age	Wave I	Wave II	Wave III	Wave V	I / V ratio
7 – 12 months	1.69 ± 0.13	2.77 ± 0.2	4.1 ± 0.17	5.98 ± 0.23	4.29 ± 0.19
1 – 2 years	1.8 ± 0.19	2.79 ± 0.23	3.99 ± 0.23	5.79 ± 0.3	3.99 ± 0.18
>2years	1.68 ± 0.13	2.69 ± 0.17	3.86 ± 0.2	5.68 ± 0.4	3.96 ± 0.14

Normal BAEP wave forms : [52,53]

Wave I

This is the prominent upgoing peak in ipsilateral ear recording channel.

It appears 1.5msec after the stimulus

It is absent in patients with peripheral hearing impairment

Wave II

This is a small peak following Wave I. It may appear in downgoing or upgoing slope of Wave III with Wave latency of 2.8msec.

It is absent in cochlear nucleus lesion.

Wave III

This is a prominent upgoing peak with latency of 4msec usually absent in superior olivary nucleus lesion.

Wave IV

Usually it appears as a small wave in the upgoing slope of wave V. It has a latency of 5.1msec. It is absent in lateral lemniscal lesion.

Wave V

This is the most prominent peak in BAEP. It appears 5.5msec after the stimulus & starts above the baseline. It is absent in inferior colliculus lesion.

Measurement of BAEP Waveforms :

1. **Absolute latency**

   It is measured as the distance (ms) from beginning of the first wave to the peak of that wave.

2. **Amplitude**

   It is measured as the distance (μv) from the peak of the wave to the trough of that wave.

3. **Interpeak latencies**

   Commonly measured interpeak latencies are I – V, I – III, III – V . This is measured as the distance between the peak of both waves (ms)

I – V interpeak latency :

It is the measure of conduction from proximal VIII nerve to midbrain through pons. Normal value is 4msec. It is prolonged in demyelination , ischemia & in tumours.

I – III interpeak latency :

This is the latency difference between wave I and III, measure of conduction from VIII nerve across subarachnoid space. Normal value is 2.1msec. It is prolonged in tumours or inflammation affecting proximal portion of VIII nerve.

III – V interpeak latency :

It measures the conduction from lower pons to midbrain. Normal value is 1.9msec.

Factors affecting BAEP waveforms :

1. **Age**

`Latency is affected by age especially in early childhood. Latency is age dependent upto 2years. This effect is more pronounced in premature infants. Older adults have little longer I – V interpeak latency when compared to younger individuals.

2. **Sex**

Women have shorter latency and higher amplitude of BAEP. The I – V Interpeak latency is shorter by 0.1ms in females compared to males which may be due to higher core body temperature and shorter length of brainstem auditory pathway.

3. **Temperature**

Increased body temperature decreases the latency and decreased body temperature increases the latency. 0.17ms increase in occurs in wave V latency on 1°C reduction of body temperature.

At 32.5°C BAEP becomes abnormal & at 27° C the waveforms disappears.

4. **Drugs**

Barbiturates & alcohol prolongs the latency of wave V. They produce these effects by lowering body temperature.

5. **Hearing loss**

Hearing impairment alters BAEPs. So hearing tests to detect conductive deafness should be done before recording BAEPs.

Clinical uses of BAEP :

- BAEP can be used to evaluate hearing in infants, young children and adults who are unable to cooperate for behavioural testing
- BAEPs provide a sensitive screening test for acoustic neuromas or other lesions in cerebellopontine angle involving VIII cranial nerve.
- It is a useful method in assessing the integrity of brainstem where it localises the lesion.
- BAEPs have been used to detect subclinical brainstem pathology in patients with suspected Multiple sclerosis
- Use of BAEPs in the fitting of hearing aids is to provide an accurate audiogram which cannot be obtained behaviourally

- Hydrocephalus is associated with severe BAEP abnormalities, which may be related to ventricular dilatation. BAEP results can be improved when the child is successfully treated with a shunt.

- Myelin disorders initially delay the later components of BAEP. As the disease progresses, later components decrease in amplitude & finally disappear until only wave I is present. These findings are due to desynchronization that occurs before definite auditory symptoms.

- BAEPs recorded from children with anoxic brain damage are abnormal. Prolonged anoxia results in increased I – V interpeak latencies. Absent or abnormal BAEPs in a comatose child is a bad prognostic sign.

- Abnormal BAEPs are found in patients with autism. There is abnormally delayed interpeak latencies or raised threshold.

ADVANTAGE & DISADVANTAGE :

The BAEP provides important information about a child's cochlear and brainstem function. It can indicate dysfunction but cannot determine specific etiology of this dysfunction.

It has much advantage that it does not need the cooperation of the child. It is unaffected by sleep or sedation.[48.49]

NERVE CONDUCTION STUDY (NCS) : [54,55,56.57]

Nerve conduction studies examines Peripheral motor & sensory nerve function by recording the evoked potential in nerve or muscle in response to electrical stimulation of a peripheral nerve.

HISTORY

Motor nerve conduction studies were first described for clinical use in 1948.[58] Sensory nerve conduction studies were first demonstrated by Dawson and Scott in 1949 & were shown to be of clinical value in 1953 by Gilliatt and Sears.[57]

GENERAL PRINCIPLE IN NERVE CONDUCTION TESTING: [56]

- Both motor and sensory nerve studies should be performed when possible because certain disease may affect motor or sensory axons and staging requires knowledge of degree of compromise in motor or sensory axons .
- Several segments of the nerve suspected to be involved should be examined to know whether the disease is localised or it involves long nerve segments.
- Nerve contralateral to those of suspected of involved should be tested because side to side comparison of latencies and conduction velocities may enhance the sensitivity of testing that may reveal presence of conduction defect in the absence of clinical symptoms.

- Nerves in both upper & lower extremities should be tested if symptoms exists in both the areas as polyneuropathic diseases often produces bilateral and symmetrical involvement of both upper and lower extremity.

- Testing should be done at appropriate time in context of suspected disorder.

Electrodes :

- Surface electrodes gives information about the whole of the muscle stimulated.

- Needle electrodes gives accurate conduction time when depth of muscle under study makes a surface recording impossible. These electrodes gives more accurate information when there is severe muscle wasting.

Technical Errors :

They are the common source of error in Motor & Sensory nerve conduction studies. Any unexpected finding should be assumed to be due to technical error unless proved otherwise.

Spread of current due to excessive stimulation , small responses because of submaximal stimulation , when the stimulator is not over the nerve , incorrect limb positioning, inappropriate values , incorrect location of recording electrodes must all these to be watched and eliminated.

Risks :

Nerve conduction study is risk free in normal individuals. Nerve conduction can be risk if the current reaches the heart. It is contraindicated in patients with catheters inserted directly into heart. Stimulation near the heart has to be avoided in patients with pacemakers, cardioverters and defibrillators.[59]

MOTOR NERVE CONDUCTION STUDY (MNCS) :

The active electrode is placed over the muscle belly & the reference electrode over an electrically inactive site (muscle tendon). Ground electrode is placed between the stimulating and recording electrode providing a zero voltage reference point. Supramaximal stimulation is given at wrist & action potential is observed. The latent period is measured as the interval between the stimulus artifact & first negative deflection. Then stimulate the nerve at elbow. Action potential is observed and recorded. Latency period is noted.

Distance between the wrist & the elbow points of stimulation is measured in mm.

calculate the nerve conduction velocity (m / sec) by the formula :

Distance (mm) / Latency difference.

SENSORY NERVE CONDUCTION STUDY :

Sensory nerve action potential (SNAP) is obtained by electrically stimulating sensory fibres & recording the nerve action potential at a point further along the nerve. Recording SNAP orthodromically refers to distal nerve stimulation & recording more proximally (the direction in which physiological sensory conduction occurs). Antidromic testing is proximal nerve stimulation and distally recording the action potential.

Ring electrodes commonly used to measure SNCV. They are placed at the index finger with the help of conducting gel. Ground electrode is placed over forearm. Stimulation is given at wrist. Sensory nerve action potential was recorded using antidromic conduction. Latency was measured from the stimulus artifact to the first negative deflection from the baseline. The distance between the recording electrode & the stimulation site is measured. Sensory nerve conduction velocity is measured by the formula, **distance / onset on latency** .

F Waves

- F Waves (F for foot where they were first described) are a type of late motor response.
- When a motor nerve is electrically stimulated at any point an action potential is propagated in both directions of stimulation site. The distally propagated impulse gives rise to compound motor action potential. The

impulse conducts proximally to anterior horn cell, depolarising the axon hillock causing the axon to backfire. This leads to small muscle action potential (F Wave) at longer latency.

These F Waves allows testing of proximal segments of nerves which are inaccessible to routine nerve conduction studies. F Wave abnormality is a sensitive indicator of peripheral nerve pathology.[54]

PHYSIOLOGICAL VARIABLES AFFECTING NCS :

1.Temperature

- Low temperature results in slower conduction velocity , longer distal latency & higher muscle action potential amplitude.

- Increase in temperature increases conduction velocity by 5 % per degree. So it is important to perform nerve conduction test in temperature controlled environment (21 - 23°C) with skin temperature of the feet > 31°C and of the hand > 33°C.

  It has been found that values of conduction velocity along the segment wrist to elbow is significantly higher than along the distal fingers to wrist segments because of difference in temperature between these two segments.[60]

- For each degree celcius fall , the latency increases by 0.3ms , which may be due to cooling of sodium channels.

2. Age

Infants younger than 5 years have conduction velocity as low as 50 % of adult value due to incomplete myelination. Conduction velocity changes markedly with age between neonatal period & 6yrs with subsequent changes till adulthood caused by growth.

Conduction velocity attains adult value by 3 – 5 years of age. Conduction begins to slow after 40 yrs. The effect of age is more marked in median nerve compared to radial nerve.

3. Gender

Women generally have larger sensory nerve action potential when compared to men for both median and ulnar nerves They have shorter latency and higher nerve conduction velocity when compared to males.[56]

4.Extremity

Upper extremity have faster nerve conduction by 7 to 10m / sec than lower extremity. Usually the proximal segments conducts faster than distal segments.

5. Body Mass Index

Higher BMI produces lower SNAP in upper limb nerves.

6. Length of nerve

Longer nerve conducts slower than the shorter nerves. The reason for this difference are

- Abrupt distal axonal tapering

- Short internodal distance
- Progressive reduction in axonal diameter
- Low temperature of feet when compared to hands.

Technical variables

1. **Stimulating system**

   Failure of stimulating system results in small response. The stimulus may be a submaximal stimulus or in may not reach the target. In cases of edema needle electrodes may be used.

2. **Recording system**

   Faulty connection in recording system may result in errors. They include

   - Breaks in electrode wire
   - Connection to a wrong amplifier
   - Incorrect settings of instrument

3. **Stimulation of Unintended nerves**

   Spread of stimulation current to adjacent nerve which is not under study in frequently encountered. Here comes the importance of using needle electrodes especially in testing the innervation of individual motor branches. Needle electrode recording is not reliable for amplitude measurement as it records the electrical activity from restricted area of a muscle.

CLINICAL USES OF NERVE CONDUCTION STUDY :

- Motor conduction studies are helpful in indicating that weakness is due to pathology of peripheral nerves rather than other parts of the motor unit. Sensory conduction studies indicates that sensory symptoms are due to an impairment of peripheral nerve function or, when normal, to a lesion proximal to the dorsal root ganglia.[47]
- Principal diagnostic tests in suspected median neuropathy are nerve conduction studies. In Carpal tunnel syndrome the underlying pathophysiologic condition is demyelination of median nerve.
- Location of lesion in the length of a muscle
- Pathophysiology of different neurological diseases
- Peripheral nerve neuropathy (Involvement of axon or myelin)
- Diagnosis of isolated mononeuropathies
- Diagnosis of conduction blocks (eg. Local anaesthetic block) produces reduction in amplitude
- Prolonged latency is observed in segmental demyelination where slowing of conduction occurs.
- In Wallerian degeneration there is reduced or absent response.

BAEP findings in malnutrition :

Manuel Roncagliolo et al did auditory brainstem responses in 26 infants (6 months old) with iron deficiency anemia & compared them with 26 nonanemic infants of same age group. They observed that prolongation of absolute peak latency of wave III , V & central conduction time (I – V) which is a index of CNS development in infants with iron deficiency anemia suggesting the essential role of iron in myelin formation .[61]

Vandana & O.P Tandon compared 20 chronic malnourished children of 3 – 6 years of age with 20 healthy age & sex matched healthy controls & found that prolongation of absolute peak latencies of wave I, II, III, IV with prolonged I – III & III – V interpeak latencies in children with chronic malnutrition characterized by stunting suggested the effect of chronic malnutrition affecting the peripheral developmental process of auditory pathways in brainstem.[62]

Il Hong Moon et al studied auditory evoked potential in 183 premature infants & divided them into AGA – appropriate for gestational age , SGA – small for gestational age. They recorded auditory evoked potential to find out the neuronal development in premature infants. They showed that wave V absolute peak latency was delayed in AGA infants (p 0.042) than SGA . IPL I – V of symmetric SGA was shortened more than that of AGA ($p < 0.05$) when compared with appropriate for gestational age & symmetric small for gestational age infants. They also observed prolongation of IPL III – V in asymmetric SGA when compared to symmetric SGA (p value 0.047).[63]

S.Allen counter et at studied BAEP in lead exposed children in association with haemoglobin level. This study concluded that significant prolongation of wave I, II, III, IV, V absolute peak latencies in children with low haemoglobin level indicating the effects of low haemoglobin levels on sensory neural auditory brainstem system altering the BAER latencies.(64)

Research done by S **Durmaz et al** in 11 Kwashiorkor , 10 marasmus & 10 healthy control children, observed longer mean latency of wave V on Right ear & prolonged IPL III – V in marasmic group when compared to control group on left ear.(65)

Viresh et al assessed the effect of intrauterine growth retardation on neurosensory development. They found prolongation of wave V latency & central conduction time (I – V interval) in term , healthy small for gestational age newborn , born to undernourished mothers with p value 0.051 & 0.088 respectively compared to term appropriate for gestational age born to healthy mothers.(66)

Dursun odabas et al found the significant difference in III – V & I – V IPL on Right ear in 31 malnourished children of moderate / severe degree when they are compared with 25 healthy children .They also showed that absolute peak latency of wave I on left ear & IPL of III – V on Right ear were longer in children with protein energy malnutrition & Iron deficiency anemia.(67)

Cecila Algrain et al found the longlasting effect of iron deficiency anemia on auditory & visual system functioning in infants with prolonged absolute

latencies of all waves of BAEP & interpeak latencies (except I – III interval) supports the hypothesis that Iron deficiency anemia in infants alters myelination & provide evidence that effects on conduction through the auditory & visual systems can be longlasting.[68]

NERVE CONDUCTION STUDY IN MALNUTRITION :

Shanthi ghosh et al demonstrated nerve conduction study in 67 malnourished children of 6mon – 4 years old & concluded that nerve conduction velocity is reduced in grade III & IV malnutrition when compared to controls ($p < 0.001$). They also demonstrated reduced nerve conduction velocity in ongoing long term malnutrition when compared to acute malnutrition ($p < 0.001$). Nerve conduction velocities were lower in children with the age of onset of malnutrition < 12 months ($p < 0.001$) compared to those with onset after 12months of age.[16]

Thakur D et al studied the influence of height on nerve conduction study & showed a positive correlation between height of an individual & the CMAP amplitudes in median , ulnar & tibial nerves. [69]

Vineetachandha , S.S.Shivalkar ,Minal J.Kusalkar , S.D.Kaundinya tested the effect of BMI on nerve conduction study in median nerve and found that motor & sensory nerve conduction amplitudes correlate significantly ($p \leq 0.005$)[70]

Kumar et al evaluated the nerve conduction velocity in 93 children (38 marasmus , 13 kwashiorkor , 42 control) & showed delay in nerve conduction velocity in children of both marasmic & kwashiorkor group. [71]

G.M.Taori & Sheila pereia showed reduction in amplitude of evoked response from sural nerve in children who previously had kwashiorkor despite normal conduction velocity.[36]

Nimet kabakus et al did peripheral nerve conduction study in children with iron deficiency anemia. 18 children with iron deficiency anemia (10 boys , 8 girls) were tested against 12 healthy children (6 boys ,6 girls). Median motor & sensory nerve conduction velocity were significantly lower than control group with p value < 0.05 & < 0.01 respectively.[72]

Fusan Mayda et al measured nerve conduction velocity in patients with Vitamin B12 deficiency. 27 patients (22 women , 5 men) with vitamin B12 deficiency were compared with 21 healthy individuals. They found that there is reduction in nerve conduction velocity in patients with vitamin B12 deficiency. They suggested that Vitamin B12 is a cofactor in several enzymatic reactions. Thus deficiency may lead to pathologies of posterior & lateral column of spinal cord causing neuropathy & myelopathy. [73]

Ajay kumar, AshwaniDhavan compared motor and sensory Nerve conduction in upper limb of diabetics and nondiabetics. They observed reduced nerve conduction velocity among cases. The probable cause they suggested was median nerve involvement in Diabetic neuropathy , a common complication of diabetes which is demyelinating in nature.[74]

Jagjit S.Chopra found reduction of motor nerve conduction velocity and abnormalities in sensory conduction in mild to moderate and severe protein calorie malnourished children due to retarded myelination.

He also found the normal developmental change in myelinated fibres with increasing proportion of medium to large size fibres and transition from unimodal to bimodal distribution with appropriate relationship of internodal length to fibre diameter in children with mild to moderate PEM.

In severe PEM , myelinated fibre size distribution was impaired with persistent small myelinated fibers with failure of internodal segments on large fibres to elongate with increase in age and presence of segmental demyelination in 50 % of cases.[75]

MATERIALS

&

METHODS

MATERIALS AND METHODS

This study was conducted in Research Laboratory , Department of Physiology, Thanjavur Medical College & Hospital, Thanjavur. This Case control study period extended between August 2013 to July 2014. The children were recruited from Raja Mirasudar Hospital, Thanjavur.

STUDY GROUP :

It consists of 40 malnourished children. Out of 40 , 20 children had Grade III malnutrition according to IAP & WHO Classification for Malnutrition. They had Weight for age , 51 - 60% of expected weight & Height for age < 85% of expected height .

Remaining 20 children had Grade I & II malnutrition as they had weight of 61 – 80 % of expected weight with normal height appropriate for their age.

Table : 1

Cases total – 40 children (23 males , 17 females)

Group	Mean Height for age (cm)	Mean weight for age (kg)	Mean age group
Grade I & II	127.05 ± 9.67076	21.51 ± 3.75708	8.45 ± 1.820208
Grade III	103.4 ± 7.56307	15.325 ± 2.95704	

Control group

It consists of 40 normal children (20 males , 20 females) with weight > 80 % of expected with normal height for their age. Mean height value is 124.1 ± 1.58252 , Mean weight 26.755 ± 4.50538 with mean age group of 7.575 ± 1.73777.

Inclusion criteria :

Malnourished children with the age group of 5 – 10 years according to IAP classification & WHO Classification for malnutrition were included in this study.

Exclusion criteria :

- Genetic causes
- Endocrine causes for short stature
- Children with ear pathology were excluded from the study

Ethical committee approval was obtained from Institution before commensing the study. The aim , nature of the study was explained to the subjects & Parents / Guardians. An informed written consent was obtained from the Parent / Guardian of the child prior to the test.

A detailed clinical , Anthropometry, and Neurological examination was done. Careful history regarding the child was taken . Parents / Guardians was asked about :

- Natal history
- Birth history

- Neonatal history
- Developmental history
- Immunisation history
- Mother was asked about Weight gain, Infections during Antenatal period .

General examination :

- Built
- Nourishment
- Anaemia
- Clubbing
- Cyanosis
- Pedal edema
- Lymphadenopathy

Anthropometry :

- Weight
- Height

Electrophysiological Assessment :

Following electrophysiological parameters were measured in cases & in controls

- BAEP (Brainstem Auditory Evoked Potential)
- NCS (Nerve conduction study)

Using Eight channel digital polygraph.

Brainstem auditory evoked potential :

Pretest instructions:

- The subject , Parents / Guardians were explained about the aim & procedure of the test.
- Complete examination of external ear was done.
- Hearing tests like Rinne's & Weber's test were performed.
- Subject should be completely relaxed.
- Hypnotics may be used to achieve complete relaxation.
- The room should be quiet and comfortable.
- The skin over the scalp and mastoid should be grease free.
- The subject is asked to avoid applying hair oil after the last head bath.

Procedure :

- Skin preparation is done by gently abrading and degreasing before applying electrodes
- Electrodes are placed according to 10-20 International system of EEG electrode placement.

  Channel 1 : Ai-Cz (Active recording electrodes over ipsilateral ear)

  Channel 2 : Ac-Cz (Contralateral ear)

  Ground electrode is placed 20% from the nasion (Fz)

Headphones are placed over the ear which delivers auditory stimulus at a rate of 8 – 10/sec. Intensity of stimulus is set at 60 db. An average of about 100 were recorded using auditory click stimulus.

Recording of BAEP :

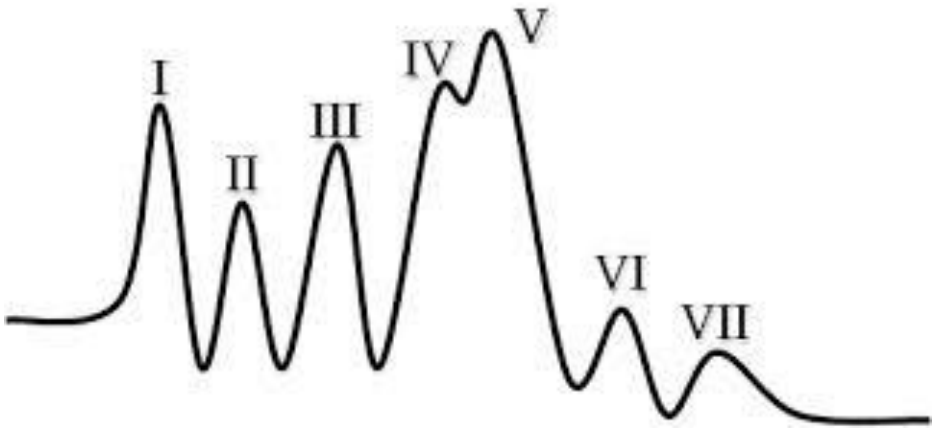
(Instrument setting for BAEP)

Settings	BAEP
Sweep	5msec
Sensitivity	10μv
Low cut	100Hz
High cut	10kHz
Pulse	11/sec
Pulse width	0.1msec
Notch	On
Decibel	30db
Recordings	Average of 100 was recorded using click as stimulus.

Figure : 4

Recording of BAER :

BAER – Waveforms



NERVE CONDUCTION STUDY :

Instructions to the subject :

- Subject & Guardian / parents were explained about the procedure
- Complete clinical examination was done including neurological examination to find out neurological deficits.
- Subject should be completely relaxed.
- Allow the subject to sit comfortably on a chair / supine position in the couch.
- Mild hypnotic may be used to ensure complete relaxation.
- The room should be quite and comfortable.
- The subject should be grounded properly.

PROCEDURE – MNCS (Median nerve) :

- Clean the area & the skin overlying the nerve with spirit at proximal (wrist) & distal (elbow) ends.
- The electrodes are placed as

  Active electrode over muscle belly (Abductor pollicis brevis)

  Reference electrode - 3cm distal to active electrode at 1st metacarpophalangeal joint

  Ground electrode - Between stimulating & recording electrodes

- With the help of stimulating electrodes, Median nerve is stimulated at wrist (3cm proximal to the distal wrist crease) . The latent period is measured as the interval between the stimulus artefact & first negative deflection.
- Then the nerve is stimulated at elbow (near the volar crease of brachial pulse).
- Action potential is observed and recorded. Latency period is noted.
- Distance between the wrist & the elbow points of stimulation is measured in mm & calculate the nerve conduction velocity (m / sec) by the formula

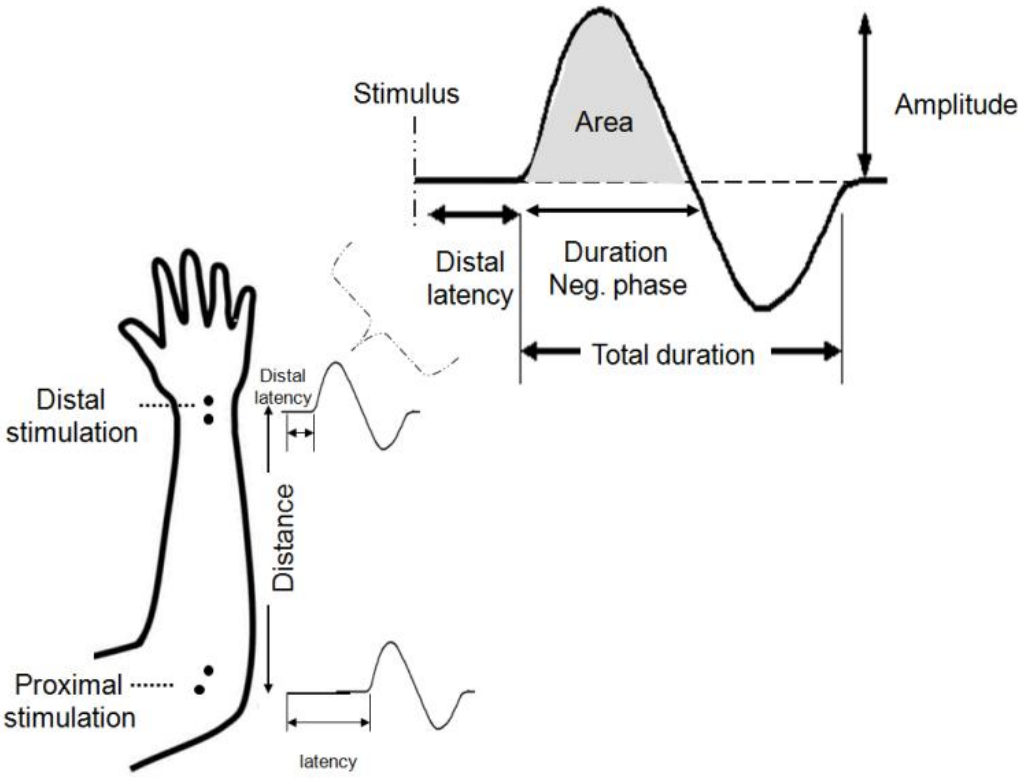
Conduction velocity = Distance (mm) / Latency difference.

Instrument setting for MNCS

Settings	MNCS
Sweep	5 msec
Sensitivity	3 Mv
Low cut	2 Hz
High cut	3 kHz
Pulse	1 / sec
Pulse width	0.1 msec
Notch	Off

Figure : 5

Method of recording of MNCV :



PROCEDURE – SNCS (Median nerve) :

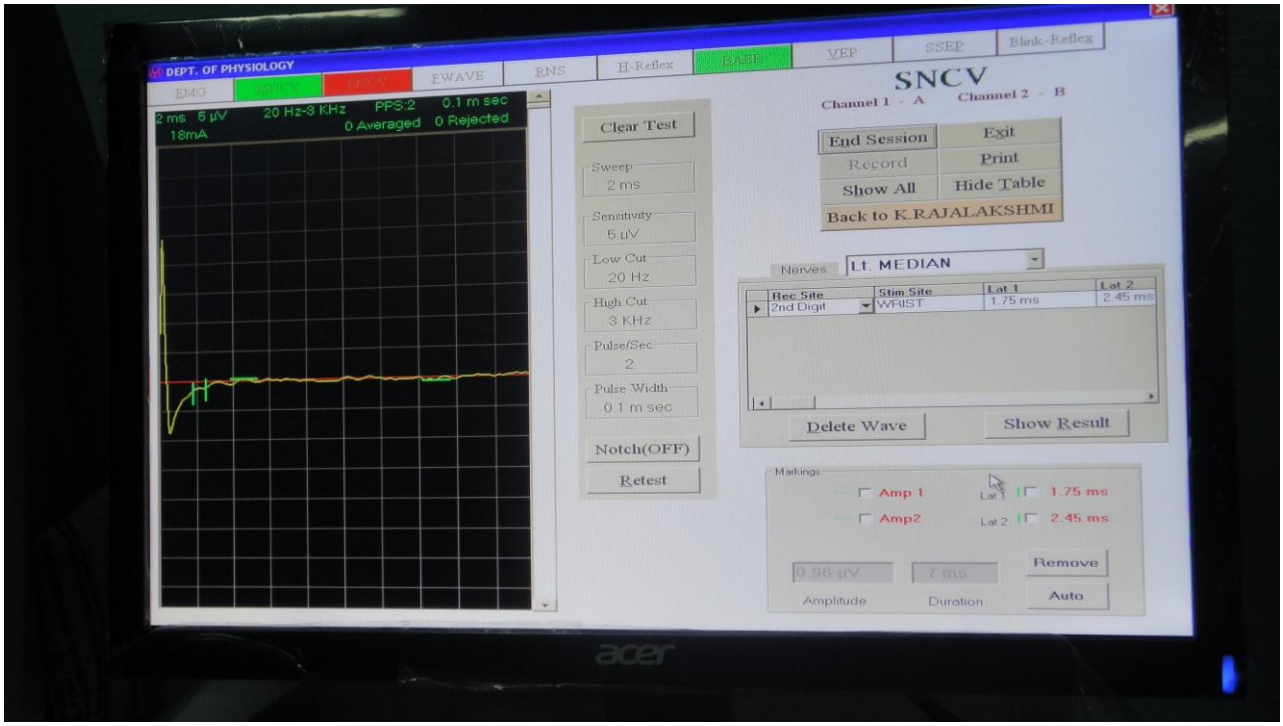
- Ring electrodes are placed at the index finger with the help of conducting gel.
- Ground electrode was placed over the forearm.
- Median nerve is stimulated at wrist. Sensory nerve action potential was recorded using antidromic conduction.
- Latency was measured from the stimulus artifact to the first negative deflection from the baseline. The distance between the recording electrode & the stimulation site was measured with inch tape.
- Sensory nerve conduction velocity is measured by dividing the distance by onset on latency.

Instrument setting for SNCS

Settings	SNCS
Sweep	2 msec
Sensitivity	5µV
Low cut	20 Hz
High cut	3 kHz
Pulse	2 / sec
Pulse width	0.1 msec
Notch	Off

Figure 6

Method of recording SNCV



RESULTS

RESULTS

The study group comprises of 40 children (10 males & 10 females with Grade I & II malnutrition with mean height 127.05 ± 9.67076 , mean weight 21.51 ± 3.75708 & 13 males , 7 females with grade III malnutrition with mean height 103.4 ± 7.56307 , mean weight 15.325 ± 2.95704) of mean age group 8.45 ± 1.820208 selected by IAP Classification for malnutrition.

The control group consists of 40 children (20 males & 20 females) with mean height 124.1 ± 1.58252 ,mean weight 26.755 ± 4.50538 of mean age group 7.575 ± 1.73777

The control & case group differs significantly in following electrophysiological parameters like MNCV ,SNCV, BAEP (Absolute peak latency of wave I, II, III, IV & Interpeak latency I – III & III – V). Group I,II & Group III were also compared. The results were analysed using Student 't' test with $p < 0.05$ was considered statistically significant.

Electrophysiological results in Grade III Malnutrition : (Table 5)

MNCV :

Mean value of MNCV was found to be 33.8110 ± 17.9319 and in the control group it was 46.5815 ± 17.03021 with p value 0.009 found to be statistically significant.

SNCV :

Mean value of SNCV was found to be 30.7570 ± 9.31557 and in the control group it was 46.7878 ± 12.08679 with p value 0.000 found to be statistically significant.

BAEP :

Wave I :

Mean value of wave I latency was found to be 1.4880 ± .34163 and in the control group it was 1.0902 ± 0.26106 with p value 0.000 found to be statistically significant.

Wave II :

Mean value of wave II latency was found to be 2.7735 ± 0.46894 and in the control group it was 2.2912 ± 0.48205 , found to be statistically significant with p value 0.001.

Wave III :

Mean value of wave III latency was found to be 4.1200 ± .53329 and in the control group it was 3.6150 ± 0.74014 with p value 0.009 difference was found to be statistically significant.

Wave IV :

Mean value of wave IV latency was 5.3500 ± 0.62942 in study group. In control group mean value was 4.7995 ± 0.84053 with p value of 0.012 found to be statistically significant.

IPL I –III :

Mean value of IPL I – III was 2.6996 ± 0.47295 and in the control group it was 2.4052 ± 0.48180 , the difference was found to be statistically Significant with p value of 0.029.

IPL III – V :

Mean value of IPL III – V was 2.5085 ± 0.56405 and in the control group it Was 2.2145 ± 0.47196 and the p value was 0.037 found to be statistically significant.

Electrophysiological finding in grade I & II malnutrition : (Table 6)

MNCV :

Mean value of MNCV was found to be 40.6625 ± 19.99633 and in the control group it was 46.581 ± 17.03021 with p value 0.236>0.05 found to be statistically not significant.

SNCV :

Mean value of SNCV was found to be 38.9675 ± 17.49511 and in the control group it was 46.7878 ± 12.08679 with p value 0.047 < 0.05 found to be statistically significant.

BAEP :

Wave I :

Mean value of wave I latency was found to be 1.1895 ± 0.30770 and in the control group it was 1.0902 ± .26106 with p value 0.196 > 0.05 found to be statistically not significant.

Wave II :

Mean value of wave II latency was found to be 2.2250 ± 0.3292 and in the control group it was 2.2912 ± 0.48205 with p value 0.583 > 0.05 found to be statistically not significant.

Wave III :

Mean value of wave III latency was found to be 3.2570 ± 0.49923 and in the control group it was 3.6150 ± 0.74014 with p value 0.056 > 0.05 difference found to be statistically not significant.

Wave IV :

Mean value of wave IV latency was 4.4745 ± 0.60298 in study group. In control group mean value was 4.7995 ± 0.84053 with p value of 0.129 > 0.05 found to be statistically not significant.

IPL I –III :

Mean value of IPL I – III was 2.0990 ± 0.44360 and in the control group it was 2.4052 ± 0.48180 difference with p value of 0.021 < 0.05 found to be statistically Significant.

IPL III –V :

Mean value of IPL III – V was 2.3400 ± 0.71968 and in the control group it was 2.2145 ± 0.47196 and the p value 0.421 > 0.05 was found to be statistically not significant.

DESCRIPTIVE STATISTICS

Table : 2

Group	Height (cm)			
	Min.	Max.	Mean ± SD	p value
Grade III (n = 20)	88	113	103.4 ± 7.56307	0.00
Grade I & II (n = 20)	108	137	127.05 ± 9.67076	0.29
Control (n = 40)	107	140	124.1 ± 10.58252	

Mean height for age in grade III group was 103.4 ± 7.56307 .mean value in control group was 124.1 ± 10.58252. The difference was found to be statistically significant with p value 0.00.

Mean height for age in grade I & II group was 127.05 ± 9.67076 .mean value in control group was 124.1 ± 10.58252 . The difference was found to be statistically not significant with p value 0.29.

Table : 3

Group	Weight (kg)			
	Min.	Max.	Mean ± SD	p value
Grade III (n = 20)	10.5	19.5	15.325 ± 2.95704	0.00
Grade I & II (n = 20)	15.5	26	21.51 ± 3.75708	0.00
Control (n = 40)	20	36	26.755 ± 4.50538	

Mean value for weight for age in grade III group was 15.325 ± 2.95704 .

Mean value in control group was 26.755 ± 4.50538 .

The difference was found to be statistically significant with p value 0.00 .

Mean weight for age in grade I & II group was 21.51 ± 3.75708 .

Mean value in control group was 26.755 ± 4.50538 .

The difference was found to be statistically significant with p value 0.00

.

Table : 4 Electrophysiological findings in control group (n = 40)

Parameter	Min.	Max.	Mean	Std deviation
MNCV	13.91	78.21	46.5815	17.03021
SNCV	10.17	80.00	46.7878	12.08679
BAEP Wave I	0.60	1.58	1.0903	0.26106
Wave II	1.41	3.33	2.2913	0.48205
Wave III	2.34	5.78	3.6150	0.74014
Wave IV	3.36	6.85	4.7995	0.84053
IPL I –III	1.28	3.71	2.4053	0.48180
IPL III – V	1.11	3.27	2.2145	0.47196

Table : 5

Electrophysiological Parameters in Grade III Children with their statistical significance

Parameter	Min.	Max.	Mean	Std deviation	p value
MNCV	12.50	79.68	33.8110	17.93197	0.009
SNCV	17.84	57.14	30.7570	9.31557	0.000
BAEP					
Wave I	0.82	2.04	1.4880	0.34163	0.000
Wave II	2.00	4.18	2.7735	0.46894	0.001
Wave III	3.36	5.30	4.1200	0.53329	0.009
Wave IV	4.60	6.47	5.3500	0.62942	0.012
IPL I –III	1.92	3.91	2.6996	0.47295	0.029
IPL III – V	1.76	3.86	2.5085	0.56405	0.037

$P < 0.05$ - Statistically significant

Table 6: Showing the findings in Grade I & II group with statistical significance ($p < 0.05$)

Parameter	Min.	Max.	Mean	Std deviation	p value
MNCV	14.28	86.49	40.6625	19.99633	0.236
SNCV	11.58	62.86	38.9675	17.49511	0.047
BAEP					
Wave I	0.76	2.05	1.1895	0.30770	0.196
Wave II	1.51	3.02	2.2250	0.32928	0.583
Wave III	2.15	4.14	3.2570	0.49923	0.056
Wave IV	3.43	5.36	4.4745	0.60298	0.129
IPL I –III	1.24	2.80	2.0990	0.44360	0.021
IPL III – V	1.20	4.38	2.3400	0.71968	0.421

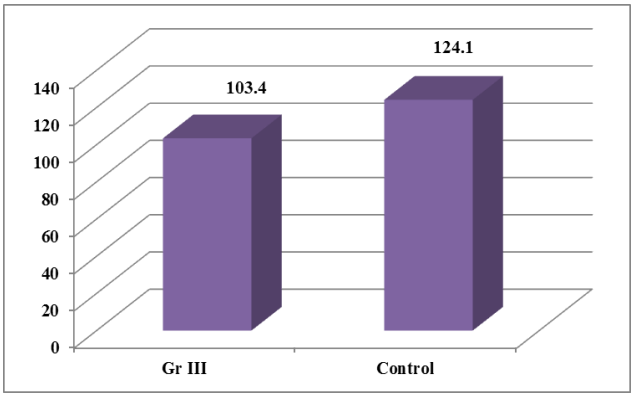
In this group, values of sensory nerve conduction velocity & Inter peak latency I – III shows statistical significance ($p < 0.05$)

Height for age : (Figure : 7)

Mean height for age in grade III group was 103.4 ± 7.56307 . Mean value in control group was 124.1 ± 10.58252 . The difference was found to be statistically significant with p value 0.00.

Figure : 7

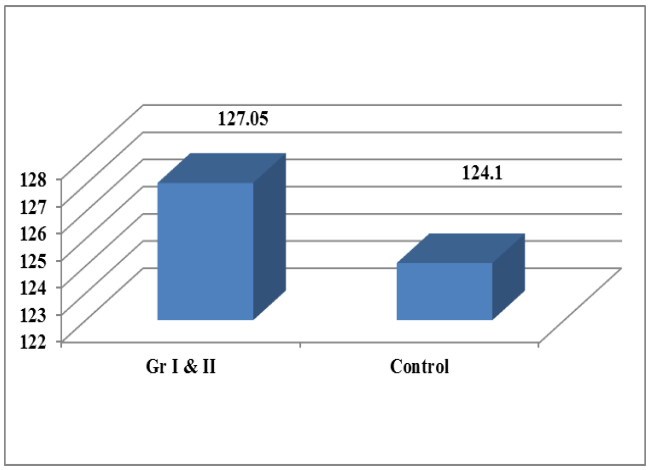
Height for age



Mean height for age in grade I & II group was 127.05 ± 9.67076 .Mean value in control group was 124.1 ± 10.58252 . The difference was found to be statistically not significant with p value 0.29. (Figure : 8)

Figure : 8

Height for age

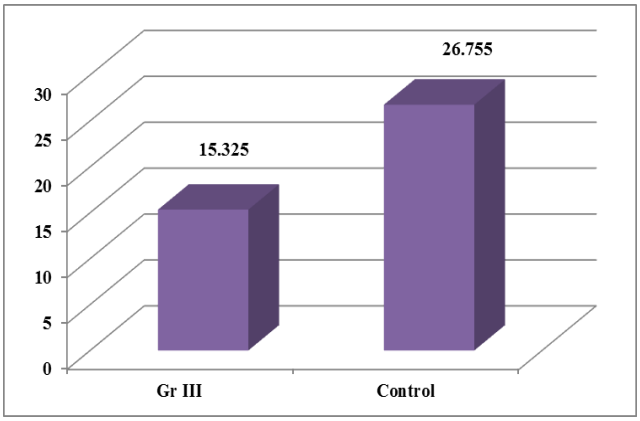


Weight for age : (Figure : 9)

Mean value for weight for age in grade III group was 15.325 ± 2.95704 . Mean value in control group was 26.755 ± 4.50538 . The difference was found to be statistically significant with p value 0.00 .

Figure : 9

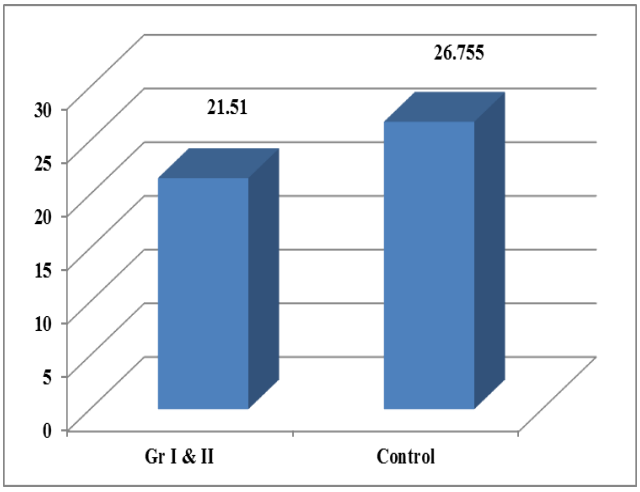
Weight for age



Mean weight for age in grade I & II group was 21.51 ± 3.75708 . Mean value in control group was 26.755 ± 4.50538 . The difference was found to be statistically significant with p value 0.00. (Figure 10)

Figure 10

Weight for age



Findings in Grade III group

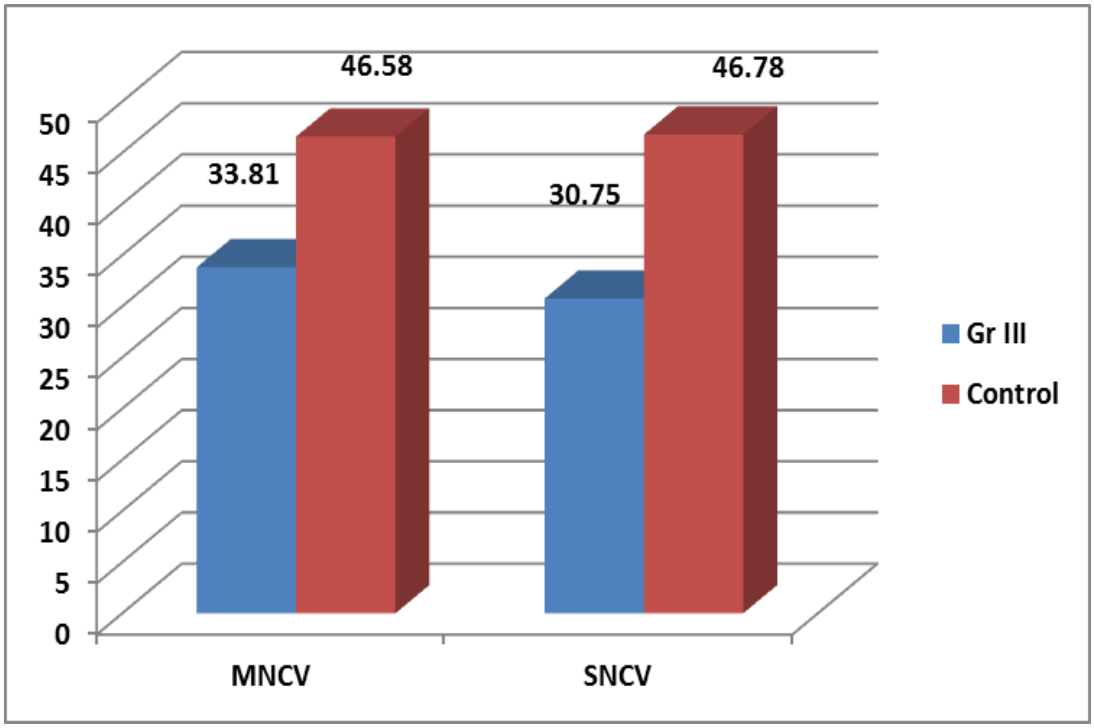
MNCV & SNCV : (Figure 11)

The mean value of MNCV in Grade III group was 33.8110 ± 17.93197 & in control group it is 46.5815 ± 17.03021 with p value of 0.009 found to be statistically significant.

The mean value of SNCV in Grade III group was 30.7570 ± 9.31557

The mean value in control group is 46.7878 ± 12.08679 found to be statistically significant with p value of 0.000

Figure 11



BAEP :

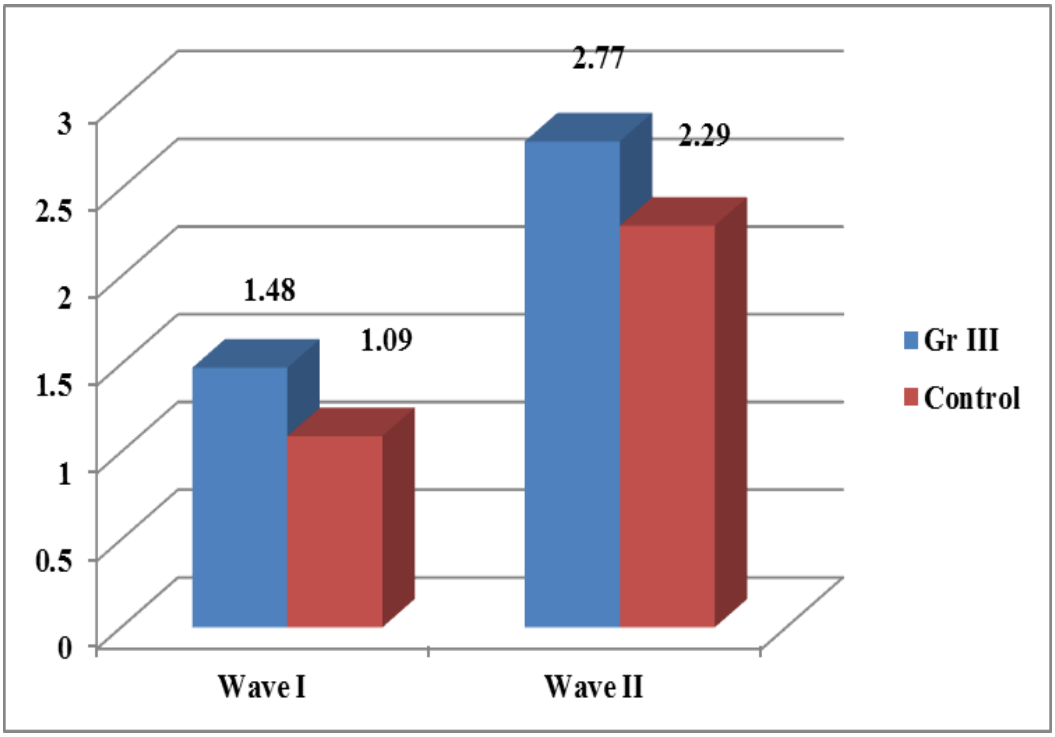
Wave I & II : (Figure 12)

The mean value of wave I latency in Grade III group was 1.488 ± 0.3416

The mean value in control group is 1.09 ± 0.26106 found to be statistically significant with p value of 0.000.

The mean value of wave II in Grade III group was 2.7735 ± 0.4689 Mean value in control group was 2.2912 ± 0.482 with p value of 0.001found to be statistically significant.

Figure 12

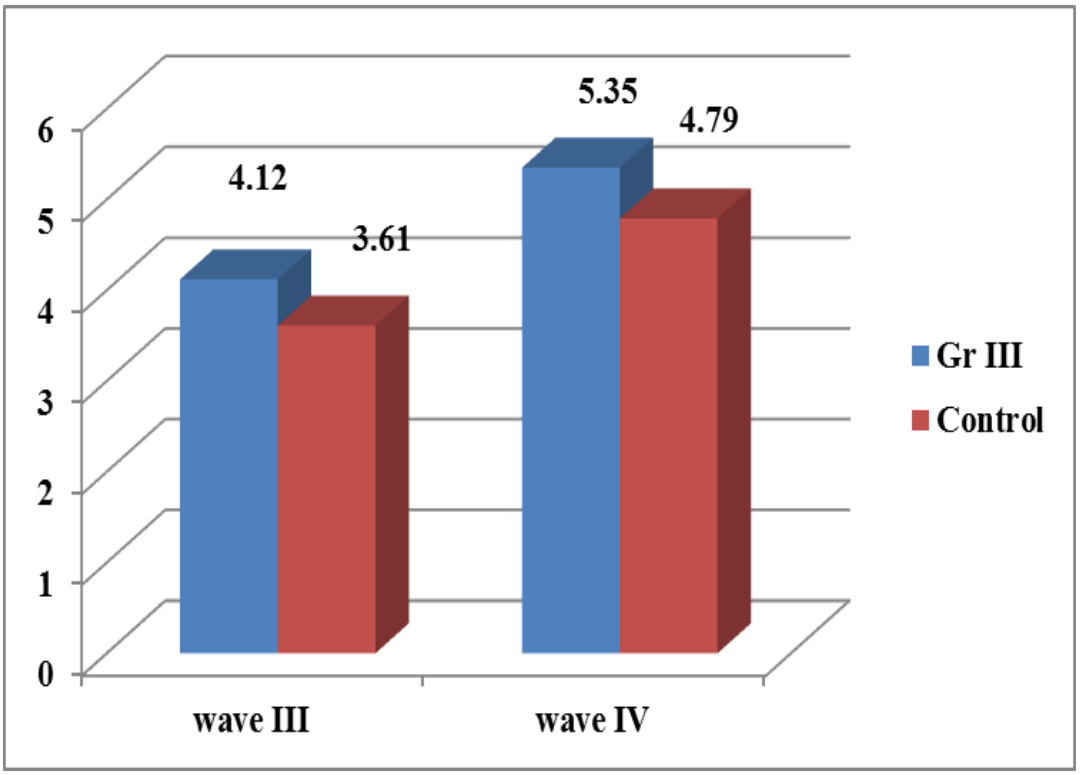


Wave III & IV : (Figure 13)

The mean value of wave III latency in Grade III group was 4.12 ± 0.53329 Mean value in control group is 3.615 ± 0.74014 found to be statistically significant with p value of 0.009.

The mean value of wave IV latency in Grade III group was 5.35 ± 0.62942 Mean value in control group is 4.7995 ± 0.84053 found to be statistically significant with p value of 0.012.

Figure 13



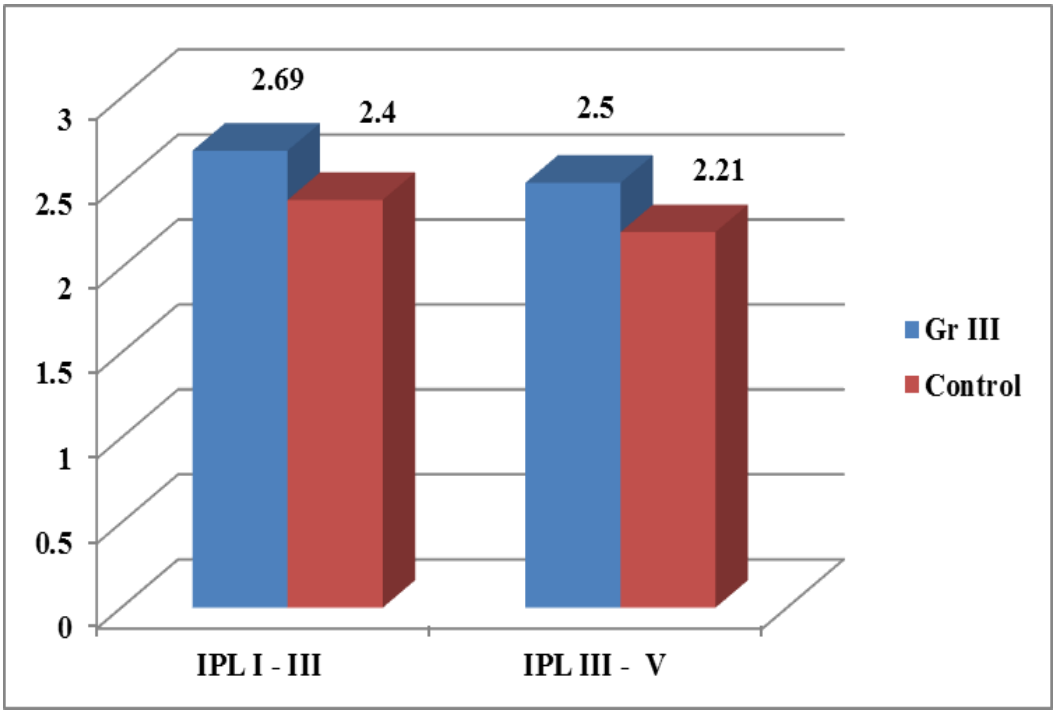
Interpeak latency

IPL I – III & III – V : (Figure 14)

The mean value of IPL I – III in Grade III group was 2.6996 ± 0.47295 Mean value in control group is 2.4052 ± 0.4818 found to be statistically significant with p value of 0.029.

The mean value of IPL III – V in Grade III group was 2.5085 ± 0.56405 Mean value in control group it was 2.2145 ± 0.47196 found to be statistically significant with p value of 0.37.

Figure 14



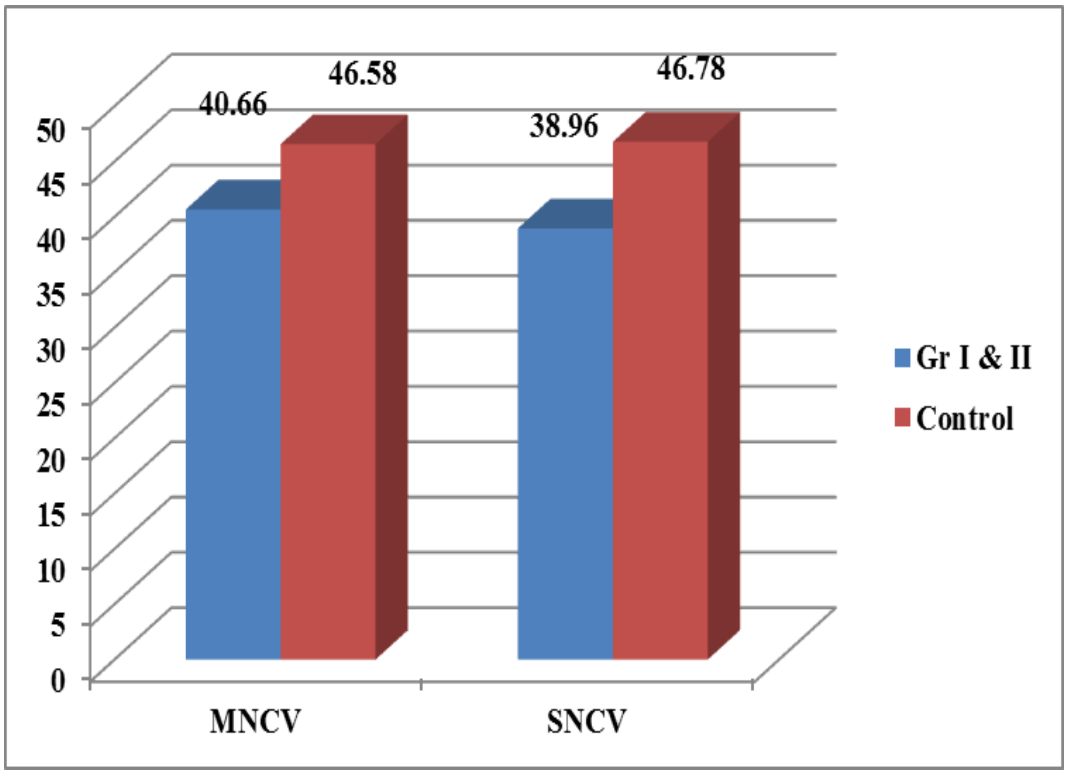
Findings in Grade I & II group

MNCV & SNCV : (Figure 15)

The mean value of MNCV in Grade I & II was 40.6625 ± 19.99633 . Mean value in control group 46.5815 ± 17.03021 found to be statistically not significant with p value 0.236.

The mean value of SNCV in Grade I & II was 38.9675 ± 17.49511 compared with control group 46.7878 ± 12.08679 , the difference was found to be statistically significant with p value of 0.047.

Figure 15



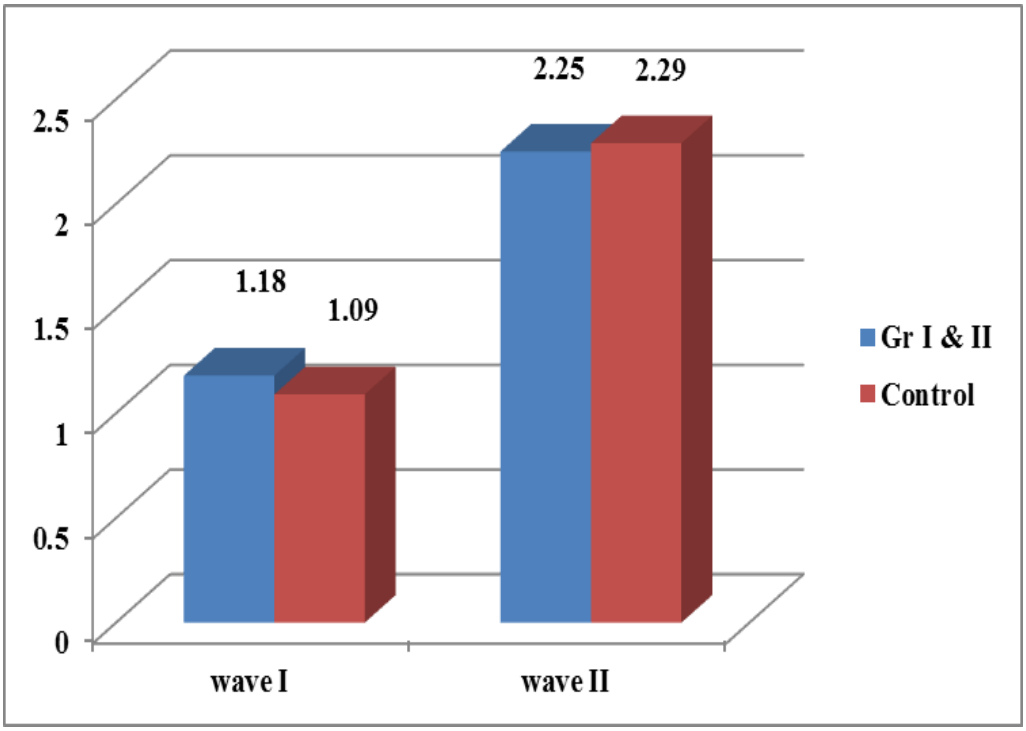
BAEP

Wave I & II : (Figure 16)

The mean value of wave I latency in Grade I & II was 1.1895 ± 0.3077 The mean value in control group is 1.09 ± 0.26106 , the difference was found to be statistically not significant with p value 0.196.

The mean value of wave II latency in Grade I & II group was 2.225 ± 0.32928 Mean value in control group was 2.2912 ± 0.482 found to be statistically not significant with p value 0.583.

Figure 16

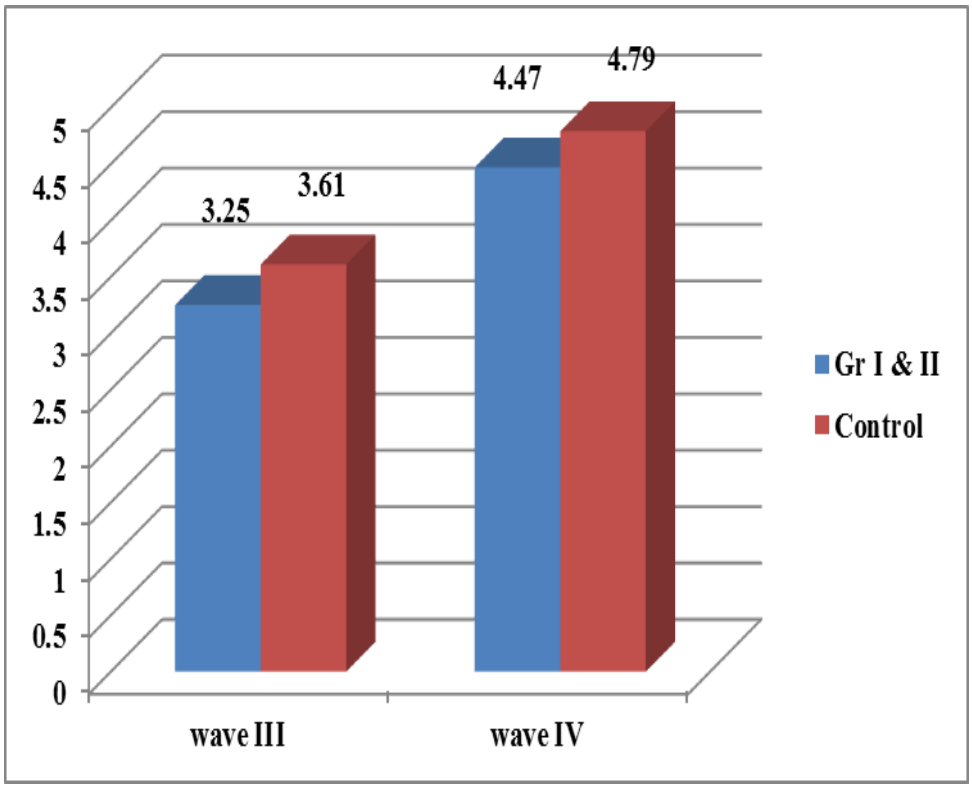


Wave III & IV latency : (Figure 17)

The mean value of wave III in Grade I & II was 3.257 ± 0.49923 Mean value in control group is 3.615 ± 0.74014 found to be statistically not significant with p value 0.056.

The mean value of wave IV in Grade I & II was 4.4745 ± 0.6029 Mean value in control group is 4.7995 ± 0.84053 , found to be statistically not significant with p value 0.129.

Figure 17

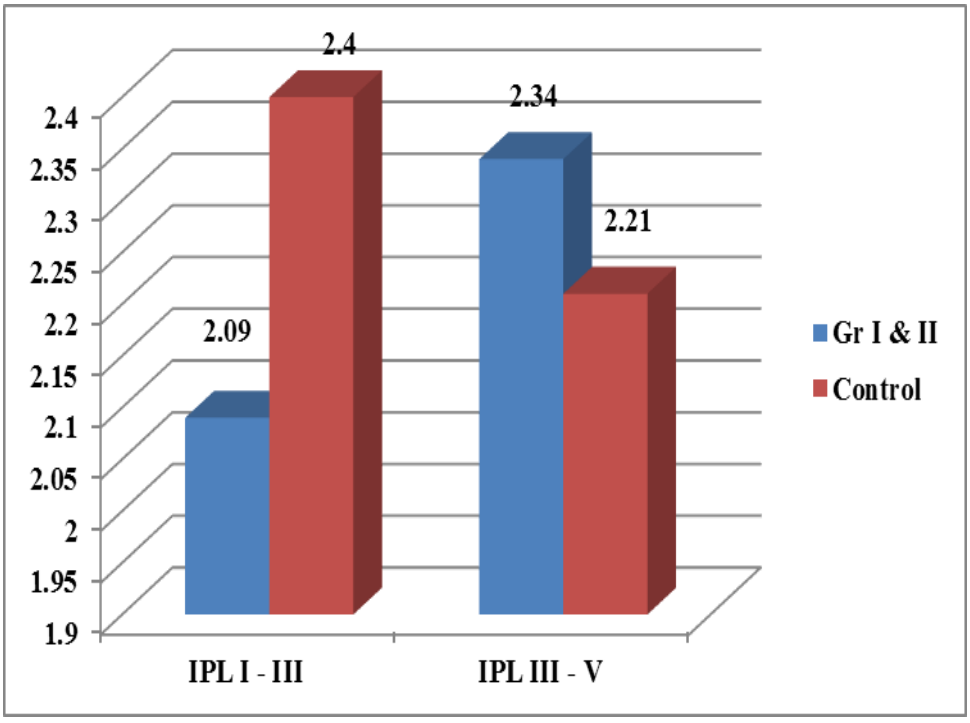


IPL I – III & III – V : (Figure 18)

The mean value of IPL I – III in Grade I & II was 2.099 ± 0.44360 Mean value in control group is 2.4052 ± 0.4818 , found to be statistically significant with p value of 0.021.

The mean value of IPL III – V in Grade I & II was 2.34 ± 0.71968 found to be statistically not significant when compared with control group (mean 2.2145 ± 0.47196) with p value of 0.421.

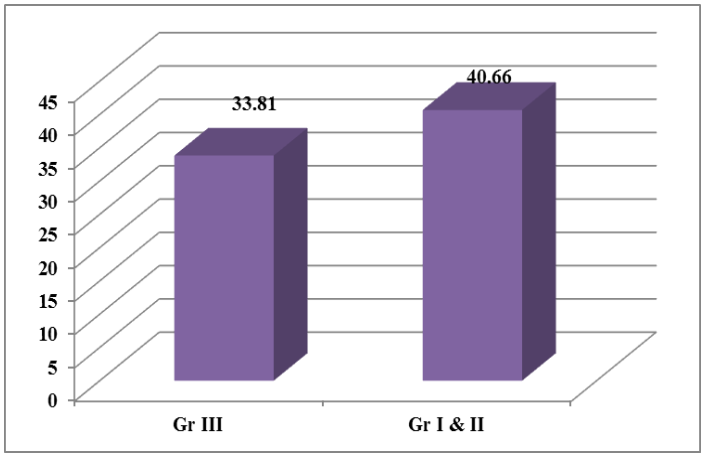
Figure 18



Comparison between Grade III & Grade I & II cases

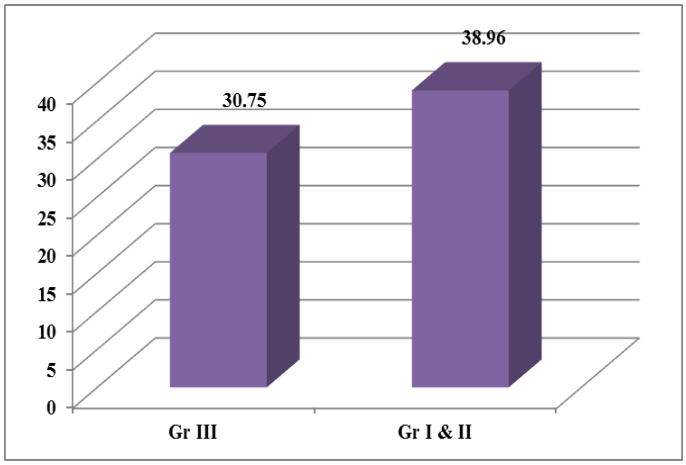
MNCV (Figure 19) : Mean value of MNCV in Grade III group was found to be 33.8110 ± 17.9319 and in the Grade I & II group it was 40.6625 ± 19.99633 with p value 0.261 found to be statistically not significant

Figure 19



SNCV (Figure 20) : Mean value of SNCV in Grade III group was found to be 30.7570 ± 9.31557 and in the Grade I & II group it was 38.9675 ± 17.49511 found to be statistically not significant with p value 0.072

Figure 20

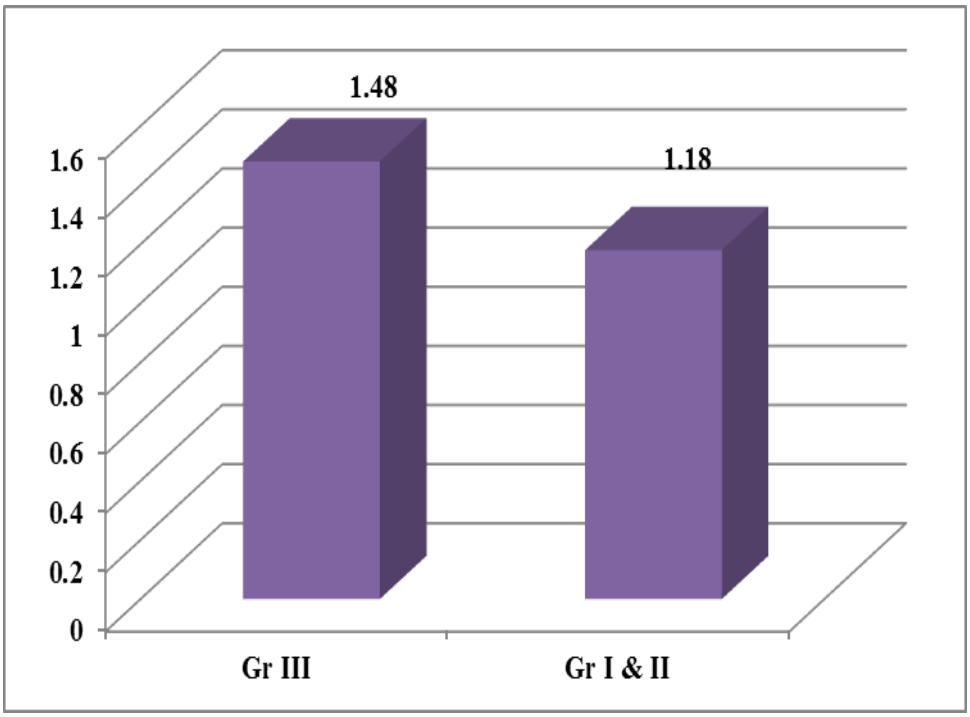


BAEP :

Wave I : Figure 21

Mean value of Wave I latency in Grade III group was found to be 1.4880 ± 0.34163 and in the Grade I & II group it was 1.1895 ± 0.30770 with p value 0.006 found to be statistically significant.

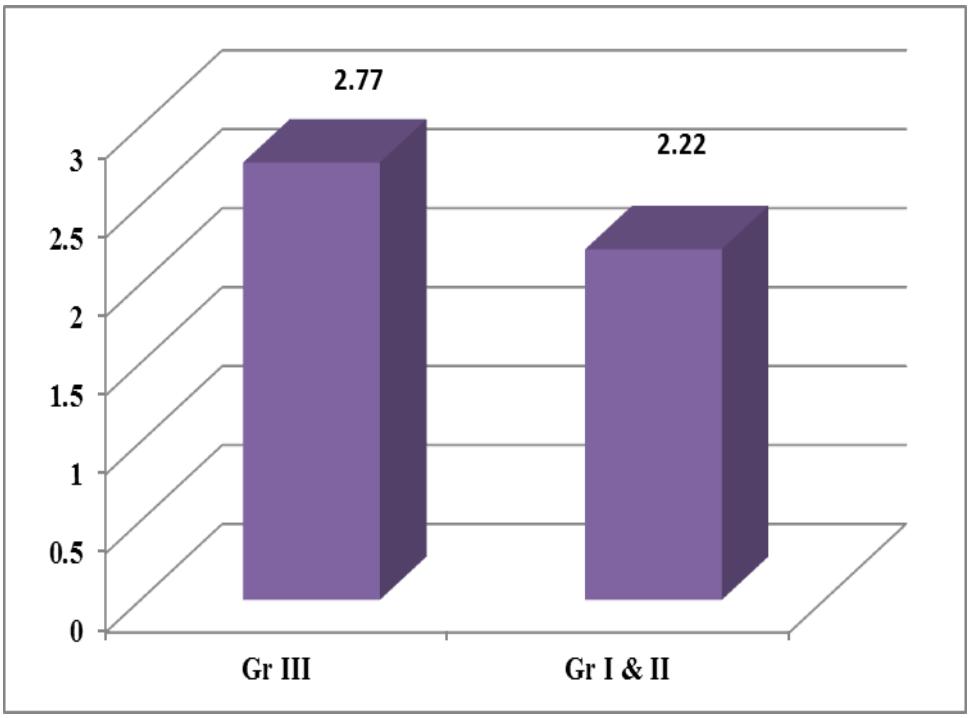
Figure 21



Wave II : (Figure 22)

Mean value of Wave II latency in Grade III group was found to be 2.7735 ± 0.46894 and in the Grade I & II group it was 2.225 ± 0.32928 found to be statistically significant with p value 0.000

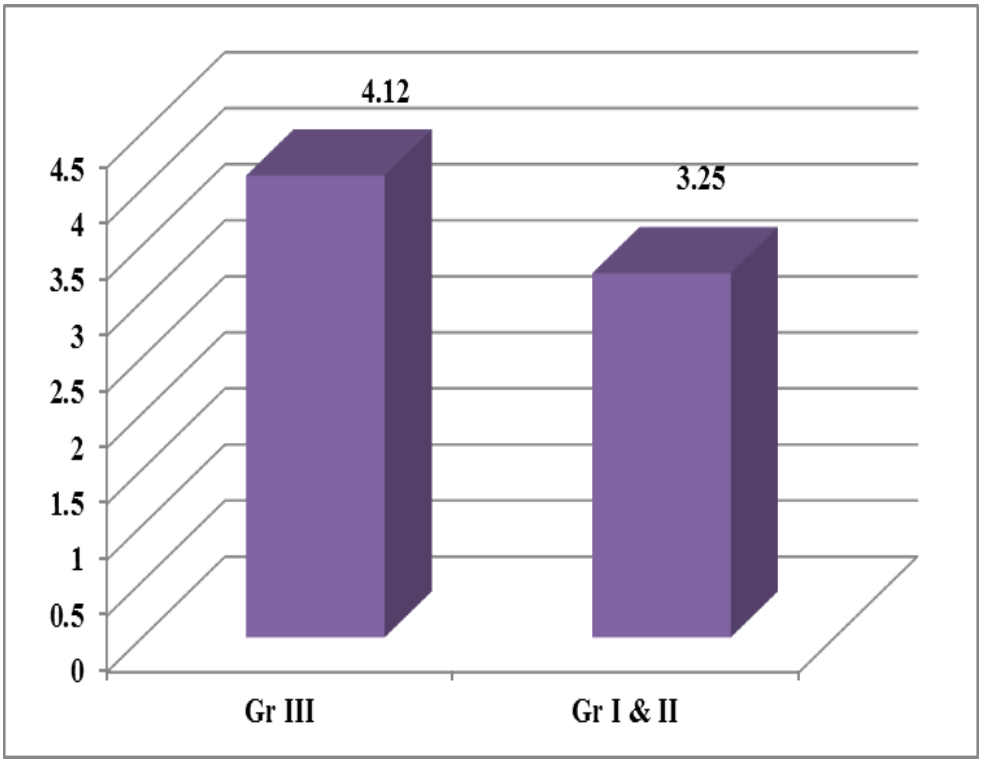
Figure 22



Wave III : (Figure 23)

Mean value of Wave III latency in Grade III group was 4.1200 ± 0.53329 and in the Grade I & II group it was 3.2570 ± 0.49923 with p value 0.000 found to be statistically significant.

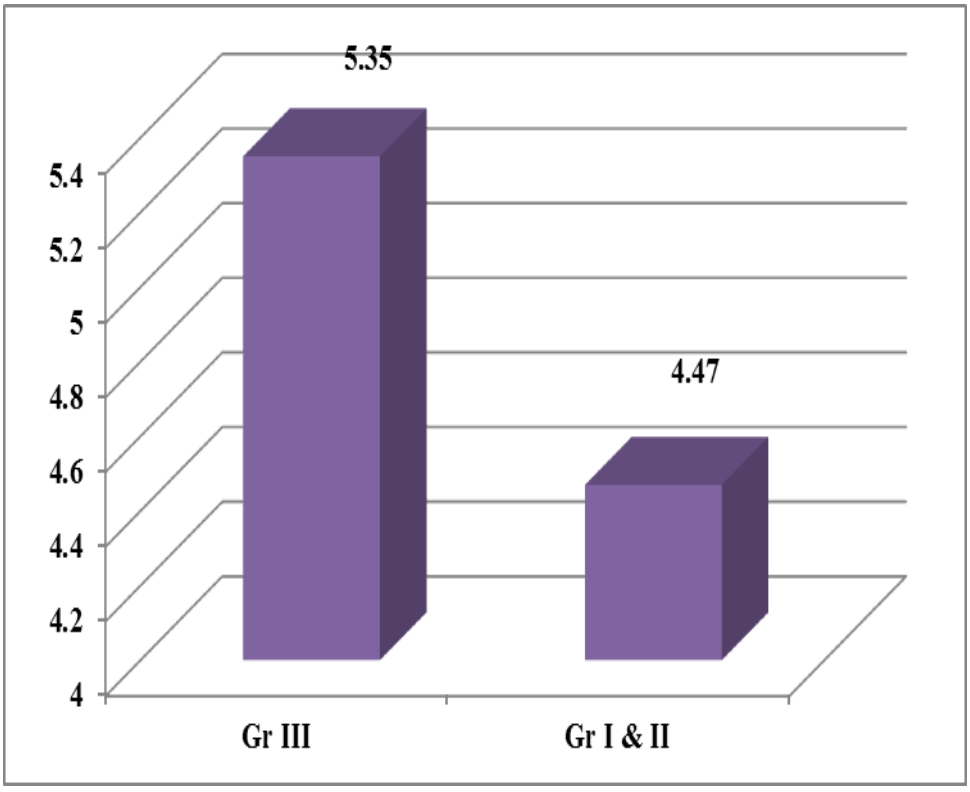
Figure 23



Wave IV : (Figure 24)

Mean value of Wave IV latency in Grade III group was 5.3500 ± 0.62942 and in the Grade I & II group it was 4.4745 ± 0.60298 difference found to be statistically significant with p value 0.000

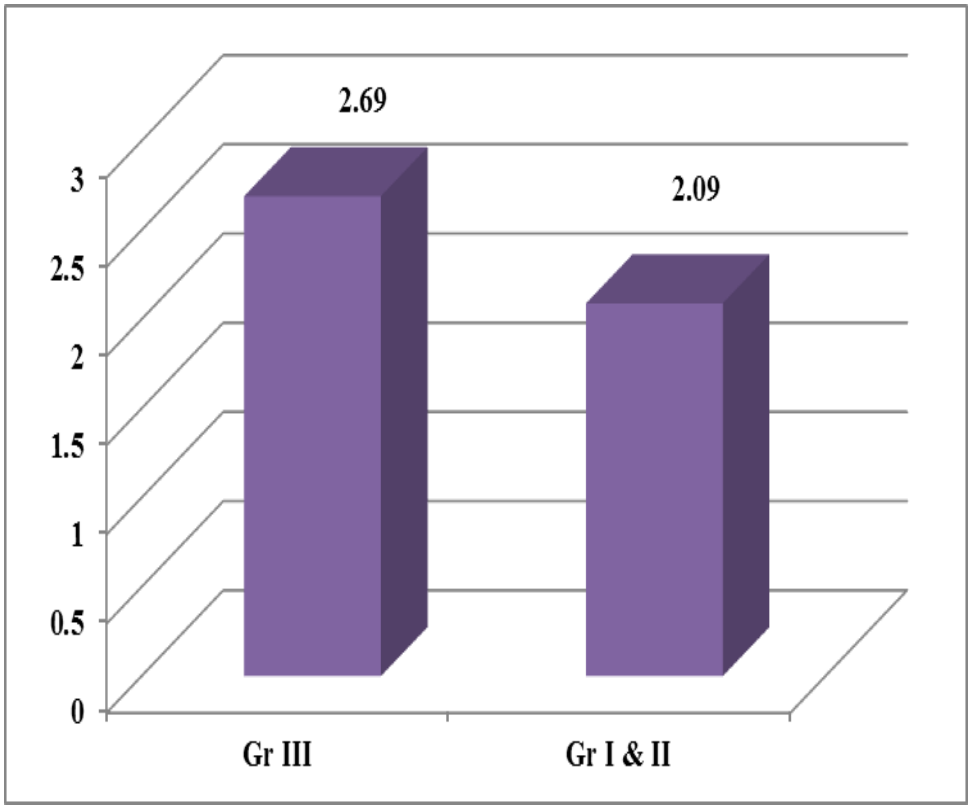
Figure 24



Interpeak Latency I – III : (Figure 25)

Mean value of IPL I - III in Grade III group was found to be 2.6996 ± 0.47295 and in the Grade I & II group it was 2.0990 ± 0.44360 found to be statistically significant with p value 0.000

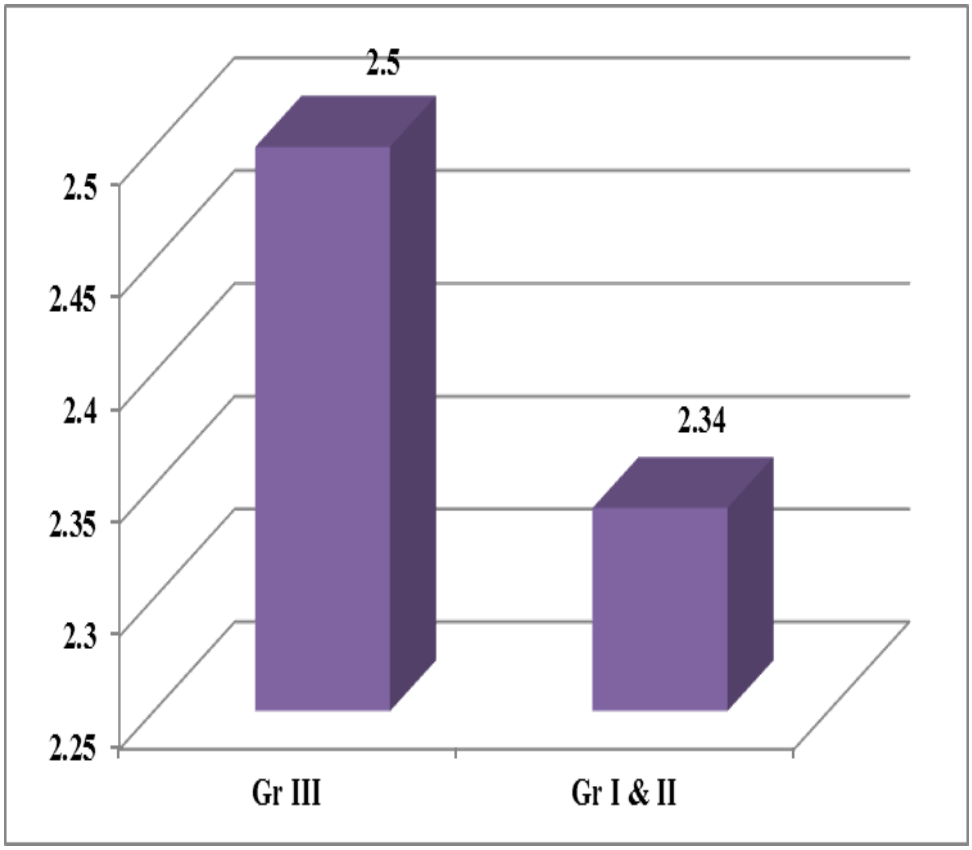
Figure 25



Interpeak Latency III – V : (Figure 26)

Mean value of IPL III - V in Grade III group was found to be 2.5085 ± 0.56405 and in the Grade I & II group it was 2.3400 ± 0.71968 found to be statistically not significant with p value 0.41

Figure 26



DISCUSSION

DISCUSSION

Malnutrition is widely prevalent in all developing countries and children are the worst sufferers. Early development of malnutrition during the critical period of brain development has devasating effect on brain growth. This period extends from prenatal to early postnatal life. Active synthesis of myelin occurs in this period.[44]

Myelin is composed of protein & phospholipid derived from cell membrane of oligodendrocytes in central nervous system and from Schwann cells in peripheral nervous system.[76]

Malnutrition in this period results in physical, chemical, & functional changes in brain. All changes occurring in this period are likely to be irreversible that has a longlasting effect mainly due to delay in myelination. Malnutrition results in poor learning abilities, impaired cognitive functions and school dropouts.

This study has shown significant alteration in the electrophysiological parameters in Brainstem Auditory Evoked Potential Response & Nerve Conduction Velocity in children with malnutrition.

Motor nerve conduction velocity , sensory nerve conduction velocity are significantly reduced in children with Grade III Malnutrition. In BAEP Recordings absolute peak latencies of wave I, II, III, IV & Interpeak latency of I – III, III – V were significantly prolonged $p < 0.05$ when compared to study group suggesting more severe and longer the disease more will be the alteration.

Children with Grade I & II malnutrition had significantly prolonged inter peak latency I –III when compared to study group. Sensory nerve conduction velocity is significantly reduced in this group.

Children with Grade III malnutrition had prolonged wave I, II, III, IV latency & IPL I – III with statistical significance of $p < 0.05$ when compared with Grade I , II children

BAEP findings :

The present study observed prolongation of Wave I, II, III, IV & Prolonged inter peak latenccy I – III , III – V with p value < 0.05 in Grade III group.

In Grade I, II Group prolongation of Interpeak latency I – III was observed. This may be due to imperfect growth and development of myelin producing initial abnormalities in an increase in interpeak latency particularly I – III.[49]

Similar results were observed by **Vandana & O.P Tandon** , **S.Allen counter et at** , **Dursun odabas et al & Cecila Algrain et al .**

Vandana and O.P.Tandon measured Auditory Evoked Potential Responses in 20 chronic malnourished children of 3 – 6 years of age and found that there is significant prolongation in peak latencies of waves I, II, III, IV. Interpeak latencies of I – III & III – V were also prolonged as malnutrition affects the peripheral developmental process of auditory pathways in brainstem. They found that longer duration of continuous protein energy malnutrition slows or arrest the process of myelination & preventing the increase in caliber of myelinated nerve fibre.[62] The results were consistent with the result of present study.

S.Allen counter et at have shown the association of decreased haemoglobin level & anemia with abnormal brainstem auditory evoked responses in children with 2 – 15years of age. Significantly prolonged absolute latencies of waves I, II, III, IV, V were observed when compared to children with normal haemoglobin levels.

The reason suggested was low haemoglobin levels may have subtle effects on the sensory – neural auditory brainstem system that are manifested as altered BAEP latencies. The results of present study is consistent with this result.

Dursun odabas et al studied the auditory brainstem potentials in children with protein energy malnutrition to determine the effects of PEM on developing brain in children. Significant differences were recorded in mean latencies of the waves I, II, III, IV, V on both ears and in the mean interpeak latencies of waves III – V & I – V on right ear between study and control group. They also observed longer latency of Wave I on left ear & mean IPL of III – V on right ear in children with PEM and iron deficiency anemia suggesting the defect in myelination of auditory brainstem pathways in children with moderate / severe PEM. The present study results agreed with this result.

Cecila Algrain et al studied the longlasting effects of iron deficiency anemia on auditory and visual system functioning in infants. Absolute latencies of all auditory brainstem responses waves and interpeak latencies (except I – III interval) were significantly longer in children with iron deficiency anemia. They showed the evidence that iron deficiency anemia in infancy alters myelination and affects the transmission through auditory system. The result of this study is similar with this result.

NERVE CONDUCTION STUDY FINDINGS :

In the present study motor nerve conduction velocity is significantly reduced in children with Grade III malnutrition.

Sensory nerve conduction study is significantly reduced in both Grade I, II & Grade III malnourished group. Because sensory nerve conduction studies are more sensitive than motor nerve conduction study in detecting early or mild demyelinating diseases.[53,57]

Studies done by **Shanthi ghosh et al , Kumar et al** showed similar findings with the present study results.

Shanthi ghosh et al conducted nerve conduction study in 67 children to assess the effect of malnutrition on peripheral nervous system.

- Significant reduction in nerve conduction velocity was observed in children with severe protein energy malnutrition and ongoing longterm malnutrition .
- Results also showed that the age of onset has an impact on central nervous system alteration. When the age of onset of malnutrition is < 12months , nerve conduction velocity was significantly reduced when compared to > 12months of age of onset.
- They found that undernutrition produces permanent molecular errors in brain membrane composition and affects biochemical maturity of brain.
- Since protein is important for lipid incorporation in brain & in nerve , nutritional protein deficiency affects myelination so the nerve conduction velocity does not increase.[16] This result is consistent with the present study result.

Kumar et al measured nerve conduction velocity in children with protein calorie malnutrition. 38 marasmus , 13 kwashiorkor children were studied. They found that conduction velocity were reduced in each type of malnutrition suggested that PEM when it occurs during the development of nervous system affects myelination of peripheral nerves.[42] Result of the present study agreed with this result.

The present study results agreed with results of **Nimet kabakus et al.** They did peripheral nerve conduction study in children with iron deficiency anemia. children with iron deficiency anemia were tested against healthy children . Median motor & sensory nerve conduction velocity were significantly lower than control group .[72]

Fusan Mayda et al measured nerve conduction velocity in patients with Vitamin B12 deficiency. They found that there is reduction in nerve conduction velocity in patients with vitamin B12 deficiency suggested that Vitamin B12 is a cofactor in several enzymatic reactions. Thus deficiency may lead to pathologies of posterior & lateral column of spinal cord causing neuropathy & myelopathy. [73] The results of present study is consistent with this result

The result of present study is similar with research done by **Jagjit S.Chopra.** Reduction in nerve conduction velocity was observed in children with protein calorie malnutrition.[75]

CONCLUSION

CONCLUSION

The present study shows significant alteration in Nerve Conduction Velocity & Brainstem Auditory Evoked Potential Responses in children with malnutrition which may be due to nutritional deficiency affecting myelination of peripheral nerves & Auditory Brainstem Pathways.

Electrophysiological parameters like MNCV , SNCV , BAEP – Absolute peak latency of Wave I, II, III, IV and Interpeak latency I – III , III – V were recorded. Electrophysiological abnormalities depends on duration and severity of malnutrition. Severe and chronic malnutrition affects Nerve Conduction Velocity , BAEP – I, II, III, IV and IPL I – III, III – V findings.

Mild to moderate malnutrition alters Sensory Nerve Conduction Velocity & BAEP – IPL I – III. Thus there is strong association between duration and severity of malnutrition with NCS & BAEP abnormalities. So these electrophysiological tests can be used to detect malnutrition at its early stage.

Nutritional deficiency during development of brain has longlasting effect on learning abilities, psychomotor development , But whether it is reversible or irreversible after nutritional rehabilitation needs to be evaluated.

With the help of advanced electrophysiological methods , the need for screening , early diagnosis , health education & initiating nutritional support at appropriate time will help the Children to improve their academic performance and to become a successful achiever in future.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Swarna Rekha Bhat . Achar's Textbook of paediatrics.4th Edition .Pages 45-53
2. A Parthasarathy .Editor in chief .IAP Textbook of Pediatrics.3rd Edition pg 120-139
3. SenP.Mishra CP.Gupta VM,Singh TB.PEM among rural preschool children of eastern U.P Indian Journal of Maternal & child health 1996 Jul-Sep;7(4):95-8
4. Santhosh kumar A.Sunil kumar D,N.C.Ashok,Ragavendrasamy Koppad. PEM & its Association with immunisation status among 1-5 aged children in southern part of India, Mysore.IJCRR vol05 issue 02
5. Agarwal. K. N.Pediatric Nutrition Handbook – 6th Edition .American Academy of Pediatrics. Pages 18-30
6. Mohammad Ramadan Hassaan.Ashgan Abd Alla Abdel-aal Alghobashy,Hadeel Mohammed Abdel-Rahman.Auditory neural efficiency in protein energy malnourished toddlers with and without iron deficiency anemia. Egyptian journal of Ear.Nose,Throat & allied Sciences2011.12,105-114
7. Aziz Abdul Rehman Jiwani. ISRA Medical journal volume 3 issue 3 Dec 2011
8. WHO.World prevalence of anaemia 1993-2005:WHO Global Database on Anemia
9. T.O.Odebode.S.O.Odebode PEM and the nervous system: Impact of socioeconomic condition, Weaning practice, Infection & Food intake. Pakistan journal of Nutrition 4(5) : 304-309,2005
10. Michel K Georgieff. Nutrition & the developing brain: Nutrient priorities & measurement. The American journal of nutrition 2007; 85:614s-20s
11. Bhoomika R Kar,Shobini L Rao, B A Chandremouli. Cognitive development in children with chronic PEM.Behavioral & Brain function 2008,4:31

12. Neil Mclniosh. Forfar & Arneil's Textbook of paediatrics. Seventh edition pg 523-526

13. David A.Levitsky , Barbara J.Strupp. Malnutrition and the brain :Changing concepts, changing concerns.J.nutr:2212S-2220S,1995

14. El-Khayat H.A,Nassar M.F,El-Khayat .N.M,Gomaa S.M..Peripheral & central nerve conduction changes in PEM. Egyptian J.Neurol Psychia. Neurosurg., 2004, 41(1):359-371

15. Juravi Goncalves de lima,Carolina Araujo Rodrigues Funayama.Effects of malnutrition & sensory motor stimulation on auditory eviked potentials..Psychology & Neuroscience,2008,1,2,121-127

16. Shanthi Ghosh,Kumkumvaid Manmohan,C.Maheshwari. effect of degree & duration of PEM on Peripheran nerves in children.J neurology , Neurosurgery & Psychiatry ,1979,42,760-763.

17. K.Park.Park's Textbook of Preventive& Social Medicine.21st Edition. Pg 13–21.

18. Sundar lal, Adarsh, Pankaj. Textbook of Community Medicine. Page 59 – 64

19. K.N agarwal .Textbook of paediatrics . nutrition .pg50-66

20. Campbell K.2003.Dorsal – ventral patterening in the mammalian telencephalon. Curr.Opin.Neurobiol 113:50-56

21. Briscoe J , Ercson J.2001.Specification of neuronal fates in the ventral neural tube.Curr.Opin.Neurobiol 11 : 43- 49

22. Murase S,Horwitz AF.2004.Directions in cell migration along rostral migratory stream.Curr.Top Dev Biol 61 :135-152

23. Schoenwolf, Bleyl Brauer,Francis-west.Larsen's Human Embryology 4th Edition. Page 247-291

24. Barres BA ,Barde Y.2000. Neuronal & cell biology. Curr. Opin. Neurobiolol: 642-648

25. Nelson . Essentials of paediatrics.Sixth edition.pg111-114

26. Peter S.Spencer.Valerie S.Palmer.Interrelationship of undernutrition & Neurotoxicity. Neurotoxicology .2012 june ;33(3) :605-615

27. O.P.GHAI . Essential paediatrics 6th edition.pg 101-107

28. P,M,Udani. PEM , Brain and various facets of child development.Indian J . paediatrics 1992;59:165-186

29. Afshan Sharghi , Aziz Kamran , Mohammad Faridan Evaluating risk factors for PEM in children under the age of six years: a case-control study from Iran. International Journal of General Medicine,16 August 2011

30. Connor Fuchs.Tania Sultana, Tahmeed Ahmed, M.Iqbal Hossain.Factors associated with PEM admitted with diarrhoea in Bangaladesh. International J . paediatrics vol 2014,1-5

31. Shaheen Banu Shaikh, Ismail Haji, Praveen Doddmani, Sarfaraj Shaikh. Clinico biochemical basis of iron profile in children with PEM.

32. Alesina,A and D.Rodrik (1994).Distributive politics and economic growth.Quarterly journal of Economics 108 : 465-490

33. Kaori Saito,Joshua R.Korzenenik,James F Jekel.A case control study on Maternal knowledge of malnutrition & health seeking attitudes in reral south India.Yale J biology & Medicine 70 (1997),pp149-160

34. Udani P M.Physical growth of children in different socio-economic groups.Indian J.Child health1983 ;12:593-611

35. Ghosh S,Bhargava SK,Madhavan S et al. Intrauterine growth of North Indian Babies. Pediatrics 1971;47:826

36. G.M.Taori ,Sheila M.Peraira. EEG & nerve conduction in survivors of Kwashiorkor.Br.J.Nutr (1974),31,59

37. Cintra,L.Diaz–cintraS.,GalvanA.,Kemper,T(1990)Effectsof protein undernutrition on the dentate gyrus in rats of three age groups.Brain Res.532:271-277

38. Cravioto,J & Robles,B.(1965). American journal of orthopsychiatry.35,449

39. Suraj Gupte, Robinson.L . I J of paediatrics 1975;12 : 100-110

40. Mathur GP,Kushawaha KP,Mathur S. Protein energy malnutrition updated.Special volume Nutrition , 1997;94-122

41. Piyush Gupta.Clinical methods in paediatrics .2nd edition pg60-93

42. Tom Lissauer & Graham Clayden.Illustrated Textbook of paediatrics ,4th edition.

43. Dilek Dilli Zenep Eras, UGar Dilmen,Evrim Durgut Sakrucu.Neuro developmental evaluation og low birth weight infants with sepsis at 18 – 24 months corrected age. Indian J . paediatrics vol 50- Mar 16,2013

44. Stoch, M. B. & Smythe.P.M.(1963).Archs Dis Children.38,546

45. Ronald G.Emerson,Thaddeus S,Walczak,Timothy A.EEg & Evoked potentials.11th edition 2005.pg 79-87

46. UK Mishra, J kalita.Clinical neurophysiology.2nd edition pg 331-345

47. Christopher G.Goetz.Textbook of Clinical neurology 3rd edition.pg 487-607

48. Edward M.Brett. paediatric neurology.Third edition pg754-784

49. Michel J Aminoff. Electrodiagnosis in clinical neurology. Fifth edition.pg 525 - 546

50. Mochizuki Y,Go T,Obkubo H et al :development of human brainstem auditory Evoked potential & gender difference from infants to young adults.Prog Neurobiol,20:273,1983

51. Hecox KE,Galambos R : brainstem auditory Evoked potential in infants & young adults Arch otolaryngol,99 :1974

52. A.K.Jain. Practical manual in physiology.pg 293-300

53. G.K.Pal .Manual of practical plysiology. Pg 298 – 302

54. A Mallik,A I weir. Nerve conduction studies : Essentials & pitfalls in practice. J neurol Neurosurg Psychiatry 2005 ;76S ii23-ii31

55. Randolph W.Evans, Diagnosting testing in Neurology. Pg 283-288

56. Andrew J.Robinson .Clinical Electrophysiology .Lippincott William & Wilkins.3rd edition pg 423-441

57. Jasper R.Daube. Nerve conduction studies.4 th edition pg 285 – 317

58. Hodes R ,Larrabee MG , German W**:** The human EMG in response to nerve stimulation & the conduction velocity of motor axons:Studies on normal & on injured peripheral nerves.Arch Neurol Psychiatry, 60:340, 1948

59. Nora LM: American association of Electrodiagnostic Medicine guidelines: Implanted cardioverters and defibrillators.Muscle Nerve, 19: 1359, 1996

60. O.Kwast rabben .Sensory nerve conduction study in children age related changes in conduction velocities.Neuropediatrics 1995 ; 26(1) :26-32

61. Manuel Roncagliolo,Marcelo Garrido, tomas walter, patricio peirano ,betsy lozoff.Evidence of altered CNS development in infants with iron deficiency anemia at 6mon: delayed maturation of auditory brainstem response.Am J clin.nutr 1998 ; 68: 683-90

62. Vandana & O.P.Tandan . brainstem auditory evoked response in chronic malnourished children. Indian J phusiol Pharmacol 2006 ; 50(1) :48-52

63. il moon,kee soo,gui sang kim, byung min choi, baik line un. auditory brainstem response in premature small for gestational age infants. Korean J pediatr 2006 ; 49: 1308 – 1314.

64. S.Allen counter , Leo H Buchanan . Association of haemoglobin levels & brainstem auditory evoked response in lead exposed children Clin.Biochem 2012 oct ; 45(15) :1197 – 1201

65. S Durmas , U karagol , g deda . brainstem auditory Evoked Potential response in children with PEM.International paediatrics 12/1999 ;41(6):615-9

66. Viresh Mahajan, Piyush gupta . brainstem auditory Evoked Potential response in children of undernourished mothers.

67. Dursan odabas , Mesud ekisli brainstem auditory Evoked potential responses in children with PEM. International J of otorhinolaryngology.07/2005; 69(7) 923-8

68. Cecilla algrain,Patricio peirano,marcelo Garrido. Iron deficiency anemia in infancy longlasting effects on auditory & Visual system functioning. J . paediatrics R J . paediatrics Res 53 :217-223,2003

69. Thakur D , JHA .S, Pandey. Influence of height on nerve conduction study parameters on peripheral nerves.J of clinical Diagnosti Research 2011 Apr,Vol-m5 (2) :260-263

70. Vineetachandha , S.S.Shivalkar ,Minal J.Kusalkar , S.D.Kaundinya . Effect of BMI on nerve conduction study. International conference of Basic & Applied physiology.

71. Kumar , Ghai O,P.,Singh N. Delayed nerve conduction velocity in children with PEM. J paediatrics ,1977 Jan ;90(1) :149 – 53

72. Nimet Kabakus ,Ahmet Ayar Tahir Kurtulus Yoldas . Reversal of Iron Deficiency Anemia-induced Peripheral Neuropathy by Iron Treatment in Children with Iron Deficiency Anemia . Journal of tropical paediatrics, Volume 48, Issue 4 Pp. 204-209.

73. Turkish Journal on Neurological sciences . 2014, Volume 31, No .1, Pg 1-10

74. Ajay kumar, AshwaniDhavan .Comparison of nerve conduction velocity in Diabetes International conference of Basic & Applied physiology

75. Jagjit S.Chopra . Effect of protein calorie malnutrition on peripheral nerves, A clinical, Electrophysiological & Histopathological study. International J paediatrics 41(6):615-9

ANNEXURES

ABBREVATIONS

- **PEM** : Protein Energy Malnutrition
- **BAER :** Brainstem Auditory Evoked Response
- **MNCV :** Motor Nerve Conduction Velocity
- **SNCV :** Sensory Nerve Conduction Velocity
- **IPL :** Inter peak latency
- **MNCS :** Motor nerve conduction study
- **SNCS :** Sensory nerve conduction study

INFORMED CONSENT

Dr.S.Rubha, Postgraduate student in Department of Physiology , Thanjavur Medical College, Thanjavur is doing the study entitled " **Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction and Auditory Evoked Potential Responses in Children**" **.**

The procedure of this study is clearly explained to me in my own language. I understand that, this is an entirely a non – invasive procedure. I hereby give my consent for the participation of my Son / Daughter in this study. The data obtained may be used for research and publication.

Station : Signature of the Parent /Guardian

Date :

Signature of the Investigator

PROFORMA

TOPIC : EFFECTS OF PROTEIN ENERGY MALNUTRITION ON PERIPHERAL NERVE CONDUCTION & AUDITORY EVOKED POTENTIAL RESPONSES IN CHILDREN.

DATE :

NAME : **AGE :** **SEX:**

INFORMANT : **RELIABILITY :**

ADDRESS :

PRESENT HISTORY :

PAST HISTORY :

FAMILY H/O :

NATAL HISTORY :

NEONATAL HISTORY :

DEVELOPMENTAL HISTORY :

IMMUNISATION HISTORY :

SOCIOECONOMIC HISTORY :

DIETARY HISTORY :

GENERAL EXAMINATION :

BUILT

NOURISHMENT

FEBRILE

ANAEMIA

CLUBBING

CYANOSIS

PEDAL EDEMA

LYMPHADENOPATHY

VITAL SIGNS :

PULSE : BP : RR

ANTHROPOMETRIC MEASUREMENTS :

WEIGHT : HEIGHT :

EXAMINATION OF CVS :

EXAMINATION OF RS :

EXAMINATION OF CNS :

EXAMINATION OF ABDOMEN :

INVESTIGATIONS :

Blood Hb :

Urine Albumin :

Sugar

PERIPHERAL NERVE CONDUCTION STUDY :

MEDIAN NERVE	MNCV (m/sec)	SNCV (m/sec)
Right side		
Left side		

2)BAEP RESPONSE :

ABSOLUTE PEAK LATENCY(ms)

Side	WAVE I	WAVE II	WAVE III	WAVE IV	WAVE V
Right ear					
Left ear					

INTERPEAK LATENCY (ms)

Side	I – V	I – III	III – V
Left ear			
Right ear			

MASTER CHART

Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction & Auditory Evoked Potential Response in Children Cases Grade I & II malnutrition

S.no	Age/ Sex	Height (cm)	Weight (kg)	MNCV (m/sec)		SNCV (m/sec)		BAEP Lt						BAEP Rt					
				Lt	Rt	Lt	Rt	I	II	III	IV	I-III	III-V	I	II	III	IV	I-III	III-V
1	5/M	108	16	38.88	65.45	58.82	57.14	1.23	2.68	3.85	4.88	2.62	2.45	0.98	2.12	3.3	4.53	2.32	2.52
2	6/M	114	17	22.44	13.91	34.29	34.29	1.42	2.25	2.88	3.72	1.46	2.24	1.15	1.9	2.72	3.48	1.57	1.48
3	7/M	120	17	28.66	40	62.86	62.86	1.23	2.05	2.88	3.8	1.65	1.94	1.38	2.2	2.95	3.85	1.57	2.75
4	9/M	131	23	68.57	56.6	15	27.91	1.42	3.25	3.98	4.78	2.56	1.27	0.57	2.08	2.95	4.2	2.38	2.17
5	10/F	137	26	18.47	75	45.71	44.68	1.65	2.75	3.45	4.65	1.8	2.2	0.98	1.6	2.55	3.38	1.57	1.6
6	10/M	136	25.5	21.48	17.54	57.14	57.14	1.08	2.48	3.28	4.5	2.2	2.3	1.23	2.2	3.25	4.3	2.02	2.17
7	9/F	130	21	29.72	41.82	19.47	27.85	0.75	2.15	3.75	4.4	3	1.65	0.78	2.33	3.12	4.05	2.34	2.03
8	10/F	135	23	11.95	24.21	28.57	28.57	0.95	2.02	3.32	5.32	2.37	2.96	1.12	2.68	4.2	5.03	3.08	1.98
9	10/M	134	22.5	93.33	79.65	28.57	28.57	1.5	2.58	3.48	4.5	1.98	2.1	0.62	1.6	2.62	3.62	2	2
10	8/M	125	21.8	45	33.46	62.86	62.86	1.9	2.72	3.58	5.08	1.68	2.52	1.05	1.88	2.7	3.5	1.65	1.65
11	10/F	133	25.5	47.17	20	40	40	1.32	2.28	4.35	6.35	3.03	3.85	1	2.02	2.85	3.8	1.85	1.93
12	10/M	134	25.5	100	20.24	62.86	62.86	2.62	3.82	5.2	6.08	2.58	1.92	1.48	2.22	3.08	3.98	1.6	2.07
13	9/M	129	20.9	34.76	29.75	45.71	45.71	1.8	3	4.1	5.6	2.3	2.6	1	2.15	3.25	4.42	2.25	2.15
14	10/M	135	25.5	34.78	37.24	15.49	62.86	0.88	1.68	2.9	3.98	2.02	2.1	0.82	1.35	2.4	2.88	1.58	1.6
15	7/F	119	19	79.81	37.78	57.14	57.14	1	2	3.02	4.35	2.02	2.2	1.3	2.17	4.47	5.52	3.17	3.65
16	10/M	136	25	26.67	23.46	26.32	12.82	0.52	1.82	4.1	4.9	3.58	1.45	1.58	2.58	3.6	4.58	2.02	1.98
17	8/M	124	19	10.1	18.46	9.94	13.22	1.52	2.05	2.68	3.3	1.16	1.24	1.65	2.25	2.98	3.58	1.33	1.17
18	10/M	137	25.5	80	52.31	37.14	6.88	1.08	1.92	2.98	5.08	1.9	4	1.82	3.08	4.03	5.3	2.21	2.29
19	5/F	109	15.5	68.57	12.96	34.62	37.5	1	1.9	2.62	5.7	1.62	4.56	0.7	1.68	2.82	5.03	2.12	4.2
20	6/F	115	16	30.75	22.46	57.14	57.14	0.78	1.7	2.68	4.82	1.9	4.04	0.82	1.88	2.8	4.25	1.98	2.72

Effects Of Protein Energy Malnutrition On Peripheral Nerve Conduction & Auditory Evoked Potential Responses In Children

Cases (Grade III Malnutrition)

S.no	Age/ Sex	Height (cm)	Weight (kg)	MNCV (m/sec)		SNCV (m/sec)		BAEP Lt						BAEP Rt					
				Lt	Rt	Lt	Rt	I	II	III	IV	I-III	III-V	I	II	III	IV	I-III	III-V
1	5/M	89	10.5	20	27.24	34.29	35.88	2.98	4	5.85	6.9	2.87	1.95	1	2.55	3.52	5.12	2.52	3.18
2	6/M	94	11	46.96	14.95	25.97	21.98	0.75	1.55	2.78	3.82	2.03	2.12	2.15	3.42	4.58	5.48	2.43	1.97
3	6/F	95	11.5	67.51	15.67	28.57	28.57	1.05	2.08	4.58	6.2	3.53	3.34	2.38	3.65	5.28	6.32	2.9	3.2
4	7/F	97	13	40	54.49	28.57	28.57	1.72	3.15	3.88	4.45	2.16	3.3	2.05	2.9	4.53	5.45	2.48	2.55
5	8/M	100	14	53.23	20.74	17.39	41.03	1.15	1.9	2.85	4	1.7	1.95	1.2	3.18	4.18	5.2	2.98	2.04
6	9/M	106	18	36.41	41.44	21.43	34.29	1.12	2.2	3.12	4.18	2	2.08	1.6	3.62	4.68	5.42	3.08	3.04
7	10/F	113	17	14.56	20	7.92	45.71	1.32	2.45	3.58	5.1	2.26	2.5	1.35	2.98	4.32	5.85	2.97	3
8	9/M	109	15.5	34.72	13.57	28.57	28.57	1.18	2.88	4.8	5.68	3.62	1.92	2.58	3.82	5.48	6.72	2.9	1.97
9	10/M	112	17	22.5	25.71	37.14	38.24	1.35	3.55	5	6	3.65	2.42	1.52	2.58	3.75	4.88	2.23	2
10	10/F	111	17.5	66.04	93.33	37.14	37.14	1.08	4.38	6.15	6.92	5.07	1.75	1.7	2.3	4.45	6.02	2.75	2.27
11	10/M	110	17.5	26.09	27.27	34.29	34.29	2.3	3.02	4.28	5.25	1.98	1.8	1.78	2.72	3.65	4.55	1.87	1.73
12	9/F	108	16	98.16	38.83	34.29	34.29	1.82	2.6	3.95	5.15	2.13	1.9	1.27	2.42	4.68	7.2	3.41	3.7
13	10/F	109	18.5	36.41	20.35	37.14	37.14	1.7	3.35	4	4.8	2.3	1.58	1.23	2.02	4.03	5.15	2.8	2.92
14	10/M	108	19.5	12.52	111.11	28.17	7.52	1	2.22	3.85	4.5	2.85	1.87	0.65	3.02	4.05	6.4	3.4	3.8
15	8/F	103	14.5	11.01	29.24	17.27	27.3	1.25	2.95	4.28	5.65	3.03	2.84	0.75	1.88	2.6	3.45	1.85	2.3
16	10/F	107	18	7.47	34.84	34.29	34.29	1.15	2.15	3.85	4.9	2.7	3.27	1.42	2.3	3.2	4.53	1.78	4.45
17	7/M	98	12	25.71	43.58	57.14	57.14	1.12	1.85	3.25	4.88	2.13	2.97	0.88	2.15	3.48	4.75	2.6	2.5
18	10/M	106	18	19.78	24.77	18.64	62.86	1.52	2.9	4.22	5.38	2.7	2.28	0.85	2.1	3.3	4.32	2.45	2.35
19	10/F	105	17	12.47	21.11	40	40	1.18	2.52	3.8	4.35	2.62	1.28	1.23	2.22	4.2	5.45	2.62	2.4
20	5/F	88	10.5	13.79	61.35	28.26	13.54	1.62	2.98	3.52	5.28	1.9	2.58	1.88	2.65	4.62	5.3	2.74	1.73

Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction & Auditory Evoked Potential Response in Children - Control

S.no	Age/ Sex	Height (cm)	Weight (kg)	MNCV (m/sec)		SNCV (m/sec)		BAEP Lt						BAEP Rt					
				Lt	Rt	Lt	Rt	I	II	III	IV	I-III	III-V	I	II	III	IV	I-III	III-V
1	5/M	107	22.1	16.93	72	57.14	57.94	1.15	2.3	3.28	4.22	2.13	1.84	1.2	2.05	3.28	5.05	2.08	3.17
2	6/M	115	21	20.57	65.22	45.71	45.71	1.05	2.17	3.28	4.25	2.23	2.17	0.8	2.1	3.4	4.6	2.6	2.25
3	5/M	109	24.7	15.27	28.33	51.43	51.43	0.6	1.98	3.98	4.92	3.38	2.82	1.15	1.98	4.15	5.4	3	2.57
4	5/M	107	25.9	41.44	13.19	57.14	57.14	1.72	2.85	3.82	5.1	2.1	3.56	0.65	2.45	3.55	4.6	2.9	2.27
5	7/F	119	24	42.45	12.63	45.71	45.71	1.6	2.4	4.2	6.05	2.6	2.98	1.27	2.72	3.65	4.7	2.38	2.35
6	6/M	116	20	56.34	45.63	62.86	62.86	1.42	2.9	4.72	8.02	3.3	4.48	0.82	2.05	3	3.82	2.18	2.28
7	8/F	131	28.4	13.22	15.22	57.14	57.14	0.57	1.7	3.18	4.15	2.61	2.6	0.88	2.17	3.18	4.1	2.3	2.07
8	5/M	108	20.1	57.14	53.48	40	40	0.9	2.02	3.28	4.3	2.38	2.17	0.65	1.78	3.32	4.22	2.67	2
9	8/M	125	26	13.33	68.44	45.71	21.62	0.95	2.52	3.82	6.68	2.87	3.46	0.5	1.95	2.82	3.5	2.32	2.63
10	8/F	126	28	40.23	70	13.16	17.26	1.02	2.08	3.08	4.12	2.06	2.24	1.82	2.52	3.9	5.28	2.08	2.95
11	5/M	107	20	53.33	80	34.29	34.29	1.62	2.33	3.42	4.58	1.8	2.18	1.32	4.03	5.03	6	3.71	3.19
12	7/F	123	26.2	14.66	13.17	57.14	57.14	1	1.92	2.92	3.98	1.92	2.03	1.2	2	2.58	3.35	1.38	1.6
13	10/M	139	34.2	68.57	60	30.77	31.25	0.78	2.25	4.58	5.38	3.8	2.47	1.78	2.95	5.32	6.02	3.54	2.1
14	7/F	120	25.6	32.4	21.43	51.43	51.43	1.75	2.72	5.03	6.15	3.28	2.89	1.25	3.6	6.02	7.52	4.77	2.6
15	10/M	137	32.6	72.52	69.34	57.14	57.14	1.2	2.22	3.65	4.65	2.45	1.95	1.3	2.38	3.62	4.75	2.32	2
16	7/F	122	24	42.35	41.1	57.14	57.14	.55	2.3	3.08	5.28	2.53	3.97	0.65	2.5	4.7	5.15	4.05	1.22
17	7/M	121	25.3	65.73	70	45.71	45.71	1.08	1.9	2.88	4.15	1.8	2.87	1.08	3.38	4.82	5.6	3.74	1.48
18	6/M	120	20.6	68	59.23	54.55	70.59	1.42	3.65	4.85	5.85	3.43	1.75	0.9	1.55	2.9	3.82	2	1.68
19	6/F	114	22.1	42.67	38.83	45.71	43.24	1.8	3.1	4.42	5.32	2.62	1.83	1.25	2.02	2.9	3.72	1.65	1.68
20	7/M	120	26	22.07	55.75	51.43	51.43	1.08	2.38	3.42	5.05	2.34	2.2	1.58	2.68	5.05	6.18	3.47	2

Effects of Protein Energy Malnutrition on Peripheral Nerve Conduction & Auditory Evoked Potential Response in Children - Control

S.no	Age/ Sex	Height (cm)	Weight (kg)	MNCV (m/sec)		SNCV (m/sec)		BAEP Lt						BAEP Rt					
				Lt	Rt	Lt	Rt	I	II	III	IV	I-III	III-V	I	II	III	IV	I-III	III-V
21	10/F	137	33	44.44	44.44	47.06	47.06	0.8	2.35	3.7	5.18	2.9	2.45	1.48	2.95	3.78	5.1	2.3	2.82
22	8/F	126	25	86.42	70	45.71	45.71	0.65	1.7	2.75	4.42	2.1	3.3	1.3	2.02	3.18	4.15	1.88	3.24
23	8/M	127	26.3	85	35.86	46.51	58.82	0.98	2.6	3.65	4.47	2.67	2.23	0.78	2	3.28	4.42	2.5	2.12
24	9/F	132	30	18.62	40	62.86	62.86	1.12	2.08	3.95	4.85	2.83	2.13	1.38	2.17	3.02	4	1.64	1.73
25	8/F	127	27.2	18.40	23.53	80	80	0.78	1.75	2.85	4.22	2.07	3.03	0.55	1.42	3	4.47	2.45	3.18
26	7/F	119	24.2	61.73	42.19	26.67	34.03	0.95	1.75	3.02	5.32	2.07	4.53	0.82	2.22	3.9	4.6	3.08	1.95
27	8/M	127	26.1	30	32.02	57.14	57.14	0.88	2.02	3.25	4.18	2.37	2.17	0.72	1.45	2	3.18	1.28	2.38
28	9/F	131	30.1	21.32	22.86	47.06	47.06	0.75	1.52	2.62	3.42	1.87	1.98	1.02	1.85	3	4.1	1.98	1.9
29	5/F	108	23.1	63.83	43.64	45.71	45.71	1.7	2.65	3.35	4.7	1.65	2.6	0.95	1.98	3.25	3.98	2.3	1.7
30	10/F	139	32.3	21.43	18.75	38.1	14.03	1.62	2.25	3.28	4.2	1.66	1.69	0.85	2.58	3.92	5.28	3.07	2.13
31	6/M	113	21.2	71.43	80.19	51.43	51.43	0.68	1.45	2.62	3.92	1.94	2.23	1	2.02	3.18	4.82	2.18	2.37
32	10/M	137	34.6	62.72	55.38	30	51.43	1.4	2.08	2.75	3.5	1.35	2.85	0.82	2.17	3.12	3.8	2.3	2.16
33	9/M	131	29.7	36.96	57.51	38.1	32.65	1.78	2.48	4.45	5.92	2.67	2.75	0.85	2	2.65	3.35	1.8	2.65
34	9/F	140	28.1	12	28.57	51.43	21.69	1.3	1.95	4.45	5.92	2.67	2.75	0.82	2	2.65	3.35	1.8	2.65
35	10/F	139	35.9	80	28.75	37.84	40	1.38	2	2.58	3.85	1.2	2.14	0.7	1.48	4	5.2	3.3	2.55
36	10/F	138	36	35.78	49.53	36.36	35.82	1.98	3.28	4.6	6.75	2.62	3.62	1.18	3.38	4.15	5.32	2.97	2.6
37	9/M	132	30	48	49.59	57.14	23.81	1.62	3.75	3.55	5.3	2.05	3.05	0.98	2.62	4.32	6	3.34	3.53
38	9/F	133	29.7	47.95	88.61	68.57	32.88	0.75	1.58	3.08	4	2.33	2.7	0.72	1.25	3.18	4.15	2.46	1.74
39	9/F	132	28	31.4	61.54	18.32	13.11	1.5	2.35	3.55	5.3	2.05	3.05	0.52	1.82	2.48	3.8	1.96	3.1
40	5/M	110	22.9	24.24	61.54	63.41	42.62	1.18	2.58	3.5	4.55	2.32	1.92	0.55	1.4	2.4	3.48	1.85	2.15