NEUROREHABILITATION
PRINCIPLES & PRACTICE

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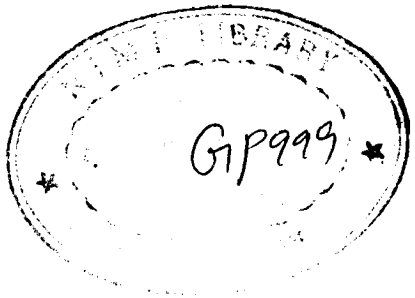
This book is dedicated to
the patients with neurological disabilities
who inspired us to serve them better.



First Published 1998
by Department of Psychiatric and Neurological Rehabilitation
National Institute of Mental Health and Neuro Sciences
Bangalore- 29

Type Setting & Printed at :

Creative Graphics & Technologies
852, 9th 'A' Main, Srinagar,B.S.K Ist Stage Bangalore- 50
Ph/Fax : 628119

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PREFACE

Advances in medicine have led to a significant improvement in the understanding of pathophysiology of many neurological and neurosurgical disorders. Improvement in critical care and therapeutics has remarkably reduced the mortality of several fatal disorders. Much needs to be done for rehabilitation as many survivors of acute disorders eg. stroke, traumatic brain injury, spinal cord injury are left with residual deficits. In addition, several chronic progressive or relapsing diseases eg. parkinsonism, muscular dystrophies, multiple sclerosis and heredofamilial disorders produce considerable morbidity and consequently affect functional abilities. Neurological disorders rank among the commonest causes of permanent impairment, disabilities and handicap requiring long term care and rehabilitation measures.

Epidemiological studies are crucial for proper organization and delivery of health care facilities and information gathered on neurological disabilities is alarming. Biological research and human studies have shown that plasticity of nervous system is a reality. The manipulation of internal milieu and external environment may significantly contribute to recovery, contrary to earlier belief. Formal and informal quantification is important for critical evaluation of rehabilitation measures. Electrodiagnostic techniques have significantly added objectivity to measurement in neurology.

Neurological disabilities are diverse and affect several spheres of life eg. mobility, activities of daily living, sphincter function, cognition and communication among others. These require joint and coordinated effort of a multidisciplinary team consisting of physicians, nurses, physical therapists, occupational therapists, psychologists, speech therapists, orthotists etc. Thus, neurological rehabilitation is a dynamic process. While general framework of rehabilitation, i.e. providing safety, efficiency and independence to an individual, is common, the relative emphasis on different aspects varies according to the disorders eg. cognitive retraining for traumatic brain injury v/s mobility adds for muscular dystrophy and changes with time eg. medical care in acute phase v/s community effort in late

phase. Many patients develop significant complications during rehabilitation process eg. behavioural problems, spasticity, pain, urinary incontinence etc. and need specific attention.

Newer developments in biotechnology eg. computers for aphasia therapy and cognitive retraining, environmental control system and functional electrical stimulation have given new dimension and expanded the scope of neurological rehabilitation. The ultimate objective of the rehabilitation process is community integration of the disabled. Societal participation is a key factor in the success of any programme. The concept of community based rehabilitation is evolving and has already made a mark.

This book provides an over view of various aspects of neurological rehabilitation. Attempts have been made to avoid repetitions and make it a practical guide for one and all involved in the care of patients with neurological and neurosurgical disorders.

A. B. TALY
K. P. SIVARAMAN NAIR
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FOREWORD

A large number of acute and chronic neurological disorders cause irreversible damage to nervous system and lead to significant functional impairment and disability. The research efforts focussed over many decades on structural, functional and molecular issues of nervous system and its disorders have fructified in development of newer therapeutic strategies and measures to limit or prevent tissue damage leading to reduction in mortality and morbidity rates. Despite these many notable advances and achievements, considerable proportions of people with neurological disorders are left with residual deficits, needing a comprehensive approach to rehabilitation for integration into the society. It is estimated that by the year 2020, with the projected population of one billion, there will be 30 million people with neurological disorders. Added to this burden is a large segment of population with traumatic injuries to brain, spinal cord and peripheral nerves. The enormity of the problem of providing neuro rehabilitation services is both a challenge and an opportunity to all those involved and committed to this task.

The concept of "Neurorehabilitaion" with multi and inter disciplinary dimensions is relatively new to this country. There are small groups of a few dedicated experts in India who have initiated programmes. One such nucleus is at the National Institute of Mental Health and NeuroSciences, Bangalore and I am happy that Dr. A.B. Taly, Dr K. P. Sivaram Nair and Dr T. Murali are bringing out this excellent book, a first of its kind in the country. The editors and all the contributors from India and abroad are to be congratulated for this effort. The contents of the book reflect the multi-disciplinary approach to Neurorehabilitation and embodies the crystallised experience of the contributors. I am confident that this book will stimulate many centres in the country to develop neurorehabilitation services. Only then the objective of the book would be truly fulfilled.

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FOREWORD

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ACKNOWLEDGMENT

The editors acknowledge the financial support for this book by the Rehabilitation Committee, NIMHANS, Bangalore, fourth ACIAN Trust and 43rd NSI Trust. We thank the staff of the department of Psychiatric and Neurological Rehabilitation and Department of Neurology for their support at various stages of the book. The editors are grateful to the Director/Vice-Chancellor, NIMHANS, Bangalore, India for the constant encouragement given in this endeavor.

CONTENTS

Epidemiology of Neurological disorders and consequent disabilities-Indian scenario: Implications for rehabilitation policies and programmes.

G. Gururaj, M. Gourie-Devi, A. B. Taly, and P. Satishchandra

Introduction:

India with a population of nearly 950 million spread over 3287.3 ^{2}km is facing a myriad of social, economical, health and technological problems at the turn of the century and the beginning of the new millennium. The country's population is expected to touch a billion mark by the year 2000 AD predominantly comprising of children and young adults. This unabated phenomenon coupled with a gradual decline of communicable and infectious diseases, gradual expansion and reforms in health care, technological advancement in identifying and managing diseases, slow acceptance of preventive technology, change in socio-economic living standards of people and increasing commitments by national and international health agencies have contributed to a significant demographic and epidemiological transition. An important contribution of this has resulted in increase of life expectancy from 32.1 years in 1951 to 60.8 years in 1992, thus adding a significant proportion of middle aged and elderly into Indian communities[1, 2]. While the country is still grappling with problems of yester years, the emergence of new problems have added significant burden on meagerly available health resources.

Hospital and community based research in the last twodecades has brought a number of non-communicable diseases to the forefront of health care delivery system. A gradual decline of communicable diseases has lead to the identification of burden of non-communicable diseases, among which neurological diseases form an important group[3, 4].

Even though neurology has made rapid strides in diagnosis and management of neurological disorders, the related disciplines of neuroepidemiology and neurorehabilitation are still in its early stages from a public health point of view in India. Neurological diseases are characterised by progressive nature, chronicity, recurrence and many of them are without specific drug treatment. Consequently significant physical, mental and cognitive disabilities occur resulting in occupational and social malfunctioning and problems in reintegration into the society. Many of the disorders are difficult to identify and manage by routine methods and it is only recently neurological disorders are begining to be identified as major public health problems[5, 6]. India with around

700-600 neurologists and 600 - 800 neurosurgeons is facing the complex and challenging task of providing care to a large number of neurological patients coming primarily from inaccessible rural areas.

The purpose of the present report is to (i) understand the problem of disabilities in India in the present health and developmental scenario, (ii) highlight the applications of epidemiology in rehabilitation research and programmes, (iii) understand the epidemiology of neurological disorders, (iv) assess the magnitude and pattern of disabilities in selected neurological diseases and, (v) identify the need and possible strategies for neurorehabilitation programmes from an Indian Perspective.

India's devolopment and health scenario:

An understanding and examination of the diversity of the situation and existing resources are crucial for planning, organising and implementing neurorehabilitation services in India for cost-effectiveness, acceptability and sustainability. As per the human development report of 1996, India belongs to the group of low income countries with a GNP of less than 695 dollars at 1993 level. With a Human Development Index (HDI) of less than 0.500, India is ranked 135th in the HDI rank[7]. The existing situation in terms of other parameters is shown in Table 1.

Table 1. Health and Development Indicators 1996.

Population	950 million
Percentage of urbanization	28%
Life expectancy at birth	60.7 years
Adult literacy rate	50.6%
GDP per capita	$ 1,240
GNP per capita	$ 300
Sex ratio (M:F)	1.00 : 0.98
Population with access to health services	85%
Population with access to safe water	81%
Population with access to sanitation	29%
Televisions/100 people	4

Ref. 7

An examination of the health situation in the country reveals a rather slow progress over a period of time. The current birth rate and death rate are 30 and 9 per 1000 population, respecti-vely. The infant mortality rate is 80 per 1000 live births. The morbidity prevalence reveals that nearly 64 and 31 per 1000 population in urban and rural India, respectively, will be suffering from any given sickness at any point of time[8].

In India, 37% of population live below poverty line, only 50% are literate and 72% still reside in rural areas. The average area covered by a primary health centre is around 143 km^2, varying from 37 km^2 in Kerala, to as high as 661 km^2 in Jammu and Kashmir. The average number of villages covered by a primary health centre is around 26, ranging from 1.4 in Kerala to 77 in Arunachal Pradesh. The number of registered medical practitioners and hospitals per 100,000 population is 48 and 16, respectively as on 1992[2]. Even though allopathic system has grown phenomenally over a period of time, ayurvedic, homeopathic and unani practitioners still constitute 36%, 16% and 4%, respectively in India. It is noteworthy that 57% of total hospitals, 32% of beds and 59% of dispensaries are managed and controlled by private sector[8]. Gross disparities have also been noticed in health sickness and health facilities in urban and rural areas, further compounded by socio-cultural practices of communities.

The global and India's pattern of health have undergone dramatic changes since the last two decades. The emerging non-communicable diseases are expected to contribute for seven out of every 10 deaths in India by the year 2020. A new concept of disability adjusted life years (DALYS) has been evolved to measure the impact of premature death and disability. The DALY expresses years of life lost to premature death and years lived with a disability of specified severity and duration. Thus, one DALY is one year of lost healthy life[9].

In India, still 51% of deaths occur due to communicable, maternal and nutritional deficiencies. Deaths from non-communicable diseases are projected to double from four million per year to eight million by 2020. The millions of disability adjusted life years lost during 1990 was 145 and 147 for males and females, respectively, with a total of 292. The rate of DALYS per 1,000 population was 331 for males and 359 for females with an average of 344. The DALYS rate per 1,000 population was higher in younger age groups with a gradual decline during middle ages and an increase again during elderly years[7]. It is argued that health trends in next 25 years will be determined by ageing of world's population, decline in age specific mortality rates from

communicable, maternal, perinatal and nutritional disorders, spread of HIV/AIDS and increase in tobacco related mortality.

Table - 2 Burden of diseases in India and World 1990(hundreds of thousands of DALYS lost).

	India			World	Developing
	Males	Females	Total		World
Communicable, perinatal and maternal causes	772.9	704.4	1477.3 (50.5)	3108.2 (47)	3182.7 (46.3)
Non-communicable diseases	578.9	601.3	1180.2 (40.3)	2234.5 (52.7)	2772.8 (42.5)
Injuries	119.4	147.6	267.0 (9.2)	480.8 (55.4)	533.9 (49.9)
Total	1471.1	1453.3	2924.4 (100)	5823.4 (50.2)	6489.4 (45.1)

Ref. 7

Epidemiology of disabilities in India:

At global level, the United Nations global survey of 55 countries reveals that the proportion of disabled vary from as low as 0.2% to 20.9% in different countries. The survey revealed the obvious lack of standardised methodologies in measuring disability across countries. It was also observed that disabled persons were more often illiterates, had higher unemployment rates and were socially isolated. The devastating effects of disablement and socio-cultural barriers in promoting equal opportunities were the major impediments in promoting reintegration efforts[10].

The 47th round (1991) of National Sample Survey Organization defined disability as "any restriction or lack of abilities to perform an activity in the manner or within the range considered normal for human being". Persons with visual, communication (hearing and/or speech) and locomotor disability were considered physically disabled and included in the survey. As per the results, it was estimated that there would be nearly 16.2 million

disabled as on 1991[11]. Based on these observations, it is estimated that as on January 1997, 17.8 million disabled persons exist in India. About 0.82 million persons become disabled every year. Added to this, an estimated 10 million children with mental retardation with or without physical disabilities exist[12]. Rural areas contribute to a greater extent compared with urban India. The sex distribution reveals that males outnumber females in both areas (Rural - 2.3% V/s 1.7%), (Urban - 1.8% V/s 1.4%). As shown in table 2, locomotor disabilities carry higher prevalence and incidence in rural India compared with urban India. The prevalence and incidence of visual, hearing and locomotor disabilities are provided in table 3. An observation from the survey was that the prevalence of physical disabilities had increased compared with the previous decade[11].

Table 3.Magnitude of the problem of disabilities in India

	Prevalence/100,000		Incidence/100,000	
	Rural	Urban	Rural	Urban
Physically disabled	1995	1579	90	83
Visual disability	525	302	25	20
Hearing	467	339	15	12
Speech	273	237	4	5
Locomotor	1074	962	53	52

Government of Karnataka undertook a total disability survey during 1991 of the entire state, based on successful organisation of the pilot study in 1990-91 in four ICDS blocks[13]. Through an intersectoral input, the survey was carried out by door to door enumeration through grass root functionaries of the health department. As per the findings, the total number of disabled people in Karnataka stands at 355,819 or roughly 1% population of the state. Men and women comprised 58% and 42% in the total series. Among the various categories 58% were orthopaedically handicapped, 14.5% were hearing impaired, 12.5% were visually impaired and 10.6% were mentally retarded. Nearly 2% had multiple disabilities. Bangalore urban/rural district's share of total state's disabilities was 9%. Across the districts, it varied from 1% in Kodagu to 10% in Raichur

districts. Nearly 80-85% of disabled people were living in rural parts of the state. The obvious limitation of this study was that the causation and severity of disability were not examined.

In the survey of disabled persons during 1992-93 in 12 ActionAid supported CBR projects in India, covering a population of 12, 87, 114, it was observed that locomotor disabilities were detected in 33% of the total disabled persons. Visual, communication and mental retardation accounted for the remaining disabilities. The prevalence rate of disabilities was 1.69%, while it was 1.62% for population above 3 years[14]. In terms of technological aid to support daily activities, only 10-20% of disabled people in need of aid and appliances are able to access them. Reasons are high cost, lack of suitability to local conditions, difficulty in maintenance and repair, low volume production, lack of information and socio-cultural barriers and others. Lack of clear-cut policies and research is another major contributing factor[15]. Further, the rejection rate of technical aids is reportedly between 50-75%[16,17].

The above mentioned landmark surveys highlight the problem of disability in the Indian context. Overall, it can be summarised that 3-4% of total country's population have one or more disabilities. Translated to real numbers, this amounts to 30-40 million people, predominantly living in remote parts of rural India. Further, this may not include the mild and moderate type of disability in the country as it requires the usage of more sensitive and specific instruments. It has also been pointed out that only 2-3% of disabled people have access to meagerly available services[15,16,17]. Futhermore, while 70-80% of disabled population live in rural areas the services are predominantly urban based.

Role of epidemiology in Neurorehabilitation process:

Epidemiology is the stepping stone for planning, organization, management and evaluation of health care services. It is an essential tool for provision of services by rationalizing available resources based on prioritization of health problems. All stages of preventive, curative and rehabilitative care depend heavily upon epidemiological methods, techniques and information.

Neuroepidemiology has been defined as "the study of the distribution and dynamics of neurological diseases in human population and of the factors that affect these characteristics"[18]. Epidemiological methods and information has wide variety of uses[4,19] and some important applications are summarised in table 4.

Table 4 Scope of Neuroepidemiology

The ultimate aim of neuroepidemiology is to reduce neurological problems and its consequences in the community by

1. Studying variation in the occurrence and distribution of neurological diseases in different communities.

2. Establishing a community diagnosis of the presence, nature and distribution of neurological disorders through morbidity and mortality rates along with identifying high risk population.

3. Identfying causes of neurological diseases by defining various geographic, demographic, genetic, environmental and social factors.

4. Estimating individual risks and chances towards disease occurrence in general or specified segments of population.

5. Identifying and describing various syndromes in the community.

6. Detecting clinical and subclinical forms of neurological disorders in communities for early diagnosis through screening mechanisms.

7. Studying knowledge, attitudes, beliefs and practices of communities towards neurological problems and to evolve appropriate intervention programmes.

8. Invetigating epidemics of neurological disorders as and when they occur.

9. Completing natural history of diseases affecting nervous system.

10. Developing strategies for planning, organizing, implementing, integrating and evaluating services in communities.

In a very broad sense "Epidemiology helps at developing a framework for understanding the health experience" of communities. It helps in answering specific questions like "what is the magnitude of neurological diseases and consequent disabilities in the community?" How can we prevent diseases or improve recovery? Answers to these

questions will direct our specialists and planners to answer the most vital question "What services are required by patients with these disease(s)?" The information required to answer these questions in the Indian context is very limited. Our extensive search failed to bring out any relevant published literature. While the ultimate aim is "prevention or eradication of diseases" aiming at a 'healthy or disease free' community, the immediate challenge is to aim at organizing medical and rehabilitation services for people who are in need of the same. With specific reference to epidemiology of neurological disabilities, it will enable neurologists, public health planners and communities to maximise the available resources and plan effective interventions for persons with neurological disorders and consequent disabilities.

Epidemiology of neurological disorders

Beginning with the first population based epidemiology study in Vellore by Mathai [20], a handful of studies have been undertaken in India by several researchers in different parts of the country at different time periods [21-27]. Extrapolating these findings must be done with caution due to methodological differences and regional variations in a socio-culturally diverse country like India.

The findings from various population based surveys have been summarized in table 5. As can be seen, the crude prevalence rates of neurological disorders vary from 967 to 3,487 per 100,000 population across the country. As shown in table 5, three of five studies have reported a prevalence of 3% in general population. Keeping this as an estimate and the population of a district in Karnataka to be around 2.5 million, it can be concluded that there would be nearly 7,500 persons with neurological disorders during a one year period. A gross estimate for the whole country would yield approximately 30 million persons with neurological disorders.

These figures are a rough estimate as it has been arrived at by using crude prevalence rates from epidemiological surveys in the absence of a national reporting and surveillance system. An examination of age specific prevalence rates from Bangalore Urban and Rural Neuroepidemiology ('BURN') study reveals that neurological diseases increase from 1st decade upto 3rd decade, a slight fall between 31-40 years and again increase steadily from 4th decade onwards [28]. The sex specific prevalence rates indicate that women suffer more from these disorders compared with men. Highest age specific rates were observed at 60 years and above. Das and Sanyal noticed the prevalence rates to increase from childhood age to 40 years, slight fall in 41-50 years, an increase

in 51-60 years and a fall again during 60+ years [27]. They also registered a higher prevalence among women compared with men.

Table 5 Prevalence of Neurological disorders - Indian scenario

Year	Place	Author	Population Source	Population Surveyed per1,00,000	Crude Prevalence rate	Data collection
1987	Gowribidanur	Gourie-Devi et at	Rural	57,660	1,382	Non-professional workers.
1988	Bombay	Bharucha et al et at	Urban	14010	984	Social workers.
1990	Ballabgarh	Kapoor et al	Rural	48,798	3,487	
1994	Kuttar Valley	Razdan et al	Rural	63,645	967	Anganawadi workers.
1995	Bangalore	Gourie-Devi et al	Rural Urban Total	51,055 51,502 1,02,557	4,070 2,190 3,126	Non-professional workers.
1996	Malda	Das et al	Rural	37,286	2,856	Non-professional workers

Further, the rural-urban differences have not been systematically examined except in 'BURN' study. Strikingly, it was evident that neurological disorders were twice as frequent in rural areas compared with urban areas [28]. This has major relevance for rehabilitation service planning since the neurological services at present are centralized in urban India, while only 27% of the whole country is urbanized as per last census. Interestingly, given the fact that rural rates are higher compared with urban areas, the major bulk of neurological disorders among all age groups are found in rural areas, which is very vital from neuro-rehabilitation point of view.

An examination of the pattern of neurological disorders also reveal the lack of any similarities across regions. However, epilepsy and headaches occupy the first or second place across different places. Subsequently, different disorders occupy various rank orders. This difference could be due to differences in screening instruments, population covered across regions and the prevalence rates[31]. As all these studies are prevalence studies, disorders more often of an incident nature (except post traumatic sequelae) do not figure prominently in this list. Ten major neurological disorders as per rank order in various Indian neuroepidemiological studies are shown in table 6.

Incidence studies of neurological disorders is conspicuously lacking in the whole country except the one on traumatic brain injuries from Bangalore. The first epidemiological study on head injuries from Bangalore revealed an incidence rate of 150/100,000 with a mortality rate of 20/100,000 per year[29]. This corresponds to about 1.5 million persons with head injuries seeking care from various health care institutions with about 2,00,000 deaths per year[30]. Injuries contributed for 7% of total deaths during 1991. With injuries to the brain and spinal cord being as frequent as in the West, the morbidity, mortality and disability due to injuries is a significant burden in India. The exact information on this is not available for the entire country as all types of injuries are not regularly reported to central or state health authorities.

Mortality studies are not available from any part of the country. As per health information of India, 1992, deaths due to disorders of central nervous system constituted 4.4% of total deaths, an increase from 3.9% in 1987 [1]. The agewise break up is shown in table 7. It is noticed that the death rate increase proportionally with age, reaching a peak after 5th decade. The reported deaths are mainly comprised of meningitis and paralysis. With the known fact that certain neurological disorders (eg: stroke, Gullian-Barre syndrome, etc.,) carry high mortality, the reported figures would be a gross underestimate of the situation. Also, the problems of non-reporting from hospitals, misclassification, incompleteness, reporting neuro-logical diseases in death certificates and, inaccurate reporting of deaths occurring at home contribute for under reporting of neurological deaths, leading to a gross underestimate of the situation.

The first generation neuroepidemiological studies in India have hitherto concentrated on studying the prevalence of neurological disorders in Indian Communities. Disabilities arising from the disorders have not been examined comprehensively in these studies. Even though there are wide differences in prevalence across regions due to differences in methodological issues, it highlights the need for more uniform data from many more

Table 6 Major Neurological disorders as per rank order in Indian Neuroepidemiological studies

Gouribidanur	Bangalore Urban - Rural	Kuthar Valley	Malda
Epilepsy	Headache	Peripheral nerve disorders	Vascular headache
Headache	Epilepsy	Seizures	Epilepsy
Mental retardation	Febrile seizures	Spinal cord lesions	Vertigo
Poliomyelitis	Tremors	Poliomyelitis	Vertebral disease
Behaviour disorders	Cerebrovascular disorders	Mental retardation	Stroke
Speech problems	Mental retardation	other extra-pyramidal disorders	Poly neuropathy
Febrile convulsions Gouribidanur	Poliomyelitis Bangalore Urban - Rural	Deaf Mutism Kuthar Valley	Paralytic Piomyclitis Malda
Cerebrovascular disorders	Facial nerve disorders	Stroke	Mental retardation
Cerebral Palsy	Peripheral nerve disorders	Parkinsonism	Movement disorders
Peripheral Neuropathy	Postencephalitic / Cerebral Palsy	Spinal cord disorders	Meningitis sequelae

Table 7 Deaths due to disorders of central nervous system

Age-Groups	<1	1-4	5-14	15-24	25-34	35-44	45-59	60+
Males	5.4	6.1	5.7	3.9	4.7	5.0	15.4	53.7
Females	5.5	8.0	6.4	5.7	4.8	5.3	12.6	51.8

centres. At the same time, it has become essential for "second generation Neuroepidemiological studies", to: study the natural course of neurological diseases at postdiagnosis, delineate the nature and course of illness, identify consequent disabilities and their impact on quality of life. An examination of these findings will bring about the need for manpower development, development of technologies and appropriate solutions for service delivery.

Disabilities among neurological disorders

Planning comprehensive rehabilitation services either in institutions or communities require a basic understanding of the extent, nature and severity of various disabilities along with availability of existing services. In the absence of any longterm followup studies, it becomes difficult to assess the impact of disabilities on individual or families, either quantitatively or qualitatively. Hospital based studies reflect the immediate presence of disabilities at admission or discharge time, which may be at variance with community level assessment due to changes in the course of illness over a period of time influenced by the recovery pattern.

Studies on neurological disabilities from hospitals or communities have been totally lacking in India. The present review is based on some of the studies undertaken by NIMHANS over a period of time. Efforts to identify published literature from the Indian region was totally unsuccessful. Wherever information is lacking, an attempt has been made to examine the issue from Western literature.

A specific neurological disability might arise in many ways from different combinations of many different impairments and, conversely, a single impairment may lead to or exert an influence on several activities[32]. Neurological disabilities are often found to have impairment of activities of daily living, cognitive skills, emotional control along with

a number of other areas. A precise examination of these disabilities is crucial in planning and organizing neuro-rehabilitation programmes. It is also essential to study the mechanisms relating to disability occurrence and the complex interactions of social, cultural, technological and economic factors in developing countries like India. Epidemiological studies are required to understand the complex interrelations of these factors.

Hewer classifies neurological disorders and disabilities under four categories[33] viz.- (i) Dis-orders causing major physical disability affecting mobility and self care (eg., Stroke, Head injury, etc.), (ii) Dis-orders causing disturbance of cognition and behaviour (eg., Senile Dementia), (iii) Disorders causing pain (eg., Migraine, Trigeninal, Neuralgia, etc.) and (iv) those causing disturbance of consciousness and function in a episodic nature (eg., Epilepsy, Migraine). However, majority of neurological disorders overlap across various categories resulting in multiple functional deficits.

From an epidemiological, public health, and rehabilitation perspective, neurological disabilities have to be understood and examined in several ways. These could be based on: (i) Etiology - traumatic, nutritional, metabolic, degenerative, inflammatory, etc., (ii) Course - progres-sive, stationary relapse, remission, etc., (iii) Onset - acute or chronic, (iv) Number - solitary or combined, (v) Diagnosis - only neurological disease or presence of combined conditions and, (vi) Spheres of life or activities affected over a period of time - daily living activities, emotion, cognition, etc.This type of information is vital to see (i) how many patients need acute rehabilitation services soon after identification or discharge of patients from hospitals, (ii) how many need continued care in the long run and (iii) what type of supportive services are required along with continued neurological services.

The NIMHANS hospital based study[34,35] undertaken with the objectives of assessing nature and quantum of disabilities along with determining its impact on social functioning on a series of hospitalised subjects revealed interesting findings. 1093 hospitalised neurological and neurosurgical subjects were evaluated with the 14 item modified Barthel's Index at discharge[36]. Highest number of subjects were in 16-40 years (45.6%), followed by 41-60 years (23.0%). Mean age of patients was 31.0 _19.7 yrs, with male to female ratio of 2:1. Infections (20%), neoplasms (17%), vascular conditions (15%), degenerative casuses (13%) and trauma (11%) were the major etiological groups in this series. Comprehensive disability grading at discharge revealed that 92% (n=1006) of individuals had significant disabilities at hospital dishcarge time. An examination of

severity indicated that 30%, 40% and 23% had mild, moderate and severe disabilities, respectively. Severe disabilities were experienced in the areas of activities of daily living (82.8%), mobility (83%), bladder-bowel control (44%), cognitive abilities (46.7%) and communication (47.8%).

At a mean follow-up time of 6 months, 474 individuals (47%) were contacted to evaluate their health and disability status after discharge. Mild, moderate and severe disabilities were present in 55%, 18% and 6%, respectively. A change over in disability status was noticed in all areas: self care (48.3%), mobility (54%), sphincter control (20.3%), cognition (39.7%) and communication (31.0%).
Futher this study revealed the impact of disabilities on day-to-day activities of the individual in social, economic and voccational spheres of life. Residual disabilities had significant impact on social functioning as noticed by the fact that 55% had reduced social interaction, 79% had decreased ability to discharge family responsibilities, 80% had lost or changed their previous job and 62% experienced economic loss. At follow-up time, 79% had residual disabilities, 24% were moderate to severely disabled and 80% required assistance for day-to-day living.

In the recently completed Bangalore Urban-Rural Neuroepidemiological survey[26,28], 1,02,557 persons were screened by standardized methods and 3,128 persons were confirmed to be suffering from one or more neurological disorders. A pilot study on disability assessment was done by using modified Barthel's index on a 3 point scale and quartile distribution of scores were obtained for classification of disabilities into severe, moderate, mild and nil disabilities. A total of 634 individuals (20.0%) had significant neurological disabilities at community level as shown in table 8. Further, 13% of urban and 23% of rural persons had disabilities, thus implying that disabilities were nearly 2 times more in rural areas probably influenced by duration of illness, nature of health care and prevalence of neurological disorders.

In a follow-up study of 258 adult neurological patients after 12 months of hospital discharge at Chandigargh, it was observed that 29% of subjects did not show any improvement in their status, while 5% had deteriorated[37]. Assessment was done with Dysfunction Analysis Questionnaire on an individual basis. Improvement was noticed significantly in 40%, while 25% improved partially. Poor or no improvement was recorded in patients with congenital, extra-pyramidal, degenerative, and demyelinating group of neurological disorders. Psychosocial disfunction was highest in cases with worsening status. Among psychosocial areas, highest disabilities were noticed in

Table 8 Distribution of disabilities in BURN study

	Disability Present	Disability Absent	Total
Rural	509(23)	1707(77)	2216(100.0)
Urban	125(13)	845(87)	950(100.0)
Total	634(20)	2552(80)	3186(100.0)

Figures in parenthesis indicate percentages.

vocational, followed by social, familial, personal and cognitive areas. Further, elderly patients had higher disabilities compared with younger subjects.

A number of factors affect recovery (total or partial) among subjects with neurological disorders. These include age, sex, physiological status, extent of underlying risk factors, duration of illness, nature of disorder, extent of lesion, nature and presence of comorbidity, hospitalization process, nature of interventions in hospital or communities, accessibility, affordability and availability of health care, type of family, support by family members and others. More important is the level of knowledge - attitude - belief and practices of communities specially in a country where traditional system of medicine is still accepted to a greater extent by people. At the same time, at the community level there is a need to examine the various disabilities from a wider perspective as delivery of rehabilitative services is an integrated approach depending on the commonalities of disabilities.

In the following sections, an attempt has been made to examine the epidemiological dimensions of specific neurological disorders and consequent disabilities. The pattern and extent of disabilities along with their impact on quality of life has been highlighted. The selection of these conditions is not random, but selective to represent different age groups, causes and nature of disorders. The disorders covered are epilepsy, poliomyelitis (children), stroke, head injuries, spinal injuries (adolescents, adults and middle aged groups), Parkinson's disease and dementia (elderly). The existing lucunae are highlighted and need for further research is emphasized.

1. Epilepsy:

The most frequent of all neurological registrations in any health care setting is epilepsy. Among the community based neuroepidemiological studies in India, epilepsy has been the most commonest one. The prevalence of epilepsy ranges from 200-1200 per 100,000 in India [31,38]. Among children, this varies from 64-177 per 100,000 children. In Karnataka, the prevalence of active Epilepsy ranges from 400-900 per 100,000 from four of the studies conducted [39].The incidence rates of Epilepsy are not known in India, but literature from the West reveal it to be around 20-50 per 100,000 person years [38]. Epilepsy constitute 25-50% of all patients with convulsive disorders. Almost all the studies have concluded the preponderance of males and younger age groups. Longitudinal studies from developed countries reveal that nearly 70-80% will become seizure free over a period of time. The relapse rate after a five year seizure free interval is about 1% [40]. The prevalence of active epilepsy was 882/100,000 in BURN study [41]. Prevalence in rural Bangalore was two times high compared to urban areas (1,192 v/s 575, respectively). Specifically, the prevalence rate among children in rural areas was 1,346 while the urban rates were 447/100,000.

In India, epilepsy may not contribute for major disability but carries significant psychosocial burden for individuals and families. Shorovon [42], in a review of problems faced by epileptic persons highlight that patients with Epilepsy face considerable problems in the areas of education, occupation, marriage and social relations. Effects of Epilepsy have also been noticed on learning and progress at school (expectation, intellectual impairment, learning disability, school absenteeism, poor self image, social isolation, guilt, overdependance), on women in the areas of marriage (divorce, depression, childbirth), leisure time activities (sports, travel, etc.), employment (driving, working in industries) adjustmental problems and other areas. Often these are difficult to quantity but their impact is considerable.

With regard to work, difficulties are experienced in obtaining-maintaining-progressing in job by epileptic patients. The PL 480 study on Epilepsy [43] observed that there was very minimal impact on work status among epileptic patients, since only in 9 out of 430 (2%), work status was adversely affected. However, 8% reported deterioration in their work performance. This increased to 25% among those without control of seizures. From a patient perspective, 6% reported that epilepsy adversely affected their prospects at work. At the end of 3 years follow-up, nearly 26% had intelligence levels of average and above, 58% average, low and borderline and mental subnormality was detected in

16% of patients. Among children where the frequency of seizures had not reduced progress in studies was hampered to a considerable extent.

As early as 1977 the Commission on Epilepsy has stated that the most neglected aspects of epilepsy are social, psychological and behaviour problems, which are very common. Levin et al[44], in a review of psychosocial dimensions of Epilepsy, observed that persons with epilepsy notice lower rates of marriage, considerable sexual difficulties, greater unemployment and underemployment, attitude of nonacceptance of self, fear of disclosure, discriminatory attitude in society along with a wide range of other psychosocial problems. Psychiatric comorbidity has been noticed significantly among epileptic patients. Prominent among them are, psychoses (7-10%), anxiety disorders (10-20%), various personality problems and others like sexual problems, preoccupations, dependency and lack of stability. Affective disorders are also known to be associated with epilepsy. The associated comorbidity in terms of depression and suicidal thoughts is also a major area of concern from a mental health point of view. A number of psychiatric problems like depression (40-50%), psychosis (2-3%) behavioural problems have been reported. In a recent large scale European study on Epilepsy and everyday life risks[45], it was noticed that there was no significant difference in terms of accidents, number of medical contacts and others over a period of 12 months. It was noticed that patients with epilepsy reported slightly more accidents compared with controls (10% v/s 8%).

2. Paralytic poliomyelitis:

Better immunization rates due to national and international commitments have resulted in a gradual decline of poliomyelitis across India with the introduction of oral polio vaccine in National Immunization Programmes in 1979. Despite phenomenal increase in immunization coverage rates, specific geographical pockets are still being identified in the country. In a selective review of progress in poliomyelitis control in selected states and union territories of India, the reported incidence of paralytic poliomyelitis has declined from 5.7/100,000 in 1980 to 1/100,000 in 1990 and a further decline is expected[46]. If Vaccination Programs are not effective, nearly 50,000 children will develop poliomyelitis every year.

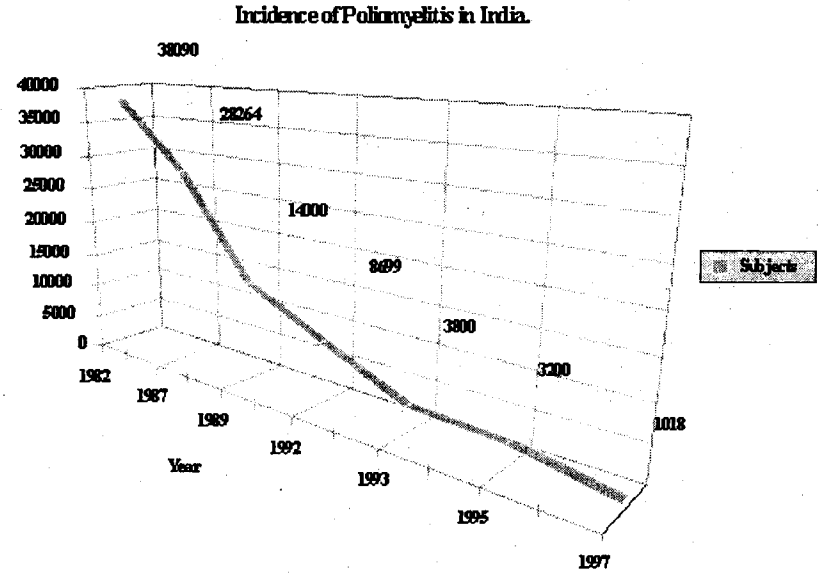
The annual incidence of poliomyelitis in the country was 1.7/1,000 children during 1981-82 based on sample surveys. The number of new cases of paralytic polio has reduced from 28,264 in 1987 to about less than 1,000 cases by 1997, thus showing the impact of universal immunization programme as shown in figure 2. Lameness surveys have

demonstrated that the earlier prevalence rate of 5-6/1,000 has significantly reduced to 1/1,000 after strengthening immunization programmes[47]. The surveillance on polio has been further intensified from 1997 with identification of all children with acute flacid paralysis as an important strategy.

Prevalence of Poliomyelitis in India from neuroepidemiological surveys is observed to be 218/1,00,000 in Kashmir[24], 99/100,000 in Gowribidanur[21], 53/100,000 in Bengal[27] and 75/100,000 in urban-rural Bangalore [25]. Das et al observed that 80% of cases were below 15 years of age with a slight preponderance of women over men [27]. The age specific rates were 132 and 156 per 100,000 population in 0-5 and 6-10 years age group, respectively. Paralytic poliomyelitis was detected among 77 subjects (23 urban and 54 rural) in 'BURN' study. Walking even short distances was affected in 60% of children. Daily activities of bathing, dressing and grooming was difficult in nearly 25% of subjects. Nearly 68% could not do any work because of residual paralysis.

3. Traumatic brain injuries:

While developed countries have successfully reduced the burden of Traumatic Brain Injuries (TBI's) through balanced, realistic and integrated strategies of engineering,



enforcement and education, countries like India are registering an increase of TBI's over a period of time. A number of factors like age and sex composition of the population, socioeconomic standards, technological progress of societies and lack of rehabilitation programmes play a key role in the nature and burden of TBI's.

Globally, injuries constitute for 5.2% of total deaths and 10-30% of hospital admissions[48]. Within this group, head injuries contribute for 5-30% of total injuries depending on the case definition and case ascertainment methods. Nearly 40% of all injury deaths are related to TBI. The incidence and mortality of TBI as a global estimate is around 200 and 30 per 100,000 per year, respectively. The prevalence of TBI is not known clearly and it is estimated to be 120/100,000 per year [49]. Head and spinal cord injuries contribute for 50% mortality in severe brain injuries [50]. It is acknowledged that 100% severe, 60-80% of moderate and 10% of mild TBI's require long term rehabilitation services 49. Majority of the studies have shown a male preponderance and affliction of younger age groups, with road accidents contributing for 60-70% of head injuries. The case fatality is around 10% across the world. Further, for each fatality, 7-10 persons are hospitalised and 60 persons seek brief emergency care[51].

In the epidemiological study of head injuries in Bangalore [29], the incidence, mortality and case fatality was found to be 150/100,000, 20/100,00 and 10%, respectively. The male to female ratio was 1:0.3. The highest age group was 20-29 years (26.5%), followed by 30-39 years (19%). Children (<15 years) and elderly constituted 20% and 5.3% of the total series, respectively. At 4 months post-discharge 415 subjects were followed-up at home to identify current health status and posttraumatic sequelae[52]. It was noticed that nearly 50% of the patients were still in different stages of recovery. Among the various deficits reported by patients and their families, posttraumatic headache, anxiety features and memory problems were predominant in 25%, 16% and 14%, respectively. Posttraumatic epilepsy was diagnosed in 5% of the subjects, for which they were receiving treatment. Locomotor disabilities were reported by 6% of subjects affecting their mobility and work. Further, 37% of adults and 20% of children could not attend to work or school, respectively, for more than 60 days, which was mainly dependant on severity of head injury. Surprisingly, even those with mild injuries had severe disabilities in terms of behaviour and memory problems.

With significant improvements in managing patients at hospital levels, the survival rates of head injured persons is significantly increasing in Indian community. Severe disabilities have been noticed in 3-40% of subjects depending upon severity of injuries and time

frame of follow-up. Several studies have noticed that 30-40% had returned fully to their previous jobs and 20-70% were still not working at the time of follow-up[52]. Also, it has to be examined whether the extent and quality of work had deteriorated as compared to the previous status. Nearly 6% of the patients had incurred an expenditure of more than Rs.15,000/- during the period of follow up for rehabilitation services.

In the NIMHANS's hospital based study[34], disabilities were assessed with the help of modified Barthel's Index on a 4 point scale. The areas of measurements included activities of daily living (bathing, dressing, grooming, eating, sitting, standing, walking and climbing), bladder and bowel control, vision and hearing, memory, communication and social interaction, family responsibility and working status. 98 patients were assessed at hospital discharge time and 42 (43%) were followed at 6 months time. The distribution of total disability at discharge in terms of - severe, moderate and mild was 46%, 30%, 23%, respectively. Examination at 6 months period revealed the disability distribution to be 5%, 14% and 81% across severe, moderate and mild groups, thus, signifying that the reduction in severe and moderate cadres was around 70%, while prevalence of mild disability had increased from 23-81%. Among the activities of daily living, nearly 30% had difficulties in their regular day-to-day activities requiring help and support from family members. About 5% had difficulty in bladder and bowel control, 25% in vision and hearing, and 53% reported memory impairments at 6 months. The social interaction was restricted and reduced in nearly 50% of patients. At 6 months follow-up, 14% could not undertake any family responsibility and 16% had not returned to work.

Sabeshan and Natarajan[53] reported adjustment problems of head injured patients and their families in a 2 year follow up study at Madurai. They noticed significant disabilities in all areas of family activities on a different scale. The presence of significant neuropsychological sequelae in 29 of 141 patients at 18 months post-discharge in Madurai was reported by Natarajan[54]. Severe day-to-day adjustment problems were noticed by patients and family members affecting day-to-day living.Traumatic brain injuries are reaching epidemic proportions in the Indian region. Unless comprehensive preventive and neurorehabilitation services are planned and implemented, the burden of this group of neuro disorders will continue to increase significantly 30.

4. Spinal cord injuries:

Traumatic Spinal Cord Injuries (TSCIs) are a serious condition resulting in severe disability or death, with survivors facing myriad of health problems and multiple

complications affecting their day to day living. Indian literature on epidemiology of spinal cord injuries and consequent disabilities is totally lacking, except one study from Bhubaneshwar, Orissa. A review of literature from around the world reveals that the incidence of TSCI varies from 9-53 per million per year[55]. Majority of the studies may not include those spinal cord injury deaths associated with prehospital deaths. Further, mild to moderate spinal cord injuries may not be identified clearly in the presence of polytrauma. Kraus et al, in a study of 18 hospitals in 18 California counties identified the total incidence as 53.4 per million population, which is much higher than the reported figures[56]. There are only two reported studies from the Asian region. The incidence rates in Malaysia was 27, in Taiwan it was 56, and in Japan 40 per million population[55,57,58]. Mortality rates for TSCI vary from 4-17%. The case fatality rates vary from 5-20% depending upon injury severity and age. The prevalence of SCI worldwide, is estimated to be around 500 per million population[51]. From the available literature, it is known that majority of the individuals will be in the age group of 20-30 years with a male preponderance (M:F ratio :: 3-4:1). Traffic related injuries were the primary cause for 50-60% of spinal cord injuries, followed by falls (20-30%), sports and occupational injuries (5-10%). About 70% new cases of SCI appear in less than 30 years of age.

Persons with SCI experience considerable morbidity due to varied neurological and other disabilities. Lan et al[57] reported that among hospitalised SCI patients, 36% were tetraparetic, 33% tetraplegic, 15% paraplegic and 12% paraparetic. Complete paralysis was noticed in thoracic cord injuries, while incomplete paralysis was more frequent among cervical cord injuries. Among newly diagnosed SCIs, lesions in cervical region contributed for 50% of total cases[55]. Complications related to genitourinary system (25%), cardiovascular complications (24%), respiratory system (14%), septicaemia (4.5%) and others, were the major problems for continuous and long term management of individuals with spinal cord injuries.

In a recent medico-social survey of spinal cord injury patients[59], it was noticed that majority of SCI subjects had serious financial difficulties either through loss of jobs or lack of alternative source of income. 42 (66%) individuals were totally confined to bed and resources were unavailable to make any changes in adaptation of home. Urinary catheters, condom drainage and drugs were totally unavailable. The unmet living needs in terms of information and referral, skills training, peer counselling, advocacy were found totally lacking.

In the only comprehensive study undertaken by the Shanta Memorial Rehabilitation Centre in Bhubaneshwar, Orissa, the annual incidence of spinal cord injuries was 20 per million population per year for the period 1985-1990[60]. The male to female ratio was 3:2. Nearly 50% of SCI subjects were in 20-40 years. Spinal injuries with cord involvement, paraplegia and quadriplegia accounted for 57%, 30% and 12% of total series. Falls, road accidents and fall of objects were the major underlying causes in 53%, 26% and 12%, respectively. Nearly 62% were admitted through primary health centres, thus indicating the increasing occurrence of SCI in rural areas. The duration of hospitalization was nearly 30 days in 67% of subjects, 30-45 days in 22%, 45-60 days in 5% and, above 60 days in 6% of total series. The case fatality rate was 11% (more than 50% being quadriplegics). Death rates during admission time was 47%, 7% and 5%, for quadriplegia, paraplegia and suspected cord lesion with spinal injury, respectively. The average cost of managing SCI persons during hospitalization period ranged from Rs.4,500 - Rs.18,000, depending upon the type of care. The study concluded the obvious lack of facilities in terms of human resources, technology, infrastructure and others and strongly recommended for setting up of spinal cord injury centres in the state and rest of the country.

In a review of psycho-social implications after spinal cord injury in India, Chandra concluded that disabilities lead onto psychological abnormalities, vocational maladjustments and personality disintegration, thus affecting the overall quality of life[61].

5. Cerebrovascular accidents:

Stroke has been identified as a major public health problem in recent decades in India due to its - (i) increasing incidence, (ii) high mortality, morbidity and disability, (iii) increasing occurrence due to general increase in prevalence of associated health problems like hypertension, diabetes, alcoholism, etc., and (iv) an ageing population. Stroke constitutes 2% of all hospital registrations, 1-5% of total medical admissions and 9-30% of total neurological admissions[62], CVD's include a wide spectrum of conditions ranging from transient attacks to recurrent strokes and each diagnostic category has an independent epidemiological profile.

World over, mortality rate due to stroke is estimated to be 50-100 per 1,00,000 population[63]. At NIMHANS, the proportional mortality rate from stroke was found to be 17.2%[64]. In a five year analysis of stroke cases registered at NIMHANS, it was noticed that TIAS, Ischemic and Haemorrhagic strokes constituted 14.7%, 73% and

12% respectively. Stroke in the young (<40 years) constituted 28% of total series and 32% of total deaths.The absence of information on incidence presents lacuna in identifying the occurrence of annual cases. Worldwide, it is known that the incidence of stroke is around 2/1,000, which means there will be nearly 200 new cases in a population of 100,000 per year[33]. Among them, it is established that 30% will die in first 4 weeks, 30% will recover and 40% will be left with one or more disabilities.

An epidemiological analysis of stroke literature from India reveal that the prevalence of stroke varies from 44-842/1,00,000 population with a male preponderance[21-24, 27, 65]. The prevalence of stroke in Kashmir 22, West Bengal[27], Bangalore[65] and Bombay[23] was 143, 126, 151 and 844 per 100,000 population, respectively. Razdan et al 22 and Gourie-Devi et al[65] noticed an increasing prevalence in age with males. As mentioned earlier, the studies are incomparable due to differences in methodology and time period changes. Two important emerging trends noticed are the occurrence of CVDs in the young[65] and increasing occurrence of cerebral venous thrombosis among women[67], requiring further understanding.

In the NIMHANS hospital based study 34, 68 subjects were hospitalised during study time. An assessment of comprehensive disability at discharge revealed that 34%, 37% and 20% had mild, moderate and severe disabilities at discharge time. At 6 months follow-up time, this had substantially changed to 52%, 15% and 6% across the same categories, respectively. At hospital discharge time, significant difficulties were experienced in the areas of activities of daily living (75%), mobility (80%), bladder control (42%), cognition (57%) and communication (50%). At follow-up time, the rates were 51%, 47%, 17%, 50% and 35%, respectively across same areas, thus revealing the persistence of deficits over a period of time.

In the 'BURN' study[65], 151 subjects of completed stroke were identified, with a mean duration of >3 years in 47% of subjects. Based on this observation, it is estimated that nearly 1.5 million persons with completed stroke are likely to be present in India. Further, nearly 1% of elderly were found to be suffering from stroke. Nearly 30% of patients were younger than <40 years. Prevalence of CVDs was higher in rural areas as compared to urban areas (61% v/s 39%). An examination of age-specific prevalence rates indicated that rates increased with age from 42/100,000 among <30 years to 1099 in 71+ age group. 61% of young stroke subjects had significant disability. Severe, moderate, mild and no disabilities were present in 8%, 22%, 41% and 29%, respectively. Severe disabilities were noticed among 13% of urban subjects compared with 3% of

rural residents, while mild disabilities was 50% among rural compared with 30% of urban subjects. This unexpected gross difference may be related to a number of factors requiring further investigation. Among the various components of disabilities, activities of daily living (bathing, dressing, grooming, mobility) was affected severely and moderately in 10-20% and 40-50% of subjects. Nearly 35% had difficulties in bladder/ bowel control and required daily support. Severe and moderate disabilities related to memory was noticed in 6% and 40% of subjects. Communication and social interaction was affected in 40-80% of persons at varying levels. Nearly 56% could not continue with their previous work and 30% worked less than before.

6. Parkinson's disease:

Another major neurological condition which is progressive and of a degenerative nature affecting the middle aged and elderly is Parkinson's disease (PD). The prevalence of Parkinson's disease in the studies at Gowribidanur[21], Kuthar valley[24], Malda[27] and Bombay[23] was 7, 14, 16 and 328 (Parsi community) per 100,000, respectively. The prevalence in the 'BURN' Study was 33/1,00,000 (n=34). An examination of age-specific prevalence rates revealed an increasing occurrence with age. The rates were 28, 95, 243, 501 and 573 at 31-40, 51-60, 61-70, 71-80 and above 80 years, respectively. Sex-specific prevalence rates indicated the male and female prevalence rates to be 83 and 64 per 100,000 population, respectively. Razdan et al[24] also noticed that the prevalence of PD was associated with age, with rates being 134 and 247 for those >50 and >60 years of age. Das and Sanyal from Bengal[(27)] made similar observations on the association of PD with age and sex. Age-specific prevalence showed a progressive increase from 86/100,000 in 41-50 years to 128 at 51-60 years and 260 for 60 + years age group. The rates among males and females was 22 and 10 per 100,000 population, respectively.

In the disability assessment of Parkinson's Disease in the pilot work of 'BURN' study, 74% of subjects were experiencing moderate to mild disabilities, and in the remaining 26% activities and partici-pation were not hampered significantly. 63-68% experienced difficulties in bathing, dressing and mobility. Support from family members was required for eating in 42% of subjects. Bladder/ bowel control was a major difficulty in 42% of subjects. Memory disturbances were moderately experienced in 63% of subjects. Social interaction, family responsibility and working pattern was affected in more than 50% of subjects. It is known that disabilities increase in severity and extent in PD along with age and significant numbers have to depend on family members or others for day-to-day living.

7. Dementia:

Population distribution across the globe has revealed that the elderly increased from 376 million in 1980 to 590 million by 2000 and then to 976 million by the year 2020. Developing countries will constitute 70% of world's elderly population[68]. The demographic transition in India along with changing life styles and values have brought geriatric health problems as an important dimension in the reforming health care systems. With advances in health care technology and decline of communicable diseases, Indian elderly are experiencing longer life compared with the past. The proportion of elderly has been increasing from 6.0% in 1980's to 7% in 90's. It is expected to be around 8% by the turn of the century accounting for 70-80 million elderly in India[1,2].

A simple definition of Dementia is "global deterioration" of the individuals intellectual, emotional and cognitive faculties in a state of unimpaired consciousness[69]. Several reports have established that 1% of those below 65 years, 4-6% of 65 + and nearly 50% of those beyond 85 will be suffering from dementia. Alzheimer's disease contributing for majority of dementia's has a prevalence of 3-15 per 100 among those aged 65 years and above, with an incidence rate of 100-1500 subjects per 100,000 per year[69]. Almost all the studies have showed increasing prevalence and incidence with age and a higher rate among females compared with males[70].

Wadia has reported the prevalence of dementia in India to be varying from 6-10% after the age of 60 years from data collected through small surveys[71]. Selective surveys of 'elderly' and 'mental health problems of elderly' have examined dementia in the Indian region. Rao V, studied 150 subjects aged 60 years and above at Madurai for prevalence of mental health problems[72]. 72% and 38% were in 60-70 years (young old) and 70+ (old old) age groups, respectively, with a male to female ratio of 3:1. Dementias were diagnosed in 20% of subjects. Alzheimer's and multiinfarct dementias constituted 35% and 65% in the total series. About 67% of elderly had sensory handicap with 63% and 33% having visual and hearing disablities, respectively, and 10% had musculo-skeletal handicaps. Nearly 15% of 'elderly ill' subjects were either rejected or unwanted by families. In a geropsychiatric survey by the same authors[73], 4.4% of population was above 60 years and chronic organic brain syndrome was detected in 10% of this population.

The prevalence of the condition in general neuroepidemiological surveys is difficult to estimate. Except the 'BURN' study[26], no other studies have detected dementia in

population based neuroepidemio-logical surveys. In urban-rural Bangalore, elderly constituted 5.1% of total survey population. Prevalence rate of dementia was 12/100,000 in urban and 14/100,000 in rural with a combined rate of 13/100,000. Examination of age specific prevalence rates revealed the rate to be 114/100,00 in urban and 110/ 100,000 in rural areas, thus revealing the fact that nearly 1% of elderly suffer from dementia. The lower rates from India may be due to lack of specificity of questionnaire to detect dementia in general population surveys. An overview of disability status in 8 subjects revealed that nearly 50% (n=4) had severe to moderate difficulties in the areas of bathing, dressing, grooming and mobility. Five subjects experienced significant difficulties in walking, 5 subjects had to depend on support of family members for bladder and bowel evacuation. Memory was severely and moderately impaired in 2 and 4 subjects at the time of study. Correspondingly, communication was deranged in all 6 subjects. Social responsibilities and interaction was affected in 8 persons under investigation. None of them were able to carry on with their previous work.

Issues in measuring neurological disabilities

Health needs of disabled persons in families and communities extend beyond medical diagnosis in terms of the disablement (includes impairment, activities and participation). With the acknowledgement that most of the neurological disorders are associated with disabilities of various types affecting physical, social, mental and vocational spheres of an individual's life, identifying these disabilities is crucial from a rehabilitation perspective. Disabilities in neurological diseases vary from acute to chronic, stationary to progressive, disease to disorder, individual to individual - with immediate to long term impact on patients and families. The perception and management of these disabilities vary across regions depending on political, social, economic, cultural and technological growth of societies, which is extremely diverse in a country like India. With a paradigm shift in health care from acute to long term care, understanding the consequences of neurological diseases and its impact on quality of life is very much essential[74]. Since health sector is a partner in the process of community development, a closer co-operation is vital in managing chronic neurological patients in communities.

The recent International Classification of Impairments, disabilities and handicaps (ICIDH2)[(75)] provides a broader framework for this understanding and a common language for research, management and social reforms. ICIDH combines the status and experiences of people in situations or circumstances of their living to examine the consequences of health related states. Shifting from an earlier model of disease -

impairment - disability and handicap, it emphasizes to examine the issue in a multidimensional sphere of impairments - activities - participation limitation - restriction in the broader context, amidst environmental and contextual factors of an individual.

Due to rapid reforms in health care policies and programmes, assessment and outcome from neurological diseases has been an important research topic of the west. A number of scales have been developed to measure impairment, disabilities and handicaps[76,77]. While controversies exist on the choice of a rehabilitation scale, more often it depends on what one is looking for and its properties of simplicity, sensitivity, specificity, reliability and validity in specific sociocultural ethos and living standards of a given community. In the area of neurological disability, it is important to measure cognitive impairment, motor impairment, focal disability, activities of daily living, quality of life, emotion and social interaction, family responsibilities, and others. While measuring these areas, it is important to develop these instruments in the socio-cultural setting where the person with disabilities lives, thus enabling professionals to develop culture specific rehabilitative measures. In India, an attempt is yet to begin in this direction.

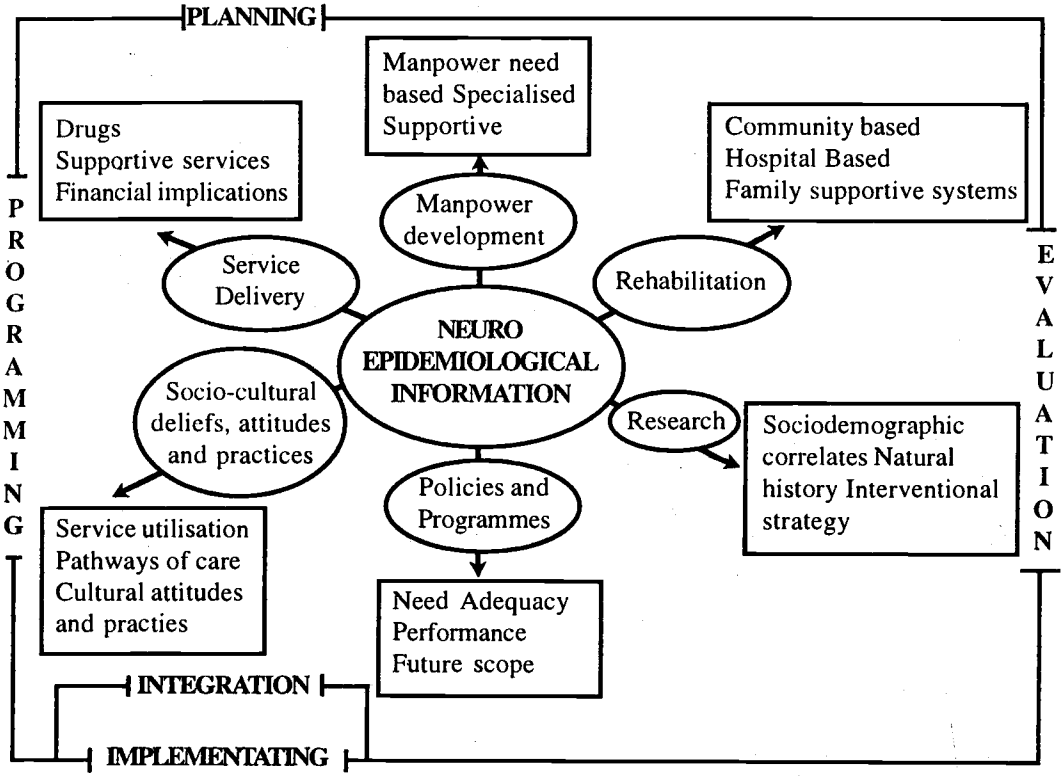
There is a need to identify the consequences of neurological diseases for rehabilitation programs. Using epidemiological data as such has some inherent limitations such as nonavailability of data on less frequent neurological diseases[33]. Also, rehabilitation needs have not been routinely examined in Indian neuroepidemiological studies. Hence, there is need to generate new information in this regard. However, the problem is further compounded by lack of suitable methodologies and culture specific instruments. The next generation of population based neuroepidemiological studies must include these aspects while, disabilities have to be examined in individual neurological disorders. In this area, epidemiological meth-odology can contribute for a better understanding of mechanisms and impact of disablement.

Implications of neuropidemioligical findings for neurorehabilitation services:

With the available evidence till date, it is estimated that nearly 25-30 million people will be suffering from neurological disorders at any given point of time in India and numbers are likely to increase in coming years. The studies have identified a wide spectrum of neurological disorders varying across place, person and time. The total extent, nature and burden of neurological disabilities is not clearly known, but the available data indicate its reflection to a greater extent. Population based epidemiological information has a wide variety of uses as shown in figure 2. Specially for neurorehabilitation services, it

provides information on the number of people requiring short term and long term care at the community level. This in itself will propel activities on manpower development, technology and continued research. Individuals with neurological disorders experience considerable difficulties in day-to-day activities of living, mobility, speech, hearing, communication, memory, social interaction, family responsibilities, etc. Further, the occurrence of disorders in prime and productive age groups place significant burden on education, work, income, family development, etc. and families have to support the disabled persons to a greater extent (at a time when family values are changing considerably)

In India nearly 3.8% of population is disabled due to one or more health problems. Translated to real terms, this amounts to about 38-40 million people requiring rehabilitation services. The precise contribution of neurological disorders in overall disabilities is not known and any estimations have to be done cautiously. However 40-50% of disabilities could be due to neurological disorders (!) with variations across type of disabilities.



Organising rehabilitation services in a country with 500-600 neurologists is a complex and challenging task. The task is further compounded due to manpower deficiencies in allied rehabilitation services. Several initiatives have been developed by government of India in recent years including setting up of the district rehabilitiation centres in selected areas [78]. The emergence of NGO's in rehabilitation during the recent decades is a noteworthy phenomenon to promote services for handicapped persons. The recent promulgation of equal opportunities act - 1995 by government of India is also a committment in the right direction. However, all these efforts in the country indicate the begining of a momentum for the rehabilitation movement.

With the known fact that only 3-5% of disabled people receive appropriate care, the vast majority hardly receive any inputs for their rehabilitation. Further, the services are still hospital and urban based, while the extent of urbanisation is only 28%. With the shift in focus from institutions to communities beginning around 1950's and 1960's, community based initiatives have gained momentum across the world. With the emergence of community based rehabilitation (CBR) services during the last decade, a paradigm shift has occurred in the issues of disabled people. CBR moves beyond traditional rehabilitation concepts and ideas, as it aims at providing efforts to CHANGE community's behaviour to ENABLE community members to have a better understanding of DISABILITY issues (socio-economic, socio-cultural, psycho-logical, etc.) for providing a POSITIVE environment (physical, psychological, socio-cultural, economic, etc.) to IMPROVE the QUALITY of life of the disabled. This is an emerging concept of CBR, which is FOR and BY people [79].

The emerging neurorehabilitation concept in India has to be viewed in a broader perspective of ongoing epidemiological transition and existing rehabilitation programmes. While the general principles of rehabilitation remain same, the techniques and skills for individuals, families and communities must be geared to meet the persisting disabilities among persons with neurological disorders. Then, the obvious question is "How can people with neurological disabilities be integrated into ongoing or expanding CBR programmes in India"? This is a debatable question as neurorehabilitation is an emerging speciality within neurology, while CBR is a becoming a grass root descipline. The widening gap is accentuated further as the technical manpower for CBR usually relies upon families and community workers, while, neuro-rehabilitation is being undertaken by neurologists and allied specialities. "The one and the only way is to take a public health and epidemiological approach to bridge this gap". At this time, answers have to obtained from concerned corners.

Agenda for the future:

Planning, organizing and implementing neurorehabilitation services in a country nearing 1 billion population characterised by: 37% living below poverty line, 48% being illiterate and 73% residing in rural areas along with diverse sociocultural factors is a challenging task for everyone involved in this process. Optimum utilization of limited manpower expertise and integration with ongoing community based rehabilitation programmes seems to be the obvious choice. Undoubtedly, health professionals have to work with other partners and develop an intersectoral approach to meet this challenge. For such programmes to be effective, cost effectiveness and local technology need to be taken into account. Community participation is the enabling yardstick for success or failure of these programmes.

Future areas of research, policy and programme issues has to answer several questions. Some of these are - (i) the need for more neuroepidemiological studies from different parts of the country to understand problem, patterns and course of neurological disorders, (ii) epidemiology apart from estimating prevalence or incidence, should incorporate disability assessment and its impact on quality of life through longitudinal studies, (iii) incorporating neurorehabilitation components into community based rehabilitation services and (iv) studying the feasibility and sustainability of such programmes in the evolving sociocultural, political and technological progress of the country.

Epidemiological information has great implications and should be viewed beyond 'rates and ratios'. For those whose heads are counted, answers have to be provided for a 'meaningful and optimal life'. For professionals and policy makers, it has to 'move from an academic exercise towards developing sustainable rehabilitation programmes'. Undoubtedly, such programmes have to be available - accessible - affordable and applicable to persons with neurological and other disabilities in the entire country.

The final goal is physical, psychological, occupational and social rehabilitation and reintegration into family and society of a person with disability consequent to neurological disorder.

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Neuronal Plasticity - a unique property for Neurorehabilitation

B.S. Shankaranarayana Rao, B.L. Meti, T.R. Raju

Introduction

Neuronal plasticity is an important property of the brain and refers to morphological, biochemical and physiological changes occurring in both the adult and developing nervous system. Although neuronal circuitry is capable of undergoing different types of changes, synaptic changes are the most critical ones because they alter communication between neurons. Synaptic plasticity pertains to changes in the complex synaptic networks of individual brain circuits in response to various interventions. The capacity of the nervous system to reorganize the pattern of synaptic connectivity needs to be understood, because it can potentially bring about changes in the behaviour following neural injury.

The potential to enhance neurological recovery by manipulating the biological adaptability of the brain and spinal cord has become relevant to clinical practice. Medical rehabilitation routines have begun to apply techniques that can build on the neuronal plasticity to optimize the recovery process. In relation to studies of neurorehabilitation, neuroplasticity generally refers to use-dependent neuronal network modifications, which include short-term modulations of functions and long-term structural changes. Knowledge of neuroplasticity is drawn from growing information that ranges from the molecular aspects of developmental neurobiology to functional cerebral imaging in health and disease.

Recent progress in our understanding of neuronal plasticity also confirms that patterns of neuronal connectivity are not rigid. Merzenich and Kaas [1] demonstrated that the sensory maps can be redrawn in adult brains and suggested that the brain is a network that is continually remodeling itself. Their findings brought everybody's attention to the idea that plasticity occurs in the adult nervous system and implied that the adult brain can reorganize itself in areas that were long thought to be completely hardwired. Cortical motor and sensory neurons are not permanently fixed in the way they subserve their limb functions. On the contrary, they quickly adapt to changing demands. In the adult and developing animals and in humans the topographic maps of sensory and motor neuronal representations are capable of physiological and structural reorganisation [1]

Morphological plasticity

A. Dendritic plasticity

Dendritic outgrowth in hippocampal and motor cortical neurons in adult mammals is influenced by various factors such as nutritional status, hormones, aging, ethanol-consumption, brain lesions and injury, social isolation, sensory deprivation, exposure to complex or enriched environment, training for various tasks and electrical stimulation.[2,3,4,5,6] It has been shown that restraint stress for 21 days causes a selective atrophy of apical dendrites of CA3 neurons of hippocampus.[7] As a compensatory mechanism, the density of dendritic spines was increased in these neurons to overcome decreased dendritic arborization.8 Interestingly, the stress-induced atrophy of dendrites was reversed by 45 days of rehabilitation[7]

Somatosensory cortical pyramidal neurons showed an increase of basal dendritic branches in kittens after training for shock-avoidance learning.[9] Greenough et al [10] have reported that training the rats to reach for food, results in increased branching of apical dendrites of layer V pyramidal neurons in the anterior cortical region, which are involved in sensory and motor innervation of the contralateral forelimb of the rat. Similar changes have also been observed in layers II-III pyramidal cell basilar dendrites in rat motor-somatosensory forelimb cortex.[11] Jones and Schallert[12] demonstrated the use-dependent dendritic growth in layer V pyramidal neurons of the motor cortex. In addition to the effect of various environmental factors, the treatment of (-) deprenyl, a selective monoamine oxidase-B inhibitor resulted in a significant increase in the dendritic arborization of CA3 hippocampal and layer III prefrontal cortical neurons in monkey brain [13,14].

Dendritic outgrowth in adults can also occur by electrical stimulation. Rutledge et al[15] observed the additional outgrowth of apical dendrites in the cortex of adult cats after long-term electrical stimulation applied to the left suprasylvian gyrus. After this period, it was observed that apical dendrites of layer II and III pyramidal neurons were shown to have significantly more dendritic branches and spines in the cortex contralateral to the stimulated side. They interpreted this as evidence that increased use of specific pathways to and within the cerebral cortex of adult cats can induce postsynaptic growth.[15] Furthermore, self-stimulation rewarding experience for 10 days resulted in an increase in the dendritic branching and dendritic length in CA3 hippocampal and layer V motor cortical pyramidal neurons[4,5] and such changes were found to be long-lasting.[16]

In addition to the dendritic growth, the size of brain regions is known to be altered significantly by different experiences. For example, Walsh et al[17] observed the medial area of the hippocampus was significantly thicker in animals reared in complex environments than those reared in isolation. Rosenzweig[18] also reported that there was an increase in the size of hippocampus as a consequence of exposure to complex environment. Recent studies conducted in our laboratory have revealed a significant increase in the thickness of lacunosum, radiatum and lucidum laminae in the CA^3 region of hippocampus in self-stimulation experienced rats[4].

B. Spine plasticity

Dendritic spines are tiny, specialised postsynaptic receptive sites that cover the surface of many neurons and they serve as major targets for excitatory synaptic input onto principal neurons in the hippocampus, the neocortex and other brain regions. The density of dendritic spines over any given length is subjected to wide fluctuation depending on variety of environmental factors, electrical stimulation of afferent pathways, sensory deprivation, stress, social isolation, one-trial learning, hibernation, aging, hormones and radiation.[3]

In the CA^1 region of the hippocampus, the dendritic spine density varies by 30% or more, over a 5 day estrus cycle of the adult female rat.[19] Other studies have shown that the shape of spines, in particular the length and diameter of the neck changes during the course of neuronal development,[20] and in response to behavioural or environmental cues such as light, social interaction or exploratory motor activity.[21]

Recent studies involving light and confocal microscopic analysis of spines indicate that excessive synaptic activity leads to the formation of new spines. The increased synaptic transmission is known to produce new spines, for example, enriched environmental stimulation[22] and exposure to chemical stimuli such as N-methyl D-aspartate (NMDA)[23] or even to a single experience in the life of a young chick.[24] Papa and Segal[25] have found an increase in spine density up to 60%, following enhanced spontaneous activity in cultured hippocampal neurons. Alterations in spine density were also observed in various pathological conditions such as brain tumour,[26] alcohol consumption,[27] undernutrition[28] and sleep deprivation.[29] In addition, changes in both spine density and the appearance of abnormal spines have been reported in Down's syndrome and mental retardation.[30,31]

Recent studies have thrown more light on the training induced changes in dendritic spine density in different regions of the brain. Changes in synaptic efficacy may be accomplished by alterations in synaptic density, which could change the strength of input to a particular neuron. Chang and Greenough[32] have shown increases in the number of shaft and spine synapses, 10-15 min after induction of long-term potentiation (LTP), which persist up to 8h. Desmond and Levy[33] have also found a significant increase in the density of spines following LTP in the rat hippocampal dentate gyrus. We have also demonstrated a significant increase in the number of spines in different categories of dendrites in hippocampal and motor cortical neurons.[34,35]

C Synaptic plasticity

The synaptic plasticity denotes long-term changes in synaptic potency resulting from transient changes in synaptic activity. It includes the growth of new synapses or the activation of previously silent synapses, as well as changes in the efficacy of existing synapses.[36] Synaptic plasticity provides the neural basis for learning and memory. The pioneering work of Eric Kandel and his colleagues demonstrated a direct link between synaptic modifiability and behavioural changes.[37]

The nervous system of mammals retains the ability to modify the number, nature and level of activity of its synapses throughout the animals' life span.[38] Synaptic plasticity is maximal during development and is expressed after maturity in response to external or internal perturbations. Numerous examples of synaptic plasticity can be found in the literature.[21] Synaptic density in various regions of the central nervous system (CNS), including neocortex and hippocampus are altered in different conditions such as changes in hormonal levels, undernourishment, aging and Alzhimer's disease, exposure to drugs such as nimodipine, 5-hydroxytryptamine (5-HT) receptor agonist and antagonists, Visual training and cerebral ischemia or lesions (for review see 3, 21).

In addition, alterations in synaptic number, size and shape have also been reported following a number of manipulations, such as stimulation, LTP and NMDA administration.[39,40,41] Similar synaptic changes have also been noted following behavioural manipulations such as learning, imprinting, passive avoidance training, and environmental enrichment.3 Ben-Ari and Repressa[42] reported that brief high frequency stimulation induces LTP and mossy fiber sprouting in the hippocampus and suggested the usage-dependent structural rearrangement of the neural network, a model for plasticity in the adult nervous system. An increase in the population spikes in dentate gyrus induced by

perforant path stimulation has also been observed on the completion of learning.[43, 44] Mitsuno et al[45] have reported the training induced potentiation of population spikes in CA3 neurons of hippocampus occurred with the advance of learning and suggested that LTP in CA3 neurons induced by learning may be related to memory storage. The LTP can set in motion a cascade of events that include changes in gene expression, sprouting of fibers and the establishment of new synaptic contacts.[42]

Our recent studies have also shown an increase in the number of synapses in lucidum, radiatum and moleculare layers of CA3 hippocampal region and molecular layer of the motor cortex following self-stimulation rewarding experience.[46] It has been shown that LTP can cause structural changes by strengthening the existing synapses or resulting in the formation of new synapses. Evidence for changes in the number of synapses following LTP in the hippocampal region was first demonstrated by Lee et al.[41] They showed that LTP induced either in vivo or in vitro led to a rapid increase in the number of synapses.

It is clear from these studies that CNS retains a remarkable ability to modify the number, morphology and activity of synapses. The capability of synapses to alter their structure and number in response to physiological usage may underlie or play a critical role in plastic neurobehavioral processes.

Cytoskeletal plasticity

Experience-dependent structural plasticity is now a well documented phenomenon, however the central question about how functional demand operates at the cellular level to influence the expression of cytoskeletal proteins, namely, neurofilaments, still remains to be answered. Neuronal growth, whether it is dendritic growth or the formation of new synapses are found to be accompanied by changes in the expression of cytoskeletal proteins, which are responsible for structural maintenance of the neurons. Neurofilaments are neuron-specific intermediate filament proteins, which constitute a major component of the neuronal cytoskeleton and play a critical role in determining shape and volume of neuronal processes including complex dendritic arborization and axonal caliber.[47] It has been suggested that the concentration of neurofilaments can serve as indicators for axonal development, neuronal plasticity, neuritic sprouting and nerve fiber regeneration.[48, 49] Few studies have also correlated the expression of neurofilaments and neuronal maturation during development.[50, 51]

Several studies have shown that under normal conditions the neurofilaments in the neuronal perikarya and dendrites are non-phosphorylated while in axons they are

phosphorylated.[52] Altered expression of neurofilaments following kainic acid induced seizures[53] and in other pathological conditions such as Alzheimer's disease and aging[54] has been demonstrated.

Our recent studies have revealed an increased immuno-reactivity for phosphorylated and non-phosphorylated forms of neurofilaments in self-stimulation experienced rats in hippocampal and motor cortical pyramidal neurons.3, [55] The increased dendritic arborization after self-stimulation experience 4,5 was associated with an enhanced expression of neurofilament proteins. These findings suggest a close relationship between the expression of cytoskeletal proteins and experience-dependent structural changes.

Neurochemical plasticity

Neurotransmitters are known to play a critical role in the regulation of neuronal cytoarchitecture. Recent studies have begun to realise that neurotransmitters are not only involved in information coding but may also play important roles in defining the structure of circuits in which they participate.[56]

Various neurotransmitters such as acetylcholine, noradrenaline, dopamine, gamma-amino butyric acid (GABA), glutamate, serotonin, somatostatin and neuropeptides are found to regulate the neuronal structure in the developing and adult nervous system (for review see 56). The importance of noradrenergic innervation during the development of the cortex is suggested by the experimental findings, which show that the ocular dominance shift is abolished when noradrenaline is depleted by 6-hydroxydopamine (6-OHDA).57 Interestingly, the plasticity was restored by microinfusion of noradrenaline into the cortex.[57] The depletion of serotonin in developing rat somatosensory cortex has been shown to delay the barrel formation.[58] Furthermore, the rate of maturation of cortical neurons was dependent on the presence of serotonin[59] and noradrenaline.[60] Monoamines and acetylcholine are involved in the formation of specific dendritic morphology, lamination of cortex or the formation of topographical afferent and efferent projections.[61]

The intracellular concentration of dopamine and serotonin has been shown to vary inversely during the growth of neurons. Several aspects of postnatal striatal development are altered by the neonatal depletion of dopamine by reserpine or destruction of dopaminergic afferents by using 6-OHDA. In addition, dopamine and serotonin inhibit neurite elongation and elevate intracellular calcium when applied directly to neurons in vitro.[62] This may constitute a means of stabilising dendritic growth during synaptogenesis

in vivo. When GABA and its potentiator diazepam were added to growing neurons, the outgrowth of both the axon and dendrites was suppressed.[56] It was observed that somatostatin enhances the neuritic outgrowth in regenerating neurons.[63] Our recent studies have correlated the relationship between increased dopamine metabolism64 and altered dendritic morphology in hippocampal and pre-frontal cortical pyramidal neurons13,14 in adult monkeys following chronic (-) deprenyl administration.

Glutamate is the predominant excitatory neurotransmitter plays a critical role in the morphological changes following LTP. Glutamate was also found to specifically affect the cytoarchitecture of dendrites of hippocampal pyramidal neurons in a graded manner, which suggests that glutamate may be involved in : establishing hippocampal circuitry during the brain development; maintaining and modifying circuitry in the adult and inducing neurodegeneration in several disorders including epilepsy, stroke and Alzheimer's disease.[56]

Acetylcholine is known to enhance the neuritic outgrowth and in tuning the nerve growth cones.[65] Acetylcholine esterase (AChE) can also regulate the neuritic outgrowth and survival of neurons. It has morphogenic and axogenic role in the developing nervous system.[66] Our recent studies have demonstrated an increased AChE activity following (-) deprenyl administration in hippocampus and pre-frontal cortex which was well correlated with an increase in the dendritic arborization in CA3 hippocampal and layer III cortical pyramidal neurons in adult monkeys.13,14

Recently, we have demonstrated alterations in the levels of biogenic amines, amino acids and AChE activity following self-stimulation[67] and this rewarding experience is known to cause structural changes in hippocampal and motor cortical neurons.4,5 These results indicate the role of neurotransmitters in altering the morphology of neurons in adult nervous system. The correlations between neurochemical and structural changes indicate that neurotransmitters are likely to play important roles in bringing changes in the adult brain structure for functional adaptation.

Behavioural Plasticity

The importance of experience for the development of behavior and for the maturation of the central nervous system in normal individuals is widely recognised. Only recently, however, it has become clear that experience may play an equally important though as yet poorly defined role in neuropathological development. A growing number of studies

have provided evidence that recovery of function following brain injury in early life does not necessarily occur spontaneously but rather depends on the opportunities that the environment provides for stimulation during the course of post-operative development. The recognition that environmental stimulation may be a potent factor governing an organism's capacity to compensate for brain injury raises certain hopes for the treatment of brain injury and also poses some basic questions about the mechanisms by which functions are restored in a damaged nervous system.[68]

Landsdell[69] subjected rats to anterior or posterior cortical ablations as infants or as adults and kept them in enriched environments for 3 months to determine if such rearing would offset the expected behavioral deficits. Smith's[70] later investigations have analysed the effects of cortical lesions in rats raised under impoverished conditions as compared with enriched environments. Taken together, the two studies provided support for the idea that enriched rearing conditions could ameliorate behavioral deficits following bilateral cortical lesions performed in weaning rats; later the finding that experience offset brain injuries were extended to rats incurring cortical lesions at birth[71] and to young animals with subcortical lesions.[72,73] Beneficial consequences of environmental complexity have been most clearly shown in studies in which brain-injured rats were raised in enriched environments and then subsequently tested on maze problems.[74]

A number of studies on adult animals, however, pointed to a non-specific function for the post-operative sensory environment. Of special interest are the findings of Harrell et al[75] that rats with hypothalamic lesions recover feeding behaviour more quickly if exposed to daily one hour episodes of low-level electrical stimulation through lesion electrodes. Likewise, there is evidence that recovery of visuo-motor functions can be enhanced by centrally active drugs given postoperatively or between serially imposed lesions.[74]

Recently the neuronal regeneration following early postnatal olfactory tract transection (OTS) was also studied. It was observed that after 240 days of OTS, the olfactory tract had regenerated and the tract fibres had re-established the connections.76 This resulted in a recovery of passive avoidance behaviour after a period of eight months following OTS.[77] Studies from our laboratory have also shown that spinal cord ischemia induced spinal motor neuron degeneration and locomotor deficits can be attenuated by (-) deprenyl.[78,79] In addition, transplantation of embryonic tissues in to lesioned adult hippocampus and substantia nigra resulted in the recovery of functions such as learning and self-stimulation behaviour, respectively.[80] These studies suggest that neuronal

plasticity constitutes a potential mechanism for the recovery of functions after brain injury. Furthermore, we have demonstrated that prior self-stimulation experience facilitated the operant and spatial learning tasks in adult rats.[81] In addition, chronic restraint stress is known to alter the acquisition and retention of spatial memory task in rats.[82]

Thus, it clearly indicates that a regenerative and continuing mechanism for adaptive reorganisation of the brain occurs because of the property of neuronal plasticity. This unique property of the nervous system may be responsible for the recovery of functions in injury as well as in various neurological disorders. However, the means to manipulate the promotors and inhibitors of neuronal plasticity are still in early development. Although single technique for directing cells, their processes, scaffolds in their milieu, their navigational methods and the usefulness of the synapses they make are still only a vision, basic research will provide promising plasticity enhancers for clinical trials. As with the potential mechanisms for restoration of neuromorphology, the functional effectiveness of any biological manipulation will depend in part on the training strategies developed by specialists in rehabilitation to optimally induce activity in neuronal circuits.

"Brain is an 'enchanting loom', the threads of the loom can be broken either by internal perturbations like lesion or by external perturbations like learning, new threads then form, branch out, and give a different pattern- always shifting, but a meaningful pattern."

Sir Charles S Sherrington (1906).

ACKNOWLEDGMENTS: We sincerely thank Dr. Narender K. Dhingra for typing and critical reading of the manuscript.

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Neurological rehabilitation : Concepts and Dynamics

J. R. Chaudhuri , A. B. Taly

Introduction

Rehabilitation is a process of active change by which a disabled person acquires and uses the knowledge and skills necessary for optimum physical, psychological and social functions. Contrary to the popular image of rehabilitation which suggests psychiatry based therapy following trauma, neurological rehabilitation is an active process which seeks to reduce the impact of neurological disabilities on daily life. Neurorehabilitation is essentially the management of patient with neurological disability and requires active collaboration from professionals constituting the rehabilitation team and patients. Neurological disorders account for large proportions of all severe and complex disabilities and thus neurologists are expected to play a pivotal role in coordinating the many processes that constitute rehabilitation[2].

Restorative Neurology

Restorative Neurology is a subsepeciality of neurology which deals with techniques and strategies used to restore a disordered nervous system to a state of optimal function[3]. Following an insult, the disordered nervous system recovers to a variable extent. Functional improvement usually occurs till six months or so and is attributed to various factors like dendritic sprouting, synaptogenesis, restoration of axonal transport, alternation of neurotransmitters, unmasking alternate pathways, etc[4]. When natural recovery slows down, alternate means are sought to restore the functional gains by integrating pharmacological, orthopaedic, neurosurgical and physical treatment modalities.

Proper quantitative evaluation of neurological deficits and useful application of clinical neurophysiology are essential for restorative neurology[5]. Exploiting plasticity of the nervous system, pharmacological modulations, functional surgery, neural grafting and genetic engineering are the exciting frontiers currently being explored in modifying the natural history of the diseases and thus limiting disability[6].

Restorative procedures available are many, though most of them have to stand their usefulness overtime. They include : modification of the input into the spinal cord by

selective lesions of the peripheral nerves or posterior roots, chemically or surgically performed neurectomies and rhizotomies, electrical stimulation of nerves, spinal cord, cerebellum, thalamus and deep brain structures, stereotactive surgical procedures and functional electrical stimulation[7,8]. Achievements in restorative neurology are also rooted in neurobiology. Following quantitative evaluation of impairments and assessing the disability, the relationship between damaged structure and function of the nervous system can be formulated. Modification of structure requires reconstructive neurology (i.e. neurobiological approach) and modification of function may include restorative neurology (i.e. applied neurophysiology)[9]. Replacement of upper limb function, in hemiparesis has been achieved with functional neuromuscular stimulation (FNS) using sensory feedback mechanism from tactile sensory receptors of glabrous skin and designed artificial sensors. Improved neural prosthesis and increasing research in this direction may replace partial motor functions after disease or disability[10].

Neurorehabilitation

This involves combined and coordinated use of medical, social, educational and vocational measures to assist disabled individual in regaining highest possible level of functional abilities.

Needs

Nervous system diseases are among the leading causes of disability[11] and account for 80% of rehabilitation inpatient admissions in United States[12]. In a recent prospective study of 1093 hospitalized neurological and neurosurgical patients evaluated at NIMHANS, Bangalore, majority had disabilities of varying severity at discharge in various spheres of life and at 3-6 months post discharge 25% of these patients had moderate to severe degree of disabilities and required assistance[13]. Neurological and neurosurgical disorders producing disabilities are diverse in nature and require input from several professionals[14].

Wade[15] reported that a population of 2.5 lakhs contained 3500-5000 patients with neurological disabilities in United Kingdom and the figure may not be less in our country. The enormous patient load with neurological disability speaks for the need for neurorehabilitation services. However, there is a wide gap between the burden of disability and availability of satisfactory rehabilitation services. European Federation of Neurological Society task force after a questionnaire based study observed a significant

lack of adequate facilities across Europe. Very few countries have any established network of neurorehabilitation centers with adequately trained physicians, therapists or nurses[16]. In a recently carried out survey during the 4th Annual Conference of the Indian Academy of Neurology at Bangalore, all the participants (n=98) felt that neurorehabilitation services were essential. However, only 28% of them expressed satisfaction with the available services. It was suggested that public and patient awareness on the availability and the benefits of neurorehabilitation services and number of centers with trained personnel for providing comprehensive care needed to be increased[17].

Conceptual framework

The first concern of the neurologist when confronted with patients is to establish the underlying cause by analyzing the symptoms and signs and to seek a pathological diagnosis. The specific treatment plans are sought to halt or reverse the disease process. When medical treatment is partially effective or not available, as it is for many diseases, patients are left with deficits. The World Health Organization (WHO) has put forward a model[18] which divides the consequences of disease into three levels : Impairment. Disability and Handicap. These are additional to the more well known International Classification of Diseases (ICD) which includes etiology and pathology. Impairment refers to the anatomical or physiological loss and really includes most traditional symptoms and signs such as weakness, ataxia or sensory loss. Disability refers to the functional consequences of the impairments. For example, weakness, ataxia and sensory loss can cause inability to run, walk and climb stairs. Handicap refers to the social consequences of the disability and impairment such as loss of a job or inability to do house work, that was once the major role of the patient.

Generally there exists a relationship between the extent of impairment, severity of disability and the level of handicap, but it is not invariable. Handicap can arise directly from an impairment, as well as from a disability. For example, a right hemianopia may cause no disability for a professional driver because it may be fully compensated, yet it may cause major handicap if it leads to loss of driving license ! Loss of a finger may not affect a labourer yet could devastate a musician ! This impairment - disability - handicap model has few drawbacks. It is not easy to separate these three levels in all settings. Aphasia may be an impairment or a disability ! Wade[1] suggested that in aphasia, the language loss is the impairment and poor communication is the disability. This model also fails to incorporate the psychological consequences of the disease (eg. emotional and behavioural) and the effects this may have upon disability and handicap.

Assessment

Assessment refers to the acquisition of information regarding impairment, disability, handicap, quality of life, prognosis and interventions. Impairment is influenced by many factors and is directly proportional to the volume, location and number of lesions and inversely to natural recovery, plasticity and therapeutic efficacy. Impairments result in disability which may be reversible or irreversible and reflects disturbance at the level of the individual. Theoretically, disability depends on impairment(s), functions involved, evolution of diseases and complications, and indirectly on compensation, therapy and motivation. Handicap represents socio economic consequence of disability and is estimated as a product of disabilities and socio economic factors [19].

Evaluation of impairment depends on neurological examination and detection of abnormal signs and symptoms. Presence of a sign may decide the diagnosis, but the intensity of signs acquires importance for prognosis. Thus neurological examination in a quantitative way is crucial. Unfortunately, such measurements are not always possible or reliable, e.g. briskness of muscle stretch reflexes, though a common neurological sign, is not yet scored in a uniform way. Some signs, e.g. muscle power (MRC grade), and spasticity (Ashworth scale) [20] can be quantitated. Whenever possible, direct measurements can be performed and expressed in physical units. Often time required to perform a task e.g. walking 10 meters or climbing 4 stairs can be used to quantify impairment. The real problem in clinical assessment is that only a few methods have been evaluated and recognized. To cope with the problem of interrater reliability, video recording of patients is becoming increasingly popular.

Disability is appreciated by the way a patient performs composite activities and responsibilities, that are generally accepted as essential for every day life. The ICIDH recognized the potential disabilities in the category of behaviour, communication, personal care, locomotion, domestic activities, dexterity and specific situations including bowel and bladder functions. The phrase used to describe the overall level of composite activity in day to day life is referred to as "Activities of Daily Living" (ADL). Several self care related disability scales are available to quantitate disability and to function as an important tool in neurologic rehabilitation studies. Most of these scales include 3,4 or 7 levels for the degree of dependency for each item of disability. Commonly used levels include "independent", "needs assistance" (which again can be minimum, moderate or maximum assist) and "dependent". Discussion pertaining to the details of these scales is exhaustive and beyond the scope of this write up. Barthel Index (BI), Functional

Independence Measure (FIM) and Functional Assessment Measure (FAM) are the most popular, widely used scales and are better standardized than the rest[12]. Interrater reliability for these scales is good. The FIM detects the severity of disability among the patients with neurologic disorder, correlates with the burden of care required and contributes usefully to responsiveness[21].

Handicap related to neurologic disease has received less attention than impairments and disabilities. The World Health Organization Handicap Scale uses eight grades to describe the difference between individual's performance or status and what that person expects of himself or herself or of those in a similar situation. The domains assessed are orientation and interaction with surroundings, physical independence in ADL, mobility, occupation, social integration and economic self sufficiency. The reliability of this scale needs validation[12].

Quality of life (QOL) measures use the patient's perspective to assess domains that include physical, mental, social and general health. These measures are meant to evaluate the overall impact of a disease and its treatment. Components of QOL domains include physical health consisting of mobility, ADL, pain, impairments and disability, mental health including emotional and cognitive functioning, social well being and general health which includes medical symptoms, fatigue, sleep difficulty, life satisfaction, health perceptions and distresses and overall quality of life.

The development of clinically relevant measures of complex, integrated functional performance has been a great challenge for rehabilitation specialist. Neurological rehabilitation team should thrive to know whether what they do for the patients is worth or not.

Practice

Rehabilitation probably starts as soon as the patient is admitted to the hospital. Focus of medical attention is on reducing impairment and preventing complications. Rehabilitation measures include passive range of movements at all joints, use of splints to prevent early contractures, frequent change of postures to prevent pressure sores and respiratory physiotherapy to facilitate drainage of secretion. Prophylaxis for deep venous thrombosis may reduce incidence of embolism. All these measures aim to prevent further complications which may aggravate disability and require prolong hospitalization. After stabilization in the "acute" care, the patient is usually referred to

the "subacute" set up. The patient is evaluated in the neurorehabilitation units, improvements are quantified, disabilities are established, goals are defined and programme for rehabilitation is outlined. Relatives and care givers are interviewed, their expectations and problems are assessed and primary counselling is done. Many patients make significant recovery and are thus discharged from neurological wards and home programmes are encouraged when required. Patients with significant disability are transferred to neurorehabilitation ward for inpatient care. Aims of rehabilitation during this stage are to promote intrinsic recovery, teach adaptive strategies, facilitate interaction with surroundings and encourage reentry in community This period is devoted to reassessment, planning of short term and long term goals, scheduling programmes involving physiotherapy, occupational therapy evaluation of bowel and bladder functioning among others and intervention. Symptomatic medications and modification of treatment are also done during this phase. Weekly assessments are done by the team, goals are modified, programmes are changed if required. Late phase of rehabilitation is most crucial and focusses on community reintegration[22,23].

Team

The rehabilitation process involves "team effort". In neurological rehabilitation, the team is composed of a physician, nurse, physical therapist, occupational therapist, speech therapist, recreational therapist, neuropsychologist, dietitian, orthotist and social worker. Each member of the team has specific goal but all of them work in the same direction ie. for optimizing functional recovery.

Physician : The primary role of the neurologist is to use experience and knowledge regarding neurologic recovery to help and direct the rehabilitation team. Physician is also involved with the medical issues relating to underlying disease, DVT prophylaxis, bladder evaluation and care, bowel care, treatment of infections, sleep disorders, depression and others. Physician helps to predict and prevent impending complications and provides information about prognosis. Neurologist is likely to review the original diagnosis regularly and intervene as situation demands. Drug requirements are seldom static and modern advances in investigations and therapeutics can radically alter prognosis.

Rehabilitation Nurse : Patients admitted to rehabilitation wards often have contractures, spasticity, pressure sores, urinary catheter, irregular bowel function, nasogastric feeding tubes and varying treatment schedules. All of them require utmost nursing care. The

nurse's role is to oversee the rehabilitation programme for an individual patient from beginning to disposal. Nurse is responsible for delivering the medical and surgical treatment and acts as a liaison between the team members and patient's family. The nurse is integral in discharge planning, working with social workers and family. She ensures proper skin care, supervises bladder and bowel programmes and encourages patients to practice the skills learned in therapy sessions in the ward and daily life. Rehabilitation nursing focuses on health and well being and the importance of self care, independence and safety [23, 24].

Physical Therapist : Over 80% of patients in rehabilitation practice have problems regarding mobility and transfers. Physical therapist is primarily involved with gross motor control, strengthening of muscles, improving endurance and in optimizing mobility skills. The therapist evaluates sitting and standing balance, transfers, walking and wheel chair propulsion. Physiotherapist initiates the gait training and when applicable suggests assistive devices for mobility. Families are integrated into the rehabilitation process at the therapy level to work with the patients on these skills in anticipation of their returning home. They give advise regarding structural modifications (e.g. ramp in place of stair, fixing grab bars in toilet, widening of doors, etc.) in home. Optimal management of neurological disability with physical therapy differs from the more common field of orthopaedic trauma. Ultrasound and short wave, hydrotherapy, accupunture, laser therapy, transcutaneous nerve stimulation and functional electrical stimulation are also used by the physiotherapists.

Occupational Therapist : Activities of daily living are variably affected in disabled patients. They may have normal power in upper limbs but do badly in self care activities owing to their poor truncal balance, incoordination, cognitive problems, motivation or "dependency" on care givers. Occupational therapist works primarily on improving fine motor skills, upper extremity range of motion and strength, truncal balance and activities of daily living such as bathing, dressing, feeding, grooming and toilet use. In suitable patients higher level skills such as cooking, house making may also be addressed. Occupational therapist also assesses upper limb functions and advises regarding splinting or orthosis when required. They also make home visits and recommended environmental changes to suite disabled individuals.

Speech Therapist : Language dysfunction has a wide spectrum and is a major disability among stroke victims and to a certain extent in head injured individuals. This impairs communication and causes frustration. Many patients also have dysarthria, swallowing

difficulty, orolingual apraxia and nasogastric tube. The primary role of speech therapist is to evaluate and treat disorders of language, communication and swallowing. When aphasia or dysarthria are present, attempts are made to establish reliable communication system. Bed side swallowing test and video fluoroscopy are carried out to evaluate dysphagia. Patients are then taught compensatory swallowing techniques to minimize the risk of aspiration.

Clinical Psychologist : Cognitive impairments, behavioural problems such as depression, agitation, apathy and violence are common in patients with head injury, stroke, multiple sclerosis and epilepsy. These factors along with amotivation interfere with therapy and evaluation. Clinical psychologist along with speech therapist evaluates patients cognitive functions, assesses them with specific neuropsychological tests and plays an important part in cognitive retraining. They help the family to adjust emotionally to chronic disability and play a crucial role in community reintegration and vocational counselling.

Social Worker : Disability and consequent handicap try to drift the patient from main stream of the society. Rehabilitation ultimately aims to integrate the disabled patient back into the family and society. Social worker plays key role in identifying the community resources available to patient and family. This may involve arrangement of funds for orthotic and mobility devices, outpatient visits and home therapy services by various therapists. They help the patient to overcome handicap by discussing about vocational training and providing financial help. They are also expected to look-after medicolegal and insurance matters of the patient.

Recreational Therapist : Recreation is essential for total well being of healthy and disabled individuals alike. It helps the patient to decrease frustrations and boredom and binds the disabled to the family. Recreational therapist tries to organize and generate activities in wards like singing, playing indoor games, enacting a drama and engaging them in arts and crafts activities etc. Recreational therapist helps to reintegrate the patient into the community through a programme of community outings e.g. shopping, dining and interacting with others. Socialization is thus encouraged because outings are typically done in groups.

Dietitian : Inadequate nutrition, hypoproteinemia and obesity are common among the patients with disabilities and proper nutrition is essential for recovery. Many of them are unable to consume normal food due to dysphagia. Dietitian supervises the dietary

schedules, prescribes appropriate diets as required and trains the care givers. The dietitian works with speech therapist and occupational therapist in arranging an appropriate diet based on the patient's swallowing and self feeding abilities.

Orthotist : Many patients benefit from orthotic devices for mobility and self care, while waiting for natural recovery, e.g. cane for hemiplegics, ankle foot orthoses for foot drop, walkers for paraplegics etc. The orthotist's role in rehabilitation programme involves working with physical and occupational therapist to evaluate patients for upper and lower extremity orthosis. If needed, orthosis are custom fit to meet the patient's functional requirements. Orthotic devices provide comfort and safety to patients and help to prevent deformities.

Other professionals may also be consulted by the rehabilitation team when required. Ophthalmologist who may help in the treatment of coexisting eye problems of the disabled like cataract, diabetic retinopathy, tarsorrhaphy for facial palsy and surgical correction of ptosis. Audiologist helps in assessment of hearing impairment and provide hearing aids and along with speech therapists may impart communication training. Plastic surgeons may help in pressure sore care and urologists may guide for bladder management. Vocational counselors, biomedical engineers and biomedical statisticians are also consulted from time to time when required [12].

Dynamics

Neurological disabilities vary widely between patients and hence services need to be individualized. Patients with spinal lesions may have significant mobility and sphincter disturbances while cognitive and communication spheres are normal. Hence focus in such patients is on locomotor rehabilitation [26] and urodynamic assessment and appropriate bowel and bladder management programmes, while psychological counselling may be enough for his secondary depression if any. On the other hand head injury patients may require series of cognitive retraining sessions while most patients may not require locomotor training. Table I shows differential needs of the patients in various disability spheres.

Comprehensive inpatient rehabilitation is rather expensive and therefore optimally utilized. Patients admitted to the rehabilitation service must be able to tolerate at least three hours of therapy a day and have rehabilitation needs in at least two of the following areas : nursing, physical therapy, occupational therapy, or speech therapy [12]. The

Table I : Differential needs

Disabilities	Services	Stroke	Head Injury	Spinal cord injury	Neuromuscular disorders
Mobility	Physiotherapy	+ +	+	+ + +	+ + +
ADL	Occupational therapy	+ + +	+ +	+ +	+
Sphincter	Nursing	+	+	+ + +	-
Cognition	Psychologist	+ +	+ + +	-	+
Communication	Speech therapy	++ +	+ +	-	+
Psychosocial	Social counsellor	+ +	+ + +	+	+

members of the rehabilitation team set both short and long term rehabilitation goals for each patient within their discipline. An individualized rehabilitation programme is designed to meet these goals and discharge date is set accordingly. Following linear approach traditionally, several disciplines work on the same patient, butfrom within their own professional disciplines and working practice. The team meets weekly (Team rounds) and discusses about individual patient. Goals are revised as the patient progresses through the programme. The overlapping approach is encouraged because most patients require them. Unnecessary duplications and/or omissions in treatment are discussed and inter professional conflicts are resolved, wherever possible.

Problems

Problems faced in Neurological rehabilitation arise from two levels - the individual patient and underlying organization of services, and medical complications seen during neurological rehabilitation. Wide variety of neurological diseases and variable disabilities need high level of care. This is compounded with enormous load of disabling neurological diseases in the community. Range of deficits vary, may be system specific, local or diffuse, and may affect central and peripheral nervous system [13, 14]. Family and society adds up and influence the decisions variably [27, 28].

Organization of neurological rehabilitation involves large number of people. The team members come from various disciplines[29,30]. Often integration of their services may retard the individual's growth. The conflicts, though normal and necessary for team development frequently impair team functioning when they come repeatedly with no resolution. Complacency sets in and it may be detrimental. Due to progressive natural history of many degenerating neurological diseases dissatisfaction builds up among the team members and create frustration. Medical problems in neurorehabilitation are common and under-recognized. Frequency varies according to patient's profile, disability, length of stay, disease severity and age. Medical complications contribute significantly to the cost of treatment, morbidity, loss of therapy time, transfer to acute care set up and death.

Recommended standards

Recently task force on standards in neurological rehabilitation established European federation of (EFNS) scientific panel recommended minimum standards for the prevention of neurological disability including access to health education, genetic counselling and emergency resources. It has outlined some minimum standards for the staffing of a neurological rehabilitation set up including training for the neurologist, physician and other team members. The task force supported a two tier system of neurorehabilitation services. The local community service should refer complicated and significantly disabled brain or spinal cord injured patient to the regional specialist service centres. The regional centre would provide multidisciplinary expertise, wheel chairs, urological services, aids and equipments including community aids, problems etc.The task force recognized the limited resources available for neurorehabilitation and the need for more funds for rehabilitation research. It realized the poorly developed rehabilitation services in Europe and developing countries and endorsed international co-operation [16].

Conclusion

Neurological deficits lead to handicap. Spontaneous recovery, medical rehabilitation and learning to live with the handicap are key issues in rehabilitation. This coupled with restorative techniques and reconstruction strategies may complete neurological rehabilitation. It is easy to manage neurologically disabled patients badly and probably impossible to do it perfectly. The difficulty arises from variety of diseases and disability, need for multifactorial approach and paucity of research. Access to adequate resources

and timely, accurate and relevant information are important for effective management of neurological disability.

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Quantification in neurological rehabilitation

S. Ramar

Introduction

Assessment and quantification of neurological deficits and their consequences are an important aspect of rehabilitation. The quantification is essential for monitoring the progress of the patient, programme evaluation, quality assurance and improvement of services. The scales used in measurement should be simple, sensitive, valid, reliable and accurate. International classification of impairment, disability, handicap was developed to classify the consequences of the diseases such as the disruption of daily activity and social disadvantages that accompany illness [1].

Impairment

It is any loss or abnormality of psychological, physiological or anatomical structure or function. As a result of impairment, there is functional limitation resulting in partial (or) total inability to perform motor, sensory and mental functions within a range and manner of which a human being is normally capable. Impairment is divided into: (1) Physical (2) Intellectual and psychological and (3) Generalised and other. Physical Impairment refers to impairment of physical function or structure. It is divided in to aural impairment in relation to auditory sensitivity, language impairment in relation to language function and speech, ocular impairment in relation to visual acuity, visceral impairment in relation to bladder, bowel, sexual and mastication & swallowing functions, skeletal impairment in relation to head & trunk regions, mechanical & motor impairments of limbs and deficiencies of limbs and disfiguring impairment in relation to head, trunk and limbs. Intellectual and Psychological impairment refers to disturbance in function in relation to intelligence, memory, thinking, consciousness and wakefulness, perception and attention, emotive and volitional functions and behaviour.

Quantification of impairment

The neurological impairment is related to the site and size of the lesion. Some common measures used to quantify the different areas of neurological impairments are as follows[2]

Consciousness : Glasgow coma scale, Galveston orientation and amnesia test
Cognition : Minimental status examination
Memory : Wechsler memory scale, Benton visual retention test and Rivermead behavioural memory test
Attention : trial making test, visual cancellation test, Wechsler digit span.
Language : Western aphasia battery
Sensorymotor:Medical research council grading (table 1), modified Ashworth grade for spasticity (table 2) and Motricity index.
Gait : Dynamic electromyography, kinematics and kinetics.
Balance : Static posturography and dynamic posturography.

There are scales devised for evaluation of impairments in specific disorders. They include National Institute of Health stroke scale, Expanded Disability Status Scale for multiple sclerosis, United Parkinson's disease rating scale, Rancho Los Amigos level of cognitive

Table 1 MRC grading of muscle power.

	Description of Power
Grade 0	No tension is palpated in the muscle or tendon on maximum voluntary effort.
Grade 1	Tension is palpated in the muscle (or) tendon but no motion occurs at the joint on voluntary effort
Grade 2	The part moves a gravity eliminated plane with no added resistance, on maximum voluntary effort
Grade 3	The part moves against gravity on maximum voluntary effort
Grade 4	The part moves through full range of motion against gravity as well as against less than moderate resistance on maximum voluntary effort.
Grade 5	The part moves through full range of motion against gravity and maximum resistance on repetitive attempt.

Table 2 Modified Ashworth Scale

Grade	Description
0	No increase in muscle tone
1	Slight increase in muscle tone manifested by a slight catch and affected part(s) is moved in release or by minimal resistance at the end of ROM when the affected part(s) is moved in flexion or extension
2	Slight increase in muscle tone manifested by a catch followed by minimal resistance throughout the remainder (less than half)of ROM
3	Marked resistance in muscle tone through most of the ROM but affected part(s) easily moved
4	Considerable increase in muscle tone and passive movements is difficult
5	Affected part(s) is rigid in flexion or extension

function for head injury and American Spinal Injury Association neurological and functional classification for spinal cord injury. [2].

Henry Kessler's Method

This method emphasizes that the function alone represents the true measure of one's ability or disability. Thus, this method employs evaluation of functions of the upper & lower extremities, spine and activities of daily living. The functions of upper limb include, motion, strength and coordination for the arm component and prehension and sensation for the hand component. Each function is given a score of 100% impairment. The functional losses of each function of the arm component are telescopically combined by the formula A+ B(100 - A)/100. Similarly the functional loss of hand component is also arrived using this formula. Finally the functional loss of whole upper limb is arrived at by combining the arm component and hand component using the same formula.

Evaluation of permanent physical impairment based on the uniform definitions notified by Government of India

Though many methods of disability evaluation have evolved, universally acceptable standard system is yet to be developed. The disability evaluation was based mainly on methods of individual interest. Invariably an arbitrary score has been given based on personal assessment of the certifying physician / surgeon. Some times different values were given for the same patient with the same locomotor disability on different occasions. Government of India has notified uniform definitions for evaluation of permanent physical impairment. Evaluation should be done when the physical condition is stationery after maximum recovery at the completion of treatment.

Upper Limb

The permanent physical impairment in relation to upper limb is divided into arm component and hand component. Arm component included active range of movement, power and coordinated activities.

Active range of movement : The active range of movement is evaluated at shoulder, elbow and wrist. The movements examined at shoulder include flexion and extension(0-220 0), abduction and adduction (0-180 0) and rotation(0-180 0). Flexion and extension (0-150 0) and pronation and supination (0-180 0) are evaluated at elbow. The movements measured at wrist joints are palmar and dorsiflexion (0-160 0)and radial and ulnar deviation (0-55 0). The percentage of loss of movement is calculated at each joint and is multiplied by 0.3.The allotted maximum a score for each joint is 30. The score for the shoulder, elbow and wrist joint is summed up to obtain a score for the arm component.

Muscle Power : The percentage of loss of power is calculated at shoulder, elbow, and wrist and is multiplied by 0.3.The allotted maximum a score for each joint is 30. The score for the shoulder, elbow and wrist joint is summed up to obtain the score for the arm component.

Coordinated activities : The coordination is measured on a ten-item scale. Each component has nine points.The activities evaluated are: lifting overhead objects, removing and placing at the same place, touching nose with end of extremity, eating Indian style, combing and platting, ablution (Indian style), putting on shirt, drinking a glass of water, buttoning, tie nara/ dhoti and writing.

The percentage of loss of a score for active range of movement, power and coordinated activities for the arm components are combined by using telescopic formula A+B (90-A)/90.

The Hand component is divided into prehensile function, sensory function and strength. The percentage of loss towards prehensile function, sensory function and power of hand is added directly to reach the percentage of loss for hand component.

The percentage of loss of arm component and hand component is combined by telescopic formula A+ B(90 - A)/90.To this score 4% is added if dominant hand is involved. In case of deformity, malalignment, abnormal mobility, infection and changes in cosmetic appearance disability increases by additional 10%.

Lower Limb

Evaluation of permanent physical impairment in relation to lower limb is divided into mobility component (range of movement and muscle power) and stability component.

Mobility component: The active ranges of movements at hip, knee, ankle and foot are measured. The movements examined at hip include flexion - extension ($0\text{-}140^0$), abduction adduction($0\text{-}90^0$) and rotation ($0\text{-}90^0$). The flexion and extension are measured at knee($0 - 125^0$). At ankle and foot, plantar and dorsiflexion and inversion ($0\text{-}70^0$) and eversion is checked ($0\text{-}60^0$). The percentage of loss of movement is calculated for each joint and is multiplied by 0.3. The maximum score for each joint is 30. The score for the hip, knee, ankle & foot is summed up to obtain a value for mobility loss.

Muscle Power : The muscle power is examined at hip, knee and ankle and foot. The muscles examined at hip are flexors,extensors,abductors,adductors and rotators. The percentage of loss of power is calculated to each joint and is multiplied by 0.3. The allotted maximum score for joint is 30. The score for the hip, knee, and foot is summed up to obtain muscle score. Both the percentage of loss for active range of movement and muscle power are combined by a telescopic formula to reach the score for the mobility component.

Stability : The stability is assessed using a nine-item scale. The activities evaluated are: inability to walk on plain surface. inability to walk on a slope, inability to climb, inability

to stand on both legs, inability to stand on affected leg, inability to squat, inability to sit cross legged, inability to kneel and inability to turn

The percentage score for the mobility and stability components are combined by using the telescopic formula A + B(90-A)/90 to reach the percentage of permanent physical impairment for lower limb .To this 10% is added for infection, deformity and loss of sensation.

Evaluation of Upper motor neuron lesion

In evaluation of UMN lesions such as monoparesis, monoplegia, hemiparesis, hemiplegia, paraparesis, paraplegia, quadriparesis and quadriplegia, the following percentage of physical impairment is allotted
Monoparesis -25%, Monoplegia - 50%, Hemiparesis - 50%, Paraparesis- 75%, Paraplegia -100%,Quadriparesis-100% and quadriplegia -100%.

In evaluation of sensory system, impairment rate of 10% for anaesthesia, hypaesthesia, paraesthesia for each limb and impairment rate of 25% assigned for involvement of hand /hands, foot/feet.

In evaluation of speech, the individual is tested by a 100 word text, ability to read, comprehension when readout, answer question on text clearly and ability to write a synopsis. Impairment rate of 25% for mild, 50% for moderate, 75% for severe and 100% for very severe impairment is assigned.

Anomalies

The percentage of impairment for a patient with postpolio residual paralysis involving one lower limb is 90%. But the percentage of impairment for both lower limbs following postpolio residual paralysis is also 90%, and for muscular dystrophy involving both upper and lower limbs with difficulty to get up from squatting position is only 85.05% ! Thus uniform definitions notified by Government of India is endowed with anomalies. Further, this system has the disadvantage of ceiling the maximum impairment to 100%. If the maximum physical impairment is allowed to exceed 100% it will represent the true magnitude of the resultant disability such as attendant care for mobility and ADL especially in multiple handicaps.

Disability

WHO defines disability in the context of health experience as any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner (or) within the range considered normal for a human being. WHO classified disability in relation to physical functioning, social functioning and other.
Disability in relation to physical functioning is divided into several subcategories:
Locomotor disability : It refers to movement disability
Communication disability : It concerns with speaking and listening
Personal care disability : It refers to personal hygiene, dressing, feeding and excretion.
Body disposition disability : It refers to domestic disabilities viz., preparing & serving food, care of dependents and disabilities of body movement viz. gripping and holding.
Dexterity disability : It refers to disability in relation to bodily movement including manipulative skills and ability to regulate control mechanism.
Behaviour disability It refers to disabilities in reaction and awareness
Situational disability : It refers to dependence and endurance and environmental disability in relation to tolerance of environmental factors.
Other disabilities : It refers to disabilities of particular skills and other activity restriction.

Based on the progression of the disease the disability also can be divided into:
a. Temporary total disability:-It is the period during which the individual is totally unable to work.
b. Temporary partial disability:-It is the period during which the recovery has reached a stage to begin a gainful employment.
c. Permanent disability:-It is period after the stage of maximum improvement from any medical treatment and the condition is stationary.

Quantification of Disability

There are several scales that measure disability. The scales are devised for measuring disability either in specific patient groups or in all clients. Some of the commonly used scales are Barthel index, Functional independence measure, Rivermead Activity of Daily living index ,Ranking disability scale, Glasgow outcome scale and disability rating scale.

Functional independence measure

The FIM was introduced in 1986 by the task force to develop a uniform data system for medical rehabilitation[3]. This is an 18 item ,seven level ordinal scale.The seven levels

of functioning are shown in table 3. The activities scored are:

Self care : Eating, grooming ,bathing, upper body dressing ,lower body dressing and toileting.
Sphincter control : Bladder management and bowel management.
Mobility : Bed, chair and wheel chair transfer ,toilet transfer, tub/shower transfer
Locomotion : Walking/wheelchair over level ground, stairs
Comminication : Comprehension and expression
Social cognition : Social interaction , problem solving and memory

The lowest score is 18 and represents total dependence . The highest score is 126 and represents independent ,normal and safe functioning .

Handicap

It is defined as a disadvantage for a given individual resulting from an impairment or a disability that limits or prevents fulfilment of a role that is normal depending on age, sex, social and cultural factors for that individual . Considering a case of post polio residual paralysis involving both lower limbs, chromatolysis of the anterior horn cell due to polio virus constitutes the lesion; loss of muscle power and movement constitutes the impairment; inability to stand, walk and climb constitutes the disability and inability to attend to the school / work constitutes the handicap . Measurement of handicap in neurological diseases has received a lower priority than impairment and disability . The WHO handicap scale has eight graded categories . They describe the differences between an individual's performance and expectations . The domains assessed are orientation and interaction with surroundings, independence in ADL, mobility , occupation , social integration and economic self sufficiency . The reliability is still not clear.

Quality of Life (QOL)

The QOL measures physical , mental , social and general health using patient's perceptions. The health related QOL measures currently in use include sickness impact profile, Functional status questionnaire, Health status questionnaire, Nottingham Health profile and quality of wellbeing scale. There are several scales for measuring social adjustment, support, mood and other domains of QOL. The reliability and sensitivity of these scales need to be established [3].

Table 3 Functional Independence measure: Levels of Function

Independent : Another person not required for the activity (No helper)	
7	Complete independence - All taskes described are performed safely without modifications, assistive devices or aids and within a reasonable time.
6	Modified independence-The activity involves any one or more of the following: An assistive device is required, more than a reasonable time is needed, or safety considerations exist.
Dependent: Another person is required for either supervision or physical assistance for the activity to be performed, or its is not performed (requires helper)	
Modified dependence: Patient expends 50% or more of the effort: The levels of assistance required are the following:	
5	*Supervision or set up* - The patient requires no more help than stand assistance, cueing or coaxing, without physical contact or the helper sets up the items or applies orthosis
4	*Minimal contact assistance* - With physical contact patient requires no more help than touching and expends 75% or more of the effort
3	*Moderate assistance* - The patient requires no more help than touching or expends 50% or more upto 75% of the effort.
Complete dependence : Patient expends less than 50% of the effort: maximal or total assistance is required, or the activity is not performed: levels of assistance required are the following	
2	*Maximal assistance* - The patient expends less than 50% of the effort, but at least 25%
1	*Total assistance* - The patient expends less than 25% of the effort

Conclusions

Quantification is an essential component of the Neurological rehabilitation programme. This is required for accurate assessment of diseases and their consequences, evaluation of interventions, communication between different team members and different rehabilitation teams, legal procedures for compensation and availing social benefits for disabled. Several accurate and valid scales are available for measuring impairment, disability and handicap. However there is no single scale suitable for all patients and all situations..The selection of the scale depends on the nature and etiology of the disability ,purpose of quantification and familiarity of the investigator with the scale. The quantification methods in Neurological rehabilitation need to be constantly evaluated and refined.

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Neurophysiological methods in Restorative Neurology

A. B. Taly

Introduction

Rehabilitation in the context of neurological disorders can be viewed from two angles : (1) Neurological Rehabilitation which involves combined and coordinated use of medical, social, educational and vocational measures to assist disabled individuals in regaining highest possible level of functional abilities and (2) Restorative Neurology wherein one plans active procedures to improve the function of the impaired nervous system through selective structural or functional modification of abnormal neural control according to the underlying mechanisms and clinically unrecognized residual function.

Clinical electrophysiology is the best means for objective evaluation of the functional integrity of the nervous system and therefore it has tremendous potential in the practice of Restorative Neurology. A wide variety of neurophysiological tests applicable to different parts of the nervous system are now available. Some of these are qualitative while others are quantitative. Majority of them are routinely practiced in most clinical Neurophysiological laboratories.[1,2] These techniques can be used to establish the diagnosis, localize the precise site of lesion, understand the mechanism of impaired function, quantitate the deficit responsible for disability, record the residual function, plan the appropriate therapies and monitor the effect of various therapeutic interventions.(Table1,2). These neurophysiological techniques are also applicable to Restorative Neurology [3-5]. Most patients are referred to rehabilitation after complete clinical and electrophysiological evaluation and therefore routine the performed tests such as electromyography (EMG), nerve conduction and evoked potential studies are not described in this chapter. What follows is a brief account of some of the neurophysiological methods used for evaluation and management of common problems in neurological rehabilitation.

Table 1: Electrophysiological tests - Central Nervous System

Neuraxis	Test
Cerebral cortex	EEG and Quantitative EEG Functional brain imaging Cortical stimulation Sensory evoked potentials Long loop reflexes (LLR) Bereitschafts potential Movement related potential
Brain stem	Auditory evoked potential Blink reflex Electro nystagmography
Spinal Cord	
Descending tracts	Cortical and spinal stimulation Central EMG LLR Audiospinal facilitation
Ascending tracts	Somatosensory evoked potential (SSEP) LLR
Motor neurones	M, F and H study Electromyography
Inter neurones	Vibration inhibition "H" reflex recovery curve Silent period
Others	Sympathetic skin response (SSR)

Table 2 Electrophysiological test - Peripheral Nervous System

Neuraxis	Test
Nerve roots	
Anterior Posterior	F' wave H reflex SSEP
Peripheral nerves	Motor conduction Sensory conduction F, H and SSEP Sympathetic skin response Refractory period Microneurography
Myoneural function	Repetitive nerve stimulation Single fibre EMG Stapedius reflex
Muscle	EMG - Concentric needle Single fibre Macro Scanning Acoustic Quantitative EMG Muscle conduction

Kinesiologic elecromyography

The technique of kinesiologic EMG (KEMG) is a window through which muscle can be seen alive and in action. It enables one to assess the pattern of muscle response, onset and cessation of activity, involvement of different muscles in complex and coordinated movements and level of muscle response in relation to effort, type of muscle contraction and position of joint. Recording of conventional EMG alone does not give

information concerning the strength and type of contraction. Force transducers, goniometers, photographic precision, accelerometers and microswitches is needed for simultaneous documentation of other data. Instrumentation for KEMG includes surface or wire electrodes, multichannel amplification and recording system for simultaneous muscle and other physiologic data[2,6]. Surface electrodes are more commonly used. Skin is abraded and cleaned to maximize signal transfer and electrodes are placed using coupling electrode gel, close together on the muscle to be studied and parallel to the direction of muscle fibers. When small, deep or weak muscles need study, flexible wire electrodes are used. They are also used as a preliminary to research when surface electrodes have to be applied. Using sterile technique the wire electrodes are inserted through a hypodermic needle. When the electrodes are appropriately fixed the subject can move freely without discomfort.

During conventional EMG a raw signal is displayed for visual analysis. However, in KEMG overall activity of muscle during specific action or task is important and therefore EMG signal needs special processing by microcomputers. Through a process called "rectification" both the positive and negative components of the signal appear above the base line. Another type of circuit provides "integration" of EMG signal through accumulation of energy on a capacitor. Integrated EMG results from summation of the area under the curve and can be expressed in units. The signal can also be analyzed for spectral frequency and displayed in various forms. Because of the variability of the EMG procedure and anatomy and movements characteristics of muscle it is important to "normalize" the data for validation of comparison. EMG is initially recorded at maximum voluntary contraction or at defined submaximal level of contraction. All subsequent values are then expressed as percentage of this "control" value.[2] Due consideration should be given to instrumentation, equipment and supplies required and physical set up of the study. Subject's comfort and safety should be ensured. Recording techniques should be standardized by repeated trials. Raw data should always be stored for review before rectification, integration or alteration by computers. This will prevent wrong interpretation of artifacts caused by motion, cable artefact, poor electrode contact, wire sway, electrode failure or extraneous electrical signal.

When multichannel KEMG data are recorded with electrogoniometer, pressure transducer, microswitches, accelerometers and other equipments it has several applications in rehabilitation eg. determining pattern or sequence of EMG activity in normal and abnormal motion, documentation of altered muscle function in spastic or weak muscles, evaluation of gait,measurement of strength, endurance and fatigue,

selection of most efficient exercise therapy or biofeedback training, establishment of the effectiveness of orthotic/prosthetic devices, drugs, restorative strategies and other therapeutic measures and conducting research in rehabilitation [2,6].

Muscle strength testing

Quantification of neuromuscular function through measurement of muscle strength is necessary for maintaining uniformity of data collection, understanding pathophysiology of disease, proper comparison of various studies and monitoring natural history of the disease and effect of therapeutic exercises, drugs and rehabilitation measures. Most clinical studies include manual muscle testing, recording of range of movement at different joints, documentation of activities of daily living and timed motor function measures. While these data provide adequate information for clinical use, they do not give objective measure of muscle strength. Electromyographic techniques such as single fiber EMG, concentric needle EMG and macro EMG are useful means of assessing only the function of individual motor units and total electrical strength of muscle. Quantitation of muscle strength thus requires mechanical devices.[7] Many instruments are now available which can measure muscle strength during isometric (force at constant length) and isokinetic (force at constant velocity) muscle contractions. An ideal equipment should be economic, simple to use, adopted for patients with different disabilities and not time consuming. It should measure maximal and sub maximal strength with precision and have test - retest reliability. Regular calibration of equipment is necessary for obtaining correct values. Some of the commonly used equipments are spring balance, cable tensiometer, hand held dynamometer, myometer and newer dynamometers such as Cybex, Lido and Kin-com. Each of these systems have certain advantages and limitations and attempts are being made to improve these techniques.[8,9] For accurate measurements standardized body positions are described. Patient should be comfortable and have adequate fixation of body segments. Patient needs to understand the technique and should be encouraged throughout the procedure so as to improve motivation. Test trials are usually necessary before the measurements. Due consideration should be given to factors like age, gender, anthropometric data and physiological and psychological status of the patient.

Quantitation of muscle strength has become an integral part of rehabilitation and research in the field of disorders of motor neurone, peripheral nerve and muscle. However, it is important to have adequate knowledge of the equipment and the system used, technical standards and biological variables for proper interpretation of the results.[7,9]

Evaluation of fatique

Fatigue is an important limiting factor in the rehabilitation. It is defined as a failure of muscle to produce or maintain initial peak force or torque. It could be due to failure at one or more sites between the motor cortex and the muscle fibers, eg. there may be failure of descending voluntary drive to motor neurone pool resulting in "central fatigue", abnormality of excitation contraction coupling in muscle fibers, inhibitions from periphery or inefficiency of contractile apparatus. The traditional method of testing fatigue is an incremental exercise using bicycle ergometry or treadmill. Clinical electrophysiological techniques can help in documentation and understanding of fatigue phenomenon.

It has been elegantly demonstrated that during increasing muscle contraction there is an orderly recruitment of motor units, based on their size. Initially there is contraction of low threshold, "fatigue resistant", slow twitch fibers with a prominent oxidative capacity and later higher threshold units with fast twitch are involved when more force is required.10,11 During sustained or repeated submaximal isometric contraction, contrary to the expectation, despite fatigue there occurs an increase in integrated EMG amplitude. In an attempt to maintain the same force more number of units are recruited at increasing frequency. This depends upon the central drive and is associated with a sense of increasing effort. Similarly during maximal effort, it is observed that the force declines over a period of time but EMG activity may remain constant. However, eventually due to failure of contractile machinery, both the force and EMG activity decrease. Fatiguability of muscle varies based on its composition and researchers have noted linear as well as non linear relationship between EMG activity and force. It is however, important to realize that decrease in voluntary effort may be due to pain, restriction of range of movement and motivation (psychological).12 Therefore, it is important to ensure that all motor units are recruited before making a remark on fatigue. This can be ascertained by interpolated stimulation technique. When a supramaximal electrical stimulus is given to a nerve, it can excite all non-refractory muscle fibers and generate maximal force in a subject who is making only submaximal effort. Interpolation of supramaximal stimuli will be able to activate unutilized units and increase EMG activity. However, there will be no change if the subject is making maximal contraction. Thus, the inverse relationship between the size of the evoked muscle twitch following interpolated stimuli and the background EMG of voluntary activity provides an estimate the voluntary effort. After ensuring that the subject is using maximal voluntary effort, endurance and fatigue can tested. The following methods are currently practiced to assess fatigue.[13-17]

Frequency analysis

Electromyographic evidence of fatigue may be demonstrated by frequency analysis of myoelectric signals. During maximal contraction the interference pattern can be "decomposed" and its various components studied. During fatigue there occurs a reduction in higher frequency range (150 - 300 Hz) and a gradual increase in lower frequency range (20-40 Hz). This occurs as a result of synchronization, and change in recruitment pattern, muscle fiber conduction and shape of motor unit potentials. The shift of median/centroid frequency to lower side during surface EMG is thus a useful parameter of fatigue measurement in therapeutic sessions involving muscle exercise.

Acoustic myography

Recently a technique "acoustic myography" has been used for monitoring fatigue.[16] Skeletal muscle generates sounds during contraction which can be heard, recorded and analyzed. It has been observed that there exists a linear relationship between force of contractions and "Root Mean Square" (RMS) amplitude of sound. Further, acoustic signal is intrinsic property of muscle and is unaffected by electrical activity. Therefore, during muscle contraction demonstration of dissociation between electrical and mechanical events in muscle may help in documentation of fatigue. Barry et al[16] recorded acoustic myography and surface electromyography from biceps muscles in normal volunteers during isometric contraction using standard phonocardiography. They observed that at submaximal isometric contraction there was high correlation between the reduction in force production and RMS amplitude. Simultaneous reduction of force, RMS amplitude of acoustic myography and EMG may suggest lack of effort while their dissociation may indicate true fatigue.

Vibromyography

Herzog et al[17] used vibromyography (VMG) in healthy subjects using isometric exercise of rectus femoris (RF) and vastus lateralis (VL) muscle following fatigue protocol and observed that it can be a simple method of measuring fatigue. Vibromyography involves use of an accelerometer to measure lateral oscillations of muscle during contraction and thereby quantify the mechanical property. The subjects were initially trained to follow the fatigue protocol on Cybex dynamometer. They were asked to perform 70% of maximal voluntary contraction (MVC) as long as they could. Fatigue was defined when they could no longer maintain this and test was terminated when the value fell below

50% of MVC. EMG and VMG data were synchronized with knee extension movements and later analyzed for first and last five seconds of the protocol. Power density of raw EMG revealed that there was a significant fall in EMG and VMG signals. Median frequency analysis revealed shift of EMG signal to lower side (from 73 to 54 Hz) throughout the protocol while VMG signal shift occurred rather abruptly (from 40 to 19 Hz for RF) when MVC could not be sustained. Thus, a fall of VMG frequency may indicate fatigue. These results are, however, contrary to the observations on acoustic myography and cannot be translated for exercise or work place.

Electrical stimulation

Electrical stimulation of muscle at different frequency may help in differentiating fatigue due to neuromuscular transmission defect and excitation contraction coupling abnormality. Edwards suggested that in the former, there occurs a selective decrease in maximal torque on high frequency stimulation while when the excitation contraction coupling is defective, the force produced at low frequency is comparatively less, despite a normal EMG signal.

Fatigue is complex and still incompletely understood phenomenon. Measurement of fatigue is in the phase of evolution. Studies involving NMR spectroscopy, EMG and force measurement may improve our knowledge. Currently available techniques are however helpful in optimizing therapeutic exercises and serve as a guide for preventing over-work phenomenon.

Posturography

Human balance is a sensitive and complex process involving detection of body position, integration of sensory - motor information and execution of skeletal muscle response. Sensory input chiefly comes from visual, vestibular and somatosensory systems. Any mismatch of information causes disturbance of balance and interferes in the individual's stance and mobility. Abnormalities of balance pose significant problems in rehabilitation and therefore, quantitation of balance and understanding of compensatory strategies are vital in management.[18,19]

The technique of recording balance is referred to as posturography. There are two types of posturographic recordings:(1) "static" which records swaying during quiet stance and (2) "dynamic" which assesses sensory control of balance and coordinated reflex motor responses after platform perturbations.[20,21]

Static Posturography

The equipment essentially consists of a force plate which senses vertical and sometimes horizontal force exerted by feet on the ground during upright stance. Subject is asked to stand with feet at shoulder width apart, together, and in tandem and on one feet at a time. A computer monitors the force and evaluates amplitude, speed and frequency power spectrum of sway. Static posturography has been used in monitoring physical therapy and as a research tool for dizziness, aging, vestibular, cerebellar and toxic disorders causing imbalance. However, its utility has not been proved unequivocally and it has been replaced by dynamic posturography.[22]

Dynamic Posturography

For dynamic posturographic instrument, the force plate is mounted on a device that can translate horizontally and/or rotate about an axis collinear with the ankle. Some platforms are equipped with specialized visual environments, electromyography and sensory bio feedback systems. The procedure can be divided into two essential components : (1) recording responses to brief movements of platform and (2) recording responses in relation to various sensory inputs, eg. with eyes closed and open or with stationary or moving visual environment.

Dynamic posturography thus provides valuable objective information about the motor performance, postural movements, symmetry of weight bearing and forces generated and strategy utilized (ankle/hip) for maintaining stance. Latency and amplitude of the response in relation to various stimuli can also be measured. EMG monitoring can reveal specific muscles which are activated. The subject can also be given feedback and trained to select appropriate strategies for improving balance.

The technique however has certain limitations. There are only a few types of commercially available posturographic equipments and these too are rather expensive. The information obtained is not etiologic, pathologic or localization specific. The procedure needs active participation and cooperation of the patient for reliable results. However, it has a high detection rate for CNS lesion and holds promise for research.[22]

Gait analysis

Human walking is the most common of all the movements and locomotor disability is a universal phenomenon in physical rehabilitation. Analysis and understanding of normal

and abnormal gait thus becomes very important in neuro rehabilitation [23-25]. There are two methods of gait analysis: (1) Observational and(2) Quantitative.

Observational gait analysis

This was developed at Rancho Los Amigos Hospital, Los Angeles for achieving greater precision of gait description and consists of recording events occurring at joints and adjacent sections. The procedure requires several hours, causes inconvenience to the patient and is unreliable for quantitation. Therefore, most laboratories now use commercially available instruments for quantitative analysis.

Quantitative gait analysis

Walking, being a complex motion, needs analysis of several factors. Essential components of quantitative analysis are : (1) time distance measurement, (2) kinematic factors, (3) kinetic factors, (4) electromyography and (5) metabolic factors. what follows is a brief account of human gait analysis.

Time distance measurement

Foot switches are used for studying walking speed, step frequency, step length, the duration of the stance phase and the pattern of foot/floor contact. The foot switch is a flexible insole that can be fitted into a shoe or taped on to the subjects foot. It contains compression sensors under the areas of the heel, 5th metatarsal head, 1st metatarsal head and great toe. These foot switches can be used to define stride timing, EMG features, electrogoniometric observations and force data during gait cycle. Almost every gait variable alters with a change in walking speed and therefore analysis of data is relevant only when considered in relation to walking speed. Documentation of treatment effects using time - distance measurements provides useful information concerning the patients walking ability and various components and phases of gait cycle eg. swing and stance phase, stride and step length and stride and step time for each extremity. However, time distance walking is only an end product of a complicated motion pattern. It neither explains gait pattern nor distinguishes between the primary gait problem and compensatory strategy used.[24]

Kinematic studies

Kinematic study records movement of the body, body segment or between body segments in different planes and thus involves analysis of motion. This can be performed by photographic and goniometric techniques. (a) photographic techniques : In interrupted light photography, (stroboscopy) the subject with lights attached to the body walks across a darkened room in front of the still camera with a rotating shutter. The photographs are taken at 30 times/sec and shown in one picture. (b) High speed motion picture film or cinephotography can also be used. It exposes 50 frames/second. Advantages of this cinephotography are that no apparatus is attached to the patient, multiple measurement can be obtained in the same session, EMG may be superimposed and recordings from both legs can be made out at the same time. (c) Currently TV devices requiring fast computers and large memories but with inferior resolution have replaced this method and (d) Goniometric technique : Electrogoniometers are devices applied on the exoskeleton structure of the subject. They describe the position of one body segment relative to another. These are inexpensive, easy to apply and the signal is immediately available for analysis. These electrogoniometers do not give absolute angles. Goniometers are commonly used to study knee joint movement. In Polarized light goniometry, the subject is illuminated by beams of light; when the beam is reflected by a marker on the subject, this is detected by photodiodes. For accurate analysis of data, proper placement of markers is essential.

Kinetic studies

Kinetic studies concern the causes of the motion that is the ground reaction of external forces and the internal forces within the joints. (1) Ground reaction forces : According to Newton's third law of motion, the ground or floor reaction force is equal in magnitude and opposite in direction to the force that the body applies to the ground through the foot.The ground reaction forces (vertical,.horizontal and mediolateral forces) directed reflect the accelerations of the body, a key to the study of human locomotion. The vertical floor reaction force varies above and below the body weight because of vertical upward and downward movement of the center of gravity. Transducers are used which change the force into an electrical signal which in turn can be processed and studied. The widely used force plate with piezo electric force transducers is capable of measuring very rapid changes of forces. It also measures center of pressure under the foot. For proper interpretation of data it is important that the plate is hidden, as otherwise there may be "targeting" for plate. Numerous repetitions are required for analysis. (2) Internal

Forces : Determination of 'loads' (or forces) acting on a joint in normal or pathologic state is a major aspect of orthopedic biomechanics and is crucial for the design of implants. External forces result from the weight of the body, the ground reaction force acting on the foot and acceleration and deceleration of the limb segments.Internal forces act to balance external forces and are generated by active muscle contraction and ligamentous forces. These studies require advanced technical instrumentation with computer data processing. Light emitting diodes are fixed at different part of body and motion of limb is determined. An optoelectric system measures the three dimensional position of each light emitting diode on the subject. A piezo electric force plate gives the three components of ground reaction force, the vertical twisting moment, and the location of resultant forces at the foot. Data are collected at different pace of walking and correlated with kinematic factors.

Electromyography

EMG is useful for assessing activity in various muscles during gait cycle. Technically the length of electrodes may pose limitations for the distance walked and cable artifacts may interfere in analysis. Telemetric EMG has been used with some success to overcome this. Patient can have "free" walking and EMG can be recorded. Data can be analyzed raw or after processing. Normal patterns of activation and sequence of muscle contraction have been described for subjects of different age groups and sexes. Deviations from normal timing are classified as premature, prolonged, or continuous and out of phase actions. EMG studies have given significant insight into the problems of disordered gait in patients with hemiplegia, spastic diplegia, Parkinsonism and in elderly individuals. It is possible to determine primary abnormalities and compensatory mechanisms used.[26-28]

Metabolic Energy Expenditure

Fundamental feature of human motor behavior is that a freely chosen rate of activity is preferred that represents minimal energy expenditure per unit task. During natural walking a person chooses an optional step length and step rate, achieving minimal energy expenditure per unit distance. The energy cost can be measured, as indicated by oxygen uptake, during various modes of ambulation by normal or disabled subjects. After the subject has reached steady state, the expired air is collected for a couple of minutes in a bag carried on the back. The air is then analyzed for oxygen and carbon dioxide content to determine metabolic energy factor.

For clinicians the complexity, the technical difficulties and the expertise required for these methods are prohibitive. However, like many other techniques, gait analysis has advanced and now become an integral part of gait laboratories. It has been used for pre and post operative evaluation of patients for cerebral palsy, selection of orthotic and prosthetic devices, understanding the pathophysiology of gait abnormalities in various neurological disorders, planning treatment and monitoring of therapeutic techniques and bio feedback.

Quantification of spasticity

Spasticity is defined as a velocity dependent increase in the tonic stretch reflex ("muscle tone") with exaggerated tendon jerks. It is a common and disabling problem which results from a variety of neurological disorders eg. head injury, stroke, spinal injury and myelopathies. Whenever a patient requires evaluation of spasticity the following question arise. (1) Is spasticity present ? (2) Is it tonic, phasic or combined? (3) What is the contribution of segmental hyperexcitability ? (4) What supraspinal mechanism(s) are involved ? (5) How severe is spasticity and (6) What therapy will be most suitable for the patient ? Neurophysiological methods can partly answer these questions.

Spasticity evaluation techniques can be broadly classified into two groups : (1) Mechanical methods and (2) Electro-physiological methods. Mechanical techniques rely on motion applied to a joint and involve gravitational, manual, controlled displacement and controlled torque methods and have been reviewed recently.29,30 The most easily performed mechanical technique among these is Pendulum test.

Pendulum test

For assessing the spasticity of quadriceps muscle the patient is made to sit and the leg is raised to horizontal level at knee and dropped. The leg oscillates for a few seconds before acquiring static position. Knee movement is assessed by electrogoniometer and rate of movement by tachometer. In view of increased resistance to passive stretch the amplitude and the number of oscillations are reduced in patients with spasticity. Further, it takes longer time for the leg to acquire position of rest. More recently it has been possible to study this phenomenon by using isokinetic systems eg. Cybex II and KIN-COM system. These instruments can be used for different muscles in upper and lower extremities and allow application of passive stretch at varied rate and force. Spastic limbs demonstrate resistance to joint movements, which is augmented by increasing the angle of movement and rate at which it is moved.[29]

Electrophysiological methods

Clinical neurophysiological techniques are useful in documentation and quantification of spasticity. These help in objective recording clinically observed phenomenon eg. hyperactive stretch reflex, clonus, lack of reciprocal inhibition during voluntary movements etc. In addition these are also useful in revealing the underlying pathophysiological mechanism of spasticity.[31-34] Some of these methods are described here.

Tendon jerk : In spasticity, tendon jerks have lower threshold and higher amplitude and are followed by after discharge of motor units. Surface electrodes are placed over the muscle belly and stretch reflex is elicited by electrodynamic hammer. Threshold force required, maximum amplitude of Tendon jerk ("T" wave) and ratio of "T" wave to "M" wave (direct muscle response to supramaximal nerve stimulation) are recorded. Similar to "H" max/"M" max ratio, the "T" max/"M" max ratio also provide information about spasticity (see below). "T" wave can be recorded from many muscles as compared to "H" reflex.

'H' reflex : Hoffman reflex is the electrical equivalent of tendon jerk . Using low intensity and long duration electrical stimulus spindle afferent can be stimulated to activate alpha motor neurons. In normal individuals "H" reflex is restricted to soleus and flexor carpi radialis muscles out in patients with spasticity "H" reflex can be obtained from other muscles also eg. tibialis anterior and intrinsic hand muscles of hand . The amplitude of "H" denotes the availability of excitable motor neurone pool in the spinal cord. The ratio of "H" max to "M" max is normally less than 0.5 but due to increased excitability of alpha motor neurons, the "H" max/"M" Max ratio is more than 0.5 in spasticity . This parameter is very useful in patients with unilateral lesions as the uneffected side of the subject can serve as "control".

As "H" reflex bypasses muscle spindle a dissociation in tendon reflex ("T") and electrical "H" ("T" being more than electrical "H") is believed to suggest increased sensitivity of muscle spindles and increased gamma motor neurone activity. However, this concept is now debated in the light of recent microneurographic studies."H" reflex excitability/ recovery curve also show changes in pyramidal lesion. This is studied by giving a second stimulus at various intervals from the test stimuli. In normal individuals a second 'H' reflex of identical amplitude is noted only when the stimuli are separated by 100-150 msec interval. However, in spastic condition recovery period is shorter and the

amplitude of second 'H' is higher. This depends on the influence of descending tracts on motor neurone.In normal individuals voluntary contraction of tibialis anterior or stimulation of peroneal nerve has inhibitory effect on H reflex due to reciprocal inhibitory mechanism by descending tracts. This may be lost in spasticity and can be demonstrated electrophysiologically.

When a vibrator (100 Hz) is applied over the muscle during elicitation of stretch reflex there occurs an inhibition which remains constant throughout the period of vibration. This is due to presynaptic inhibition on Ia terminals. The same phenomenon can be demonstrated on "H" reflex also. The index so obtained {H max Vibrated / "H" max X 100} is often consistent for a given individual but varies among different people. Vibration inhibition index is reduced in chronic spasticity but not in patients of Parkinsonism or hyperreflexia of other origin and therefore is considered specific. However, this index does not correlate with the severity of spasticity. It is symmetrical on both sides in healthy subjects and therefore, this test is very useful in unilateral lesion. Diazepam enhances the vibration inhibition index in spasticity while Baclofen does not. Selection of antispastic drug can thus be based on this therapeutic test.

"F" wave : "F" response (wave) is obtained from muscle by supramaximal stimulation of its motor nerve and is believed to be due to antidromic activation of alpha motor neurones. Following recovery from acute state patient with hemiplegia and patients with chronic spasticity show increased amplitude and persistence of f wave and reflect hyperexcitability of alpha motor neurone.

Tonic vibration reflex (TVR) : When a vibrator (100-120 Hz and 1-3 mm displacement) is applied to a muscle continuously, a steadily increasing number of motor unit potentials (MUPs) can be seen during EMG recording of a healthy individual. This activity reaches its plateau after a few seconds and is maintained throughout the period vibration is sustained. Simultaneous recording of force also shows the same changes. In contrast, patients with spasticity may show shorter latency and lower amplitude response for a variable period. Thus measurement of onset, amplitude, duration and irradiation of activity to other muscles can serve as a measure of spasticity. Vibration stimulates primary and secondary endings of muscle spindles, thereby Ia and II afferent. Through polysynaptic circuits, the response may irradiate to other muscles as well and can be measured. However,the test is not easily quantifiable and does not have significant practical value.[32]

Plantar withdrawal reflex : Stroking, the plantar surface of foot electrically results in flexion of great toe and adduction of other toes. In patients with spasticity there occurs an extension of great toe and abduction of other toes. This is believed to reflect global interneuron activities. In some patients excessive muscle contractions of other muscles and withdrawal of extremities is noted. This phenomenon can be recorded by polyelectromyography and the threshold, pattern size of elicited plantar response can be observed. EMG activity can help in differentiating voluntary withdrawal and genuine abnormal plantar response. A reduced threshold and increased size of response are characteristic feature of spasticity and can be quantitated.

Thus, electrophysiological methods can help detection and quantification of spasticity (Pendulum test, "H"max/"M" max ratio), understanding of pathophysiological mechanism (Abnormal "f" and "H" suggest alpha motor neurone hyperexcitability, "H" reflex recovery curve provides evidence of descending influences and vibration induced alteration in "H" reflex indicates presynaptic inhibition) as also selection of therapy (Diazepam for patients with presynaptic mechanism and baclofen for patients with abnormal "H" reflex recovery) and long term monitoring of patients.

Urogential dysfunction

A number of patients develop bowel, bladder and sexual dysfunction due to neurological disorders and require rehabilitation. When combined with clinical and urological tests, electrophysiological techniques provide useful information about the underlying mechanism for the dysfunction. Commonly used methods are : (1) sphincter electromyography, (2) study of sacral reflexes, (3) evoked potential studies and (4) pudendal motor latency.[35-38]

Sphincter EMG

This has been in use from early days of clinical EMG and has two main applications : (1) To record activity of urethral sphincter during urodynamic studies and (2) to assess the innervation of pelvic floor muscles. The striated muscle is active tonically to maintain continence all the time. Its activity increases abruptly whenever there is a rise in intra abdominal pressure and it becomes silent during detrusor contraction when voluntary voiding is performed. As urethral sphincter is not easily accessible EMG is done from anal sphincter on the presumption that both the sphincters behave in similar manner. Various electrodes used are : surface electrodes on either side of anal region, anal plug

electrodes to be put into anal canal, electrodes mounted on catheters and sponge mounted vaginal electrodes to be placed behind the urethral sphincter. Detailed EMG analysis of motor units of anal or urethral sphincter can be done by direct insertion of concentric needle or single fibre EMG needle. For male urethral sphincter needle is inserted in left lateral position through perineum, 4 cm in front of anus and being guided through finger in rectum towards the apex of prostate. While in woman it is passed through trans-vaginal approach. Sphincter EMG study helps in identifying detrusor sphincter dyssenrgia, various causes of incontinence and dysuria eg. high detrusor pressure and low urinary flow with actively contracting sphincter may suggest detrusor sphincter dyssynergia while a relaxed sphincter in the same setting may indicate obstructive pathology. These studies have also provided evidence that stress incontinence in woman may have neurogenic basis.35 Bio feedback technique using sphincter EMG is useful for children with enuresis.

Sacral reflex

Latency and type of reflex contractions of the pelvic floor muscles in response to stimulation of genitalia or perineum have been studied by electrical stimulation and measure function integrity of sacral reflex arc. Bulbocavernosus reflex can be recorded using concentric needle inserted into the bulbocavernosus muscle or surface electrodes placed over the same muscles. Stimulation of penis is done through ring or hand held bipolar stimulator. A consistence response has two components. An early response with a latency of 24-45 msec and late response with a latency of 60-70 msec. Similarly at a relatively longer latency of 59 _ 8.0 msec. recorded. Vesicourethral and vesicoanal reflex can be elicited from respective sphincter muscles by stimulation of catheter mounted ring electrode. These studies are useful in the evaluation of patients with impotence and when abnormal, suggest neurogenic basis for it[35].

Evoked Potentials

Somatosensory pathways from genitalia to cortex can be evaluated by stimulating dorsal nerve of penis or clitoris and recording potentials from scalp, two centimeters behind CZ. The so recorded pudendal evoked potential have latency and configuration, similar to posterior tibial nerve potential.[35,37] Stimulation of bladder and urethra also evoke cortical response. However, these are of lower amplitude and longer latency, because the afferents involved are probably small unmyelinated fibers.

Motor evoked potential from pelvic floor muscles, anal and urethral sphincter can be obtained by electrical or magnetic stimulation of cortex and spinal cord. It is important that the recording is done by needle electrode so that EMG contamination from other stimulated muscles is avoided.

Pudendal nerve latency

Study of conduction in pudendal nerve was todate difficult due to the inaccessibility of the nerve. A newer system consisting of stimulating electrode fixed at the tip of finger and recording electrode at the base of index finger has made it possible. Finger is introduced into anus and the pudendal nerve is stimulated at the site it crosses ischial spine. EMG response can be recorded at a terminal latency of 2.1 - 0.2 m.sec from anal sphincter and 2.4 - 0.2 m.sec from urethral sphincter.

The EMG from smooth muscle of corpus cavernosum (CCEMG) can be recorded using concentric needle electrodes. Rhythmic bursts of activity seen when penis is flaccid. Penile tumsence is accompanied by silent CCEMG. In subjects with neurogenic impotence CCEMG activity persists during sexual stimulation preventing erections[35].

Sympathetic skin response is a simple, non-invasive electrophysiological t est for sudomotor function. SSR from perineum is a potentially useful test for evaluation of impotence and incontinence[35].

These techniques in varying combination have been widely used to investigate patients of fecal and urinary incontinence, monitor children with spina bifida and neurogenic impotence.[35-38]

Assessment of residual function

Recovery of patients following neurological injury depends on the severity and extent of lesion. Accurate prognostication, selection of restorative procedures and critical evaluation of therapeutic interventions need documentation of the integrity of the descending influence and residual function.39-43 Electrophysiological methods being highly objective have played an important role in assessing residual function of the descending tracts and final motor out put. Many techniques have been used for this by Dimitrijevic and his colleague to assess residual upper and lower motor neuron control, following a protocol known as "Brain motor control assessment" (BMCA), in subjects with spinal injury.[39]

The surface electrodes are applied over the muscle belly of quadriceps, adductor, hamstring, triceps surae, tibialis anterior bilaterally and lower abdominal and lumbar paraspinal muscles. Myoelectric signals are recorded from these muscles with a high sensitivity using a number of maneuvers and data obtained from the polyelectromyography are analyzed.

Voluntary effort

EMG activity is recorded from the muscles after asking the patient to contract the muscles at multiple joint on three trials. Presence of EMG activity in clinically "paralysed" muscles suggests preservation of motor control.

Reinforcement maneuvers

After a relaxation for 10 minutes in supine position patient is required to perform reinforcement maneuvers consisting of forceful closure of eyes, clinching of jaws, forceful shrugging of shoulder against resistance, neck flexion against resistance, deep inspiration, clinching of fist etc. These maneuvers are repeated on three occasions. A dissociation between EMG activity during voluntary activity and reinforcement maneuvers provide information regarding integrity of upper motor neurone.

Reflex studies

Recording of tendon reflexes, vibration reflex and plantar reflex also form part of the study. After base line study the subject is asked to augment or suppress the response. Demonstration of patients ability to modify the response indicates residual descending influence. Further, the effect of Jandressik maneuver, caloric stimulation and audiospinal facilitation has also been studied on tendon and "H" reflex.[31,41-43] Audiospinal and caloric stimulation facilitation of stretch and "H" reflex help in evaluating reticulospinal tracts .

Transcranial stimulation

Magnetic and electrical stimulation of cortex and spinal cord can evoke motor response from muscle and provide indication about the functional integrity of pyramidal tracts. Sherwood et al[42] using Brain motor control assessment protocol analyzed data of the patients with spinal cord injury referee for rehabilitation. They observed that of the 88

patients with clinically "complete" lesion, 74 (84%) were "discomplete" lesion i.e. had evidence of residual brain influence. These tests are now routinely used prior to therapeutic intervention, eg. gait training using body weight support system for spinal injury patients, functional electrical stimulation etc. However, these tests are time consuming, require patient's cooperation, accurate application of electrodes and precise documentation of data.

Biofeedback

Feedback is an engineering term defined as a method of controlling a system by reinserting into it results of past performance. The term biofeedback is a convenient abbreviation of biological feedback. It is a technique where in covert physiological processes are made more overt to the patient. Biofeedback in rehabilitation can be used to inform the patient about muscle activity, movement, force, balance, gait, joint displacement and other physiological activates by amplification and proper display so that the subject can learn to control them. Electromyographic feedback (EMG feedback) is the commonest technique applied in physical therapy.[44,45] Its proper use requires knowledge of physiological principles of motor control in normal and abnormal states. The functional unit of motor system is single motor neurone, its axon and muscle fibers supplied by it. These have different anatomical, physiological and histochemical properties. Recordings from single motor units in healthy individual have suggested that during the process of recruitment, small low threshold units are activated first and as the tension is increased, high threshold larger unit are brought into action. Similar pattern is seen during relaxation i.e. large units derecruit earlier than the small units. Within the limitations of this size principle, an individual can be trained to recruit at a desirable frequency when the audiovisual feedback is provided. Characteristics of single motor unit (SMU) can be studied in various disorders of nervous system using EMG technique and can be used for feedback.[10,11]

The basic EMG biofeedback device includes one ground and two active surface electrodes, an amplifier, an audiospeaker and a video display. EMG signals can be displayed raw or in the integrated form after processing. The training consists of either relaxation of hypertonic muscles or recruitment of muscles that need facilitation. On the similar lines patients can be given feedback of point motion (Kinematic feed back) using rheostat, standing balance (posturography) and dynamic force feedback (Kinetic feedback) to control mobility in orthopedic and neurological disorder requiring rehabilitation. However, patient can not be attached to a machine and left alone to

practice. Continuous supervision, cuing, adaptations and variation in exercises are necessary. Similar to any other therapeutic program. It is note worthy that improperly used EMG feedback can cause increase in spasticity.[46]

EMG feedback is in practice for more than three decades and has been used in rehabilitation of patients with stroke,[47,48] head injury, back pain, cerebral palsy, spinal cord injury, Bell's palsy, peripheral neuropathies, torticollis and other dystonia. Poor designs of studies, small sample size, improper quantitations of deficits and benefits are some reasons which do not allow accurate conclusion about their utility. However, it is a potential technique for rehabilitation which has neurophysiological basis.

Neurostimulation

Functional electrical stimulation (FES) is a neural prosthesis that utilizes stimulation of neural tissue. With the development of sophisticated neurosurgical techniques and implantable electronic stimulators it is now possible to selectively stimulate the neural tissue at various sites along the neuraxis for relief of symptoms.[49-52] These stimulators not only avoid ablative surgery but are also safe for long term use. They can be monitored and removed whenever required. Several stimulation technique have been used for many disorders eg. pain (transcutaneous electrical nerve stimulation and stimulation of dorsal spinal roots, dorsal column, peri acqueductal grey and thalamus), spasticity (dorsal cord and cerebellar stimulation), epilepsy (cerebellar and thalamic stimulation), respiratory disorders such as cervico medullary lesion and sleep apnea (phrenic nerve stimulation) micturition disorders (pelvic floor, anterior sacral root, conus medullaris and dorsal cord stimulation), impotence (electro ejaculation) and others.[51,52] EMG signals from residual muscles have also been used for triggering movements for prosthesis eg. myoelectric arm.49 It has now been possible to generate reciprocal stepping pattern in patients with paraplegia using functional electrical stimulation.[50] Clinical electrophysiological methods play crucial role in selection, application, evaluation and improvement of various neurostimulation techniques.

Conclusion

Clinical neurophysiology has enhanced understanding of normal and abnormal functioning of nervous system. These techniques are available world wide, standardized, risk free, require limited patient cooperation and provide quantitative results which permit intra and inter individual comparisons. They permit assessment of impairment in physiologic

terms and allow development of therapeutic strategies which can correct these dysfunction. However, these techniques do not measure disabilities and may not be applicable in all circumstances. Further, some of these technique need mechanical devices and other methods for comprehensive evaluation. Nevertheless these techniques are complementary to clinical evaluation and to a certain extent therapeutic interventions.

Acknowledgements

The secretarial assistance of Mr.M.V.Srinivasan and Mr.K.Bhaskar and help of the staff of the departments of medical illustration and clinical electrophysiology section of Neurology are gratefully acknowledged.

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Where do I go from here ? Rehabilitation of a stroke survivor

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Introduction

Stroke has always been perceived as a devastating illness. The public perception towards it has been "it is all over ", " stop" and "the end".The fatalistic attitude towards it extends beyond the acute care. It is distressing that in undergraduate medical curriculum in India there is no emphasis on rehabilitation. It is no surprise, therefore, that most of the physicians have little understanding of the basic principles of rehabilitation. This article aims to present an overview of the principles and practice of stroke rehabilitation. It is not intended to describe the details of physical therapy, for which, nothing can replace hands-on demonstration.

What is stroke rehabilitation?

The word rehabilitation is derived from the Latin word, habile, which means to make able, and the Latin prefix, re, meaning again. It was initially used in the 18th century when sinners and transgressors who had repented and made amends were once again able to wear the dress of their denomination. "To make able again " is the essence of modern stroke rehabilitation.[1] Stroke rehabilitation is a program designed to help the stroke victim overcome the disability resulting from brain damage and to enable him to function at physical, psychological and social levels despite the disability that remains after all spontaneous recovery from brain damage is ceased. It is important to know that rehabilitation encompasses the whole range of techniques and arrangements to make a person as independent as possible. It is not synonymous with physical therapy alone as is commonly believed. Physiotherapy is just a part of it.

Does stroke rehabilitation really help?

A number of uncontrolled and controlled randomised studies have suggested that rehabilitation programs lead to an improvement in functional status that cannot be attributed merely to spontaneous recovery.[2,3] No specific, intervention, philosophy, or mechanism has been proved to account for this improvement. This benefit may be statistically small but for the individual patient it could mean much difference.[4]

When should rehabilitation begin ?

Rehabilitation should probably begin as soon as possible after the acute event. For patients who are only mildly involved with paresis and who retain voluntary movement, generally little in the way of immediate treatment is needed.[5] For individuals with complete paralysis treatment is more intense, although whether highly intensive treatment is better than moderately intensive therapy is debatable.[4]

Phases of stroke rehabiltation

The whole aim of rehabilitation is to make a person independent in his daily living. This process starts from intensive care unit itself and extends well into the home and even the work place of individual. Thus we have early, intermediate and long-term phases of rehabilitation.

Early phase

At this stage, medical and neurological stabilization is a priority. It is also necessary to initiate the plan for alleviating the disabilities that occur as a result of stroke. The ultimate goal is to enable the patient to enter intermediate and long term rehabilitation. Following things need care in acute phase:

Skin care : Lack of movement results in continuous pressure on skin covering bony prominences such as heels, hips and scapulae. This unrelieved pressure impedes blood flow , and the tissue dies due to lack of nutrients. This results in a pressure sore. Warning sign that the integrity of the skin is threatened are localized warm, red spots which do not disappear after pressure has been relieved. Persons with diabetes are already prone to skin problems caused by poor circulation in the arms and legs and should be particularly careful after a stroke. An egg crate type of pressure release mattress is recommended to prevent pressure sores. The bed should be kept flat to reduce pressure on the bony prominences and reduce the tendency toward contractures of the hip and knee. Position should be changed at least every two hours. If an ulcer has developed, it should be cleaned with antiseptic. Diet should include protein, vitamins and zinc. When adjusting the patient's' position, the staff and family should be cautioned against pulling paralyzed extremity.[6]

Prevention of contractures : Joints that are immobilized for long periods of time because of paralysis are likely to develop contractures which may eventually interfere with

rehabilitation process. Therefore, the initial goal of rehabilitation is to prevent joint and muscle contracture.. This is achieved by passive range of motion exercises done thrice daily. Family members should be taught how to provide a full range of motion. Passive range of motion exercises may help patients to mobilize remaining voluntary movement. Some patients report that passive range of motion exercise seem to make possible the initiaton of voluntary movement.[7]

Proper positioning : At all times limbs should be kept in a proper position. Learning to live in a recovery pattern will prevent crippling contractures. Normal anatomic alignment of the head, trunk and limbs should be maintained. The patient should not sit on one hip or lean on one side persistently when sitting. Each joint should be positioned in the opposite direction from its spastic position as much as possible. For example, the fingers should be kept extended at all times, and the ankle-foot angle at 90 degrees. When the patient is in bed, the paralysed arm should be slightly elevated to promote venous drainage, and the foot of the paralysed lower extremity should be placed in a posterior splint, which prevents plantar flexion and external rotation of the foot. While sitting, the chair should be low enough to allow feet to be flat on floor which allows the hips, knees, and ankles to be at right angles. The chair should be firm enough to provide support. While lying it is always best to lie on one side or the other but one should frequently reposition. [8]Head position affects muscle tone of trunk and limbs. If it is always turned to one side, an abnormal posture will develop, hindering mobility. Therefore all body parts should be kept in a normal position centred above shoulders. In bed, head should be supported with one pillow. When sitting, patient should be encouraged to keep his weight evenly distributed on both hips which will keep trunk in proper position. If shoulder is mildly dislocated, pillows should be used to keep the upper arm and elbow out and away from chest.

Painful shoulder syndrome : With flaccid paralysis of muscles around the shoulder, the weight of the arm may stretch the shoulder joint capsule and subluxation of the joint may occur when the patient is upright. This is apparent on examination and on roentgenogram. A paralysed , immobile arm that hangs loosely at the patient's side often becomes edematous when the patient is sitting upright because of impaired venous return. This can be overcome by supporting the patient's arm in a sling or by using a special device placed on a chair to keep the arm supported.This reduces stress on the shoulder joint and brings the arm is slightly above or at the level of the heart. A pressure glove can reduce hand edema. Another cause of pain in the shoulder associated with immobility of the arm is adhesive capsulitis. This pain may be constant but is made

worse by moving the arm. The problem may begin a few hours after the onset of paralysis and can be prevented by gently moving the arm through the normal range of motion several times a day. Treatment includes nonsteroidal anti-inflammatory agents and sometimes corticosteroids such as prednisone given initially in doses of 40 to 60 mg per day , tapered over about two weeks. However steroid therapy should be reserved for patients who do not respond to other means of treatment[5]

Sensory neglect : Sensory neglect can be an immediate or intermediate problem in stroke rehabilitation. This is liable to happen in right hemispheric strokes. The paralysed arm can be completely neglected, which increases the possibility of injury from trauma and presssure. The patient is not in a position to pay attention to the arm and protect it. The arm must be prevented from falling with the use of a sling.

Bladder disturbances : Retention of urine is often managed initially by insertion of an indwelling catheter, which allows for continuous drainage of urine. However, because this procedure involves the risk of infection, intermittent catheterization may be used. This involves periodically draining the bladder by inserting a small catheter every four to six hours until satisfactory bladder function returns. Incontinence is a common problem after stroke and is due to impairment of the neurologic system controlling urination and depression in the level of awareness. Several simple measures can help. Often the cause of incontinence is the inability to hold the urine long enough to make it to bathroom. One way to deal with this is to have a means of signalling for assistance as soon as the urge to void is felt. Another method is to go to the bathroom every two hours during the day (timed voiding) to keep the bladder from filling up. This can be done until better control is regained. Similarly, restricting fluids after the evening meals can prevent night time incontinence. Finally, it is important to provide as much privacy for toileting as possible. In cases of persistent incontinence caffeinated and alcoholic drinks should be avoided. If all the above measures fail, one should consult a urologist to consider infection or any other remedial urological problem.

Bowels : Constipation and bowel incontinence are frequent problems following a stroke, resulting from a variety of causes. These may include reduced fluid intake, limited mobility, lack of awareness or failure to respond to the urge for defecation, or the inability to request assistance. It is important to restore a predictable bowel movement schedule as soon as possible. An initial approach to establishing a pattern is to determine the person's prior bowel habits and make sure that there is opportunity to defecate according to that pattern. Privacy and use of the toilet are important. The sitting position

allows one to lean forward, thus increasing intra-abdominal pressure to aid in expulsion of the stool. A diet with adequate fluid and bulk is necessary. If constipation persists, a stool softner and/or bulk agent may be helpful.

Deep venous thrombosis (DVT) : It has been showed that in the first week of stroke 30-75% of patients have evidence of DVT on investigations. The efficacy of heparin in the prevention of DVT is undisputed. Reduction in DVT and pulmonary embolus with intermittant pneumatic compression devices is similar to that with low-dose heparin and may be a safer alternative in patients with spontaneous or traumatic hematoma. Preventive treatment is usually given for two weeks although the ideal time to stop heparin is when patient starts walking. If patient develpes DVT, then heparin is given intravenously for 10 days to maintain a partial thromboplastin time of 1.5 times control, after which warfarin is adminstered for 3-6 months.[9]

Intermediate phase

A clearer picture of patient's disability emerges at this time and accordingly one can choose the rehabilitation program most appropriate to the patient. All patients are not likely to derive equal benefit from rehabilitation. A conceptual scheme for assessing the functional potential of each surviving stroke patient is a basic foundation for the rational management of the rehabilitation process. This helps to keep the goal realistic. The most powerful determinant of functional outcomes in stroke would appear to be the specific neurological deficit. Certain deficits like aphasia have an obvious negative impact on the future functional levels achievable by stroke survivor. More subtle organic cognitive deficits can affect the ability to learn. Special techniques have to be used to obviate such difficulties. For example the most rehabilitation workers are trained to use visual cues in patients with left cortical lesions and verbal cues in those with right cortical lesions. Co-morbidity is crucial for in functional prognosis. For example diseases like chronic obstructive lung disease, arthritis or amputations will interfere with the patient's eventual ability to walk. Psycho-social and financial factors also determine the final outcome. A formal evaluation of mood is often useful in the initial stages of rehabilitation because psycho-social malady like depression can impede the progress of rehabilitation. There are variety of scales and scoring systems which are used for mental, psychological and motor assessment.[10] If depression is present, it should be treated medically as it may improve the outcome of rehabilitation programs.[11] In an effective rehabilitation program, all aspects of the patient's disability should be examined and carefully assessed because one aspect may severely interfere with performance in another. These problems

must be considered and dealt with as quickly as possible by several staff members .The personnel usually required to provide adequate rehabilitation for victims of stroke include 1) Neurologist or a Physician interested in stroke 2) Skilled nursing staff 3) Physical therapist 4) Occupational therapist 4) Social worker 5) Neuropsychologist and 6) speech therapist. This group should meet regularly to assess the progress. Patients should ideally be discharged to home when either they improve to the point of being able to be cared for at home or when their further progress reaches a plateau. .

Physical therapy : There are numerous techniques of physical therapy, many of which are based on old concepts. Whether any strategy currently available to enhance motor recovery is superior to spontaneous recovery remains controversial. Most techniques suggest different approaches to similar clinical problems. For example, the Bobath approach strives to suppress any abnormal patterns of movement associated with recovery from stroke, while the Brunnstorm approach actually recommends the facilitation of mass movement (synergy patterns) early in the recovery process.[12] A more recent approach proposed by Carr and Shepherd emphasises the mastery of functional motor skills which are task specific, consistent with current theories of motor learning.[13] Despite the lack of proof of efficacy, Ernst concluded after a careful review of literature that rehabilitaton is preferable to spontaneous recovery, regardless of which physiotherapy is chosen.4 Physical therapists encourage the patients to use paralysed extremity parts as often and as effectively as they can with a variety of exercise programs.

The training for ambulation : Most patients who have paralysis of the upper extremity have less weakness of the lower extremity. With suitable encouragement, education and considerable training by the physical therapist and occupational therapist most of the stroke survivors will learn to walk. The whole process goes through certain phases.

Sit up phase : Of necessity, thepatient's return to vertical tolerance must precede other mobility training. Cardiac instability, postural hypotension, truncal ataxia, and severe general weakness are most often the delaying factors and must be paid proper attention. Patients should be made to sit for at least 15 minutes every 2-3 hours and adequate back support provided. As the patient shows improvement, the back rest should be taken out and patient should be encouraged to sit unsupported. Patient should be allowed to shift weight alternatively and rhythmically from hip to hip, side to side and forward-backward. This will enhance the sitting balance. Neck and spinal extension should be maintained as this incorporates the feeling of independent sitting in patient. Wheelchair transfers must be successfully carried out to complete the Asit-up phase@

for patients who are unable to walk. In this stage the hemiplegic limb is usually in various degrees of flaccidity. An important technique for transferring patient from bed to wheelchair should be learnt at this stage. This is called A pivot transfer @. It is accompalished by the helper placing his heels together with the feet angled to form a V. The patient's hemiplegic foot is placed in the V, and the helper' knees are aligned to either side of the patient's hemiplegic knee. Using good back mechanics, the helper flexes at the hip and knee, has the patient hold on at the waist with the most functional upper extremity, gently rocks the patient forward to move the center of gravity over the feet, then stands up with the pressure of the helper's knees against the patient's knee to lock it into extension. This manoeuvre does not require the helper to physically lift the patient; rather, the helper guides the patient and provides safety against collapse of the hemiplegic leg as the patient provides the power lift with his hip extensors and knee extensors. Once the patient is standing, a pivot placing the back of the patient's thighs against a well-placed locked wheelchair is accomplished, and the patient is allowed to sit down in a controlled fashion with the helper exercising a controlled knee and hip flexion and the patient using the unaffected arm and leg to control the sitting process. The same transfer technique can be successfully used to accomplish toilet transfers from the wheelchair.

Stand up phase : Walking requires that the patient first sits up, then stands up. Standing up requires adequate cognition to understand the process, motor planning skills, freedom from contractures, balance skills, and adequate strength or useful spasticity to support the hemiplegic limb. Ankle and knee control is necessary. If patient has a flail ankle or inadequate dorsiflexion and inversion and eversion control, an ankle foot orthosis should be provided as an ankle control device. Hirschberg has long advocated a A stand-up-step-up exercise regimen in which patients may be treated in groups or individually. A simple stable chair or parallel bars may be used. To ensure success from the first therapy session, the height of the bed is adjusted upward or risers are placed on the chair so that, when the patient is asked to stand up, he can do it easily. The helper may stand opposite the patient in a position similar to that for the standing pivot transfer conducted during sit-up phase. The patient places the unaffected hand palm down and pushes the parallel bar or chair back, at the same time the unaffected leg is used to accomplish the stand up. The patient is instructed to avoid gripping the bars or chair and to push down and not pull up on the bar. These are the same mechanics necessary for successful walker or cane use. As the patient's capability to perform 10 self paced stand up movements improves, the task is increased in difficulty, the seat height is progressively lowered until standard heights are mastered. The patient's progress with this stand up

routine is easily measured by noting the number of inches or risers lowered, the number of stand ups achieved and the duration it took to achieve them. This exercise is most functional and easily followed with hand signals, even in aphasic patients. Effective and progressive strengthening of hip and knee extensors, and shoulder stabilizers occurs. Once the stand up is achieved, the patient practices balance and weight shifts side to side at the parallel bars and may perform toe standing exercises to develop the triceps surae and toe plantar flexor function. A cadence of stand up, balance, toe up, and sit down is followed. As voluntary control returns to the hemiplegic side, it too contributes to the stand-up-sit-down effort. The program lends itself to the group exercise, which is valuable with the minimum staff of the hospital. As success is obtained with the stand-up programme, a psychologic high may be experienced by the patient. Once safe stand up motion and balance is achieved, the patient advances in ADL skills training that takes advantage of verticality, including hygiene and lower extremity dressing skills.

Step up phase : For locomotion to occur functionally, hip flexion must return to the hemiplegic side, allowing for forward progression of the leg. Hirschberg advocated step-up exercises as an effective means of increasing hip flexor, hip extensor and abductor, and knee extensor strength. Using a stairway with handrails, the patients stands up with the unaffected leg while maintaining balance with the hemiplegic extremity and opposite unaffected arm on the rail. Once the unaffected leg is in place on the step and the patient steps up, the hemiplegic leg is brought to the same step as the unaffected foot, balance is regained, the hand is advanced on the rail, and a repetition of the sequence for the next step is made. The patient descends backwards, still facing the steps and with the hemiplegic leg lowered to the next lower step, and balances with the unaffected leg and the unaffected arm on the rail. The unaffected leg controls the rate of descent and is then lowered to the same step as the hemiplegic leg, while the hemiplegic leg and the unaffected arm maintain the balance. The process is repeated until the ground is reached. Only later with mature gait does the patient attempt to descend stairs while facing the ground. Until good reciprocal leg function is regained, the one-step-at-a-time pattern is retained and alternate stepping is avoided.

Step out phase : At this stage the patient is ready to apply neurologic recovery and balance, motor control, strength and endurance gains to the ultimate goal espoused by most stroke syndrome patients-to walk again. Many techniques have been advocated for device-assisted gait training, and different therapists use different approaches depending on the individual needs and preferences of patients.

Braces, Canes, Walkers and Other Devices: Patients who are well motivated and who have any residual strength in their lower extremity on the paretic side can usually, with suitable bracing and support, bear weight and become ambulatory. Such patients can be trained to walk with canes, with a walker or with other individual's support. When one leg is weaker than the other, the appropriate sequence of walking should simulate a natural pattern as much as possible. In normal persons, opposite arm and leg move together while walking. The stroke survivor should, therefore, first advance the cane held in the unaffected hand, followed by advancing the affected leg and finally advancing the unaffected leg. Patient should also be made to practice to walk backwards and sideways. Among these patients, foot drop is common. It is necessary to provide a foot drop brace so that the patient can elevate the toes while taking step. There are a number of braces that provide adequate correction of foot drop and help to overcome the tendency for inversion of the foot, which occurs with developing spasticity. Another problem associated with leg weakness is retrocurving of the knee joint because of lack of muscle strength to keep the joint in proper alignment. There are a number of braces that help avoid this problem. When patients are moving but have a paretic upper extremity, it must be supported by a suitable sling because a dangling arm may increase the stretching of the shoulder joint capsule. During the progression from wheelchair dependence to independence walking, aids like cane and support from caregiver will be needed. First there will be parallel bars in the physical therapy department. This will be followed by a large-based quad cane. Finally a standard cane will be used. In the initial phase of rehabilitation, cane or any other walking device helps to lean on, but as the patient improves, the aid helps with balance more than weight bearing. It may also reduce fatigue which increases walking endurance. Another function of cane is to prevent development of limp, which is the result of uncorrected muscle balance. The height of any walking device should be adjusted to allow a patient to stand erect, with a slight 15 degrees bend in patient's elbow. A walking aid should always be held in unaffected hand. A patient may not need a cane at all after a while but this should be discussed with physical therapist. Following a stroke, an individual may need any one of a variety of devices to help compensate for lack of muscle tone or to counteract the excessive tone found with spasticity. These devices are prescribed, ordered, fabricated, and fitted by members of the rehabilitation team. The following ones are most frequently used:

Slings are used primarily when the individual is walking. There are two kinds of slings. A hemi-sling is generally used for a flaccid arm in the early stages of rehabilitation. It holds the entire limb in close to body to prevent injury. Since there is no voluntary motion in this stage of recovery, the forearm and hand are also supported. A shoulder

girdle sling holds the shoulder joint up in its normal position. It can prevent pain and maintain joint alignment while muscles are regaining strength. The elbow and forearm are free to move with this kind of sling.

Resting hand splint maintains the affected hand in a functional position (open fingers, thumb away from palm, and wrist slightly extended). If there is spasticiy, a resting splint will slowly relax the muscle tightness and prevent finger contractures.inger spreader maintains the affected hand in a relaxed position with fingers spread and wrist in slight extension.

Swallowing disorders: Besides disrupting other motor pathways, strokes commonly cause dysphagia thereby impairing chewing and swallowing. Patients may pocket food in the mouth on the side of the hemiparesis, have a slowed or absent swallowing reflex, and have a tendency to aspirate. Modern speech pathologists are trained in the bedside evaluation of dysphagia. A swallowing study using barium of various consistencies may help the therapist determine what are best approaches to treatment. Although families are eager to feed patients right away, the physician should remember that aspiration can occur even in the absence of coughing. While speech pathologist can recommend specific treatments, most patients with an impaired swallowing mechanism can better tolerate a thickened pureed diet than a full liquid diet. Recommendations include leaning to the strong side to allow gravity to propel the food bolus to the location of the stronger pharyngeal muscles. A chin tuck maneuver is used at times to alleviate collection of material in the pharyngeal areas and improve epiglottic closure. For those who are unable to swallow without significant risk of aspiration, nasogastric feeding is recommended. Nasogastric feeding is also a helpful supplement for those who are unable to take in adequate calories due to dysphagia. Patients with a unilateral hemispheric stroke usually regain enough function to give up their feeding tubes. However, patients with bilateral strokes and brain stem strokes often have a profound impairment of swallowing that will persist for months, if not permanently. For those patients, percutaneous placement of a gastrostomy (PEG) has become an easy and advisable procedure. PEG is recommended for those who may be tube feeding for as little as 4-6 weeks because NG tubes are associated with complications such as sinusitis, nasal and pharyngeal erosions, breathing difficulties, pneumonia, and a potential for tube dislodgement.[5]

Speech therapy : The speech therapy is based on the deficit in the individual patient. The results are varied. The best results are obtained in patients with single infarct who start treatment within three months after stroke. Three hours of therapy per week are

reported to give good results. In the last few years many aided and unaided communication systems have been developed. The unaided communication system includes gestures, body language, facial expression, head nodding and blinking. The aided communication systems are sign language, cued speech, use of communication books, typewriters, computers and voice output communication systems. Another recent advance in aphasia treatment is melodic intonation therapy.

Cognitive rehabilitation: Patients with stroke may be left with cognitive disturbances which will vary from person to person. These may not entirely or even partly recover but with patient's personal resolve, determination, family support and guidance by rehabilatationists many of these difficulties can be surmounted.

Agnosia : Agnosia is the inability to associate an object with its use. A person with agnosia may use a toothbrush to comb hair, drink shaving lotion, or be unable to recognise the face of a familiar person. This can be potentially dangerous. In order to deal with this problem, it is necessary to make the living situation free of dangerous objects or poisonous substances. Activities must be structured and monitored closely. The family should also remind the stroke survivor of the correct ways to use objects, as often as possible.

Apraxia : Apraxia is the inability to voluntarily perform certain movements, despite adequate muscle strength. In other words, the person is able to perform the movement, when not thinking about it , but cannot do so if asked. There are several different manifestations of apraxia. In motor apraxia, for instance, the person may be physically able to stand up, but unable to do so if asked. Another kind of apraxia is dressing apraxia. The person may be unable to put on a sweater, if asked, but may later slip it on without thinking or may put on clothes inappropriately, such as upside down, inside out, or on the arms. Apraxia may appear to be stubbornness (when it is not) to the caregivers and can lead to misunderstandings. Caregivers can help by talking the person through the activity, guiding the hand and demonstrating the desired movement. For example it is better to tell them "get dressed" rather than "put your arm here". Stroke survivors with apraxia need a routine for activities of daily living so that daily repetition becomes a pattern.

Body Scheme Disturbances : Body image disturbances are changes in how one perceives oneself mentally and how one understands one's body and its parts. For example, the patient may not be able to differentiate his right from his left side. At times

patient may confuse his arm or leg with those of someone sitting next to him. Such patients find it very difficult to perform everyday activities correctly. It is helpful for the caregiver to give frequent reminders of right and left, as well as identifying body parts. A full length mirror or other feedback techniques, such as pictures of persons, can be helpful. With therapy and daily repitition, this problem often can be resolved or at least significantly improved.

Hemineglect : Hemineglect is the inability to perceive the environment on one side of the body, resulting in that side being ignored and not used. This problem is usually experienced more often by someone suffering from left-side paralysis. Examples of one-sided neglect include unknowingly letting the arm dangle over the side of the wheelchair into the spokes and not using one side of the body despite the return of muscle strength and sensation. The biggest hazard associated with this problem is the possibility of frequent injury to body parts. The caregiver should give frequent reminders of the ignored side by touching it, talking about it, asking the stroke survivor to Afind@ it or rub it gently with a towel. Persons having a conversation with the stroke survivor should stand in front or to the unaffected side. The stroke survivor should wear bright visual reminders on the affected side, such as scarf or a bracelet, a piece of tape on the shoe etc. It can also be helpful to wear one's watch on the affected arm, as a reminder to look for that arm.

Hemianopia : A person suffering from this disorder may run into objects on the affected side, may see and eat only half the food on the plate, or may not see someone on the affected side. The stroke survivor has to be taught compensatory techniques such as turning the head to see the whole picture and scanning the environment frequently.

Training for activities of daily living

The ADL spectrum of functional parameters has received a considerable amount of study and is the best known and most standardised set of outcome variables in stroke. The most popular way to measure independence in ADL is to classify the patient according to Barthel index. A maximum score of 100 on this index means that the individual can get along without attendent care. Patients are taught how to clean their face, teeth, and body with the normal arm and how to dress. Patients with right hemiparesis are taught to use the left hand to eat. Goals for rehabilitation must be set in keeping with the reality of the patient's physical disability. With hemiplegia, the best that can be hoped for is the ability to regain some degree of ambulation and participation

with help in performing daily activities. With improvement, expectations can rise and for those with minimal or mild weakness return to normal activities can be expected.

Long term rehabilitation

Even though most marked improvement is achieved during the first three months, rehabilitation should be continued for a longer period to prevent subsequent deterioration. As the ultimate aim is to enable the patient to return to his routine life, it is essential to evaluate the patient for his or her ability to carry out activities of daily living (ADL). There are several methods used to determine a numerical value or score for ADL. One of the most widely used scoring systems is Barthel index. Each of these daily activities is given a score depending on patient's ability to perform the activity. A score of 100 in the Barthel index indicates functional independence and a score of 0 indicates total dependence. Patients with scores under 40 at discharge after rehabilitation program rarely are independent. For those with higher scores, there is increasing independence. If the scores plateau and show no evidence of further improvement over several weeks, it is unlikely that further efforts in a rehabilitation program will be worthwhile.

Self -help devices and modification of home environment : When a patient with stroke has reached maximal improvement on a rehabilitaton program and is left with disability, independence at home can be increased by a number of self help devices and modification of the home environment. Self-help devices are largely designed to aid the stroke victim who has only one functioning hand. The patient's functional capacity should be assessed, and the home should be investigated to see what modifications might be possible to make it easier for the patient to function. Assistive devices for grooming include a long-handled bath brush, long handled shoe horn, buttoning aids, Velcro instead of laces for shoes, flexible showerhead extendors and large handled hair brush and comb. Bathroom modification is always necessary, including an elevated toilet seat, grab bars on each side of toilet, or a movable chair commode. Self help devices may be among the most effective means of attaining self care.

Urine collecting devices : Sadly, there are some stroke survivors who continue to have incontinence despite all attempts at retraining. This may occur if the stroke damage is extensive or if cognition is severely impaired. At times, the only solution is the use of an indwelling Foley catheter. This is indicated when constant wetness causes skin irritation or breakdown. For men, there are external condom catheters that attach to a leg bag for day use and a bed bag for night use. Condom catheters should be changed daily,

and the skin should be washed and dried thoroughly before reapplying the catheter. For women, disposable pant-liners, waterproof underpants, and disposable adult diapers are commercially available. If these devices are used, careful cleansing and lubrication of the skin is required.

Exercises : As far as possible, all stroke survivors should exercise which are appropriate to the patient's medical status and level of ability. The exercise program should be designed by physical and occupational therapists who are trained to develop programs based on a person's level of ability, endurance and lifestyle. Even after formal rehabilitation is completed, the need to do daily exercises at home will continue for most stroke patients indefinitely.

Newer strategies

An unusual approach to motor retraining is the forced use of a paretic extremity.[14] This strategy is based on the theory that learned non-use of a paretic limb contributes to lack of recovery. Placement of the unaffected arm in a sling for two weeks has been shown to improve speed and quality of movement in the affected hand of selected patients, even when treatment begins over a year after stroke. Gains are maintained in some patients during follow up periods of a year or longer. Another interesting approach to promote motor recovery consists of treadmill training with partial weight bearing. As patients improve, less weight support is provided and treadmill support is increased. In a small series of nonambulatory patients more than three months post stroke who had failed comprehensive therapy program most regained independent ambulation. Other training techniques have included weight bearing exercises involving the upper and lower extremities, functional electrical stimulation (FES), biofeedback, and vibration and other sensory modalities.[15] Functional electric stimulation (FES) is a non-specific term applied to variety of treatments involving stimulation of muscles and nerves. Several studies using FES, sometimes combined with biofeedback or conventional physical therapy, have shown increased improvement in functional motor activities although improvements have been modest.[16] FES has also been used as a sort of a electrical orthosis to improve ankle dorsiflexion during the swing phase of gait in patients with hemiparesis.[17] Complex microprocessor-based multichannel stimulation using implanted intramuscular electrodes has been used with limited success but remains controversial.[18] Electromyographic(EMG) biofeedback to facilitate movement of paretic muscles, often used in conjunction with traditional therapy programs, has been shown to be modestly beneficial in some studies.[19] An assessment of EMG biofeedback using a meta-analysis

of randomised studies between 1966 and 1991 suggested that biofeedback is effective for neuromuscular re-education for stroke patients.[20] Thaut and co-workers demonstrated improved weight-bearing stance time on the paretic side and improved stride rhythmicity when stroke patients walked to rhythmic music.[21] Various forms of sensory stimulation have been suggested to enhance motor recovery. Rapid skin brushing and application of vibration are sometimes used in association with therapy sessions to induce movement in paretic limbs[22] but whether long range benefit is produced remains unclear. Acupuncture treatment accompanied by electrical stimulation have been shown to improve balance , mobility skills, and ability to perform ADL's with sustained improvements for up to one year.[23,24] The investigators hypothesized that acupuncture-induced sensory stimulation promoted neuronal integration at the cortical level. The use of drugs to promote motor recovery may become increasingly important in the future. Because amphetamine improves motor function in animals[25], a similar beneficial effect may be seen in stroke patients. Although one pilot study reported favorable results[26], further trials are needed. More importantly perhaps, many commonly used drugs like benzodiazepines, phenytoin and haloperidol may impede motor recovery in laborotory animals and humans.[27] A retrospective study indicated that these drugs are frequently prescribed in patients with stroke.[28] Spasticity frequently accompanies hemiparesis but is seldom the limiting factor in motor recovery. When spasticity is accompanied by mass spasms, severe clonus, pain, or contractures, treatment is indicated. Simple physical measures such as prolonged static stretching, passive range of motion or splinting should always be atempted first. Further dantrolene, diazepam and baclofen may be tried.[29] For spasticity in an isolated group of muscles, local injection of botulinum toxin is under active investigation.[30]

Conclusion:

There is a general consensus that in many stroke survivors rehabilitation improves the long term outcome and quality of life. Unfortunately, adequate facilities for inpatient rehabilitation do not exist in India nor is domiciliary treatment practicable due to lack of qualified personnel. The rehabilitation team should make a thorough assessment to select the patients most likely to benefit from a rehabilitation programme who should be kept admitted till they reach the plateau of progress. Patients can later visit the hospital at regular intervals. There is also a great need for the formation of stroke clubs at community levels. This has to be achieved by active awareness campaigns by physicians, nurses, physiotherapists and the interested members of community.

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Traumatic Brain Injury

B. Indira Devi

Introduction

Traumatic Brain Injury (TBI) is a leading cause of short and long term morbidity and mortality. With rapid growth of population, without adequate infrastructure and safe transport, the problem is on the increase. Most patients of TBI have a protracted course of rehabilitation. It is the neurobehavioural outcome, rather than the neurological deficits which are really taxing to the patient and the family.

The victims of road accidents are motor vehicle occupants (51%), pedestrians 28% and motor cyclists [1]. Introduction of helmets and seat belts has reduced the occurrence of intracranial haematomas, concession and skull fractures [2]. Better roads, safe driving and mass education about pedestrian ways may reduce the road accidents. An analysis of 1119 patients with TBI admitted to the University of Edinburgh, Scotland during 1981, showed that severe injuries were caused by Road traffic accidents [3].In India, children are injured equally frequently by fall from height and road traffic accidents. It has been suggested that alcohol may potentiate brain damage after TBI. Studies by Nath et al [4] has been inconclusive. It is generally agreed that prompt and correct early management of TBI patient reduces the secondary insults to the brain which directly contribute to the residual deficits. Though rehabilitation of TBI patients starts in the acute phase, soon after the resuscitation, it may continue for months and years and some times life long.

Pathology of closed TBI

One of the major changes in understanding the closed head injury has been the concept of Diffuse Axonal Injury. Wide spread damage to the brain due to primary injury perse, not due to secondary insults of herniation or perfusion deficits , is now widely accepted. Though first named so by Adams [3], it was described by Strich [4]. There is microscopic axonal swelling due to retraction .Grossly there are haemorrhagic and necrotic lesions of corpus callosum, Dorsolateral quadrant of rostral pons. Long term conginitive deficits may also result from acute subdural haematomas, contusions and intracerebral haematomas.

Early Assessment and outcome

A survey of the available literature shows that there is lack of uniformity in defining the terms involved in morbidity or disability. Norsell found that awareness depends on the normal activity in the cerebral cortex [5]. But the relationship between cortical activity and awareness is found to be conditional. Wakefulness is probably best used to indicate the level of reactivity and the term arousal is probably best used for changes in reacting. Signs of wakefulness and arousal do not indicate awareness by the individual of a situation or condition. Various coma scales have been recommended for defining the consciousness level. The Glasgow Coma Scale (GCS) is at present the most widely used and accepted scale (Table 1). It is perhaps inadequate and insensitive for monitoring patients who are likely to deteriorate. Traiging is very important in the early management. Patients with TBI can be categorised into 4 grades (Table 2)

Table 1 Glasgow Coma Scale

Eye opening (E)
4- Opens eyes spontaneously
3- Opens eyes to voice
2- opens eyes to pain
1- No eye opening
Best Motor Response - M
6- obeys commands
5- Localizes to pain
4- Withdraws to pain
3- Abnormal Flexor response
2- Abnormal extensor response
1 - No movement
Best verbal response-V
5-Appropriate and Oriented
4-Confused conversations
3- Inappropriate words
2- Incomprehensible words
1- No sound

Epidemiological studies show that incidence of TBI is 200/100,000. Fifteen percent die before reaching the hospital. Among the patients reaching hospital, 10% have severe TBI, 10% have moderate TBI and 80% have minor TBI. Of the moderately injured patients, 7% die in hospital, 67% required acute rehabilitation services. About 10% severely head injured remains vegetative.

Table 2 Classification of patients with TBI

Category	Description
Grade - I	Transient loss of consciousness (<5 min) now alert oriented without neurological deficit. GCS 14-15
Grade - II	Previous loss of consciousness (<5 min) now impaired consciousness but able to follow at least a simple command no other neurological deficits GCS 9-13
Grade - III	Previously unresponsive (<5 min) now not following even a simple command. Pupils unequal inappropriate words. GCS< 9.
Grade - IV	No evidence of brain function (brain death).

Diaschisis and recovery:

Following TBI recovery of neurologic function may occur without actual neuronal regeneration. A patient with monoplegia may learn to walk again without any recovery of motor function in the paralysed limb. Functional recovery vs necrologic recovery may be difficult to distinguish but such a distinction is useful because Pharmacologic intervention may influence both. Von Monakow proposed the term diaschisis to explain temporary or permanent dormancy of function of associated areas after localised brain damage. The necrologic deficit seen after brain injury may be contributed by dormancy of associated parts of the brain. With time, this dormancy may be overcome and result in functional or necrologic recovery. Dendritic sprouting both collateral and regenerative may occur in course of time and contribute to recovery. The present state of understanding the neural recovery (necrologic or functional) is incomplete.

Prognostic indicators

The overall objective of TBI care is to minimize the occupance of avoidable mortality and morbidity and most avoidable mortality is due to delayed diagnosis and management of intracranial haematoma or overlooking of systemic extracranial events. As the salvage rate of severe TBI improves and the resources become more limited, ethical and scientific issues will be redefined. It is desirable to predict outcome so that further management can be planned. Mild and moderate disability can perhaps be avoided by proper management. Two factors of greatest significance in determining outcome are severity of injury and age of patient

The GCS is reduced proportionately by shock or hypoxia. The predictive power of GCS is such that the acute influence of shock and hypoxia on neurological function is incorporated into a priori. Patients with a GCS of 3-5 has mortality rates in excess of 60%. The addition of brainstem function tests to the GCS does not change the initial prediction. Development of pupillary abnormality in the presence of preserved brainstem functions worsens the prognosis. A mortality of 100% is observed when a systolic blood pressure of less than 95 mmHg ,pao2 of less than 65 mmHg, GCS of less than 7 and an intracranial pressure more than 30mmHg. Bradycardia of less than 50 per minute at admission is associated with increased likelihood of death severe disability. A patient with mass lesion has a poor outcome than the one with diffuse swelling. A CT scan showing a midline of more than 10mm, absent basal cisterns, small ventricles are also predictors of poor outcome[9,10] .

Jennet suggested that too much attention has probably been paid to very severely injured patients both in acute stage and during rehabilitation[11]. Physicians and therapists involved in rehabilitation are subject to pressures from family and friends and patient regarding the establishment of prognosis, need for long term rehabilitation, prolonged coma management and decision to treat or not to treat at various stages. Survivors of the TBI make variable recovery over a variable period. Some of the patients are left with sequelae 12-15 and need long term medical and rehabilitation services.

Post traumatic epilepsy

Incidence of epilepsy following TBI is variable and depends upon a number of factors. Early post traumatic epilepsy is defined as one or more seizures occurring within one week of head injury. About 25% of patients with early post traumatic epilepsy have

late seizures. Intradural mass lesions have 30-36% incidence of early epilepsy. Occurrence of early epilepsy complicate management of head injured patients. Not only it necessitates CT scans to evaluate the patient's neurological condition, it also may cause aspiration pneumonia. A mild injury with early epilepsy has 25% chance of late seizures.

Late post traumatic epilepsy occurs one week after injury. It causes significant disability. It also causes severe medical, economic, social and psychological consequences complicating rehabilitation. It lessens chance for gainful employment after rehabilitation. There is no effective prophylaxis for post traumatic epilepsy . Since late post traumatic seizures diminish with time, surgery is not indicated till 2 years after injury.

Post concussion syndrome:

This refers to a variety of symptoms such as headache , dizziness, forgetfulness, anxiety and impaired concentration following minor head injury. There are no neurological signs. The headaches are characterised by their variability. The symptoms lasting only a few days or weeks are due to local injury or subtle structural brain injury. Psychological factors play more important role in longer lasting headaches. Most patients need reassurance and encouragement to get over the problem. Psychiatric assistance is required when the symptoms last longer than 2-3 months. Other psychiatric problems seen in these patients are alcohol abuse, psychosis, mania, depression, sexual deviations and sleep disorders.

Cognitive impairment

Glasgow outcome scale is used to grade overall recovery and adjustment to daily life after TBI. However quality of intellectual and psychological recovery has not been qualified in many of the studies. In a study by Rimel among patients of moderate head injury with a GCS of 9-12 of 6 hours after admission, 90% had impaired memory and at 3 months only 31% were employed[18,19].For the majority of TBI patients improvement of intelligence does not play a role of practical importance. Temporary intellectual impairment may be due to increased mental fatigue and slowness. Permanent impairment is found in patients with very severe TBI. On the Wechsler Adult Intelligence Scale (WAIS)[17], performance test show more of the deficits than the verbal tests. A post traumatic amnesia of 5 weeks or more results in more permanent intellectual impairment. Though individual cases show variation in rate of recovery, greatest improvement occurs soon after injury. In severely injured recovery may not begin for 5-7 months.

The Neuropsychological assessment identify the cognitive deficits. The cognitive retraing attempts to overcome these deficits. The crucial first step is creation of insight about the impairments in the patients. The compensatory approach try to circumvent the effects of cognitive impairments in daily activities. If possible a spared function is used to compensate for a impaired skill. The patients are specifically trained for the desired functions. The environment is kept structured .The cognitive remediation is a cognitive process specific approach. The therapist targets distinct theoretical components of a function. The tasks are attempted in a hierarchial manner. However learning a task in the therapy setting may not generalize to other circumstances or other related tasks. Computers may be used for cognitive retraining. It allows accurate serial measurements. The computer helps in keeping the test situations constant. The tests, scores and cues will be consistent. The task difficulty can be set based on the patients performance. The computer assisted programmes help to perform therapy with only intermittent supervision.

Memory impairment

Loss of memory is a core symptom of TBI and show variable recovery. Two types of amnesia are usually recognised viz retrograde and anterograde amnesia. Memory may show greatest improvement after 4-6 months, the residual impairment is of forgetfulness

Retrograde amnesia : In this the patient cannot remember the events that occurred during the period preceding the accident. There is some evidence to suggest that the memory is irretrievable. Some of the events can be recalled when the patient recovers. Retrograde amnesia is probably not of practical importance, except that more severe the injury results in longer lasting retrograde amnesia.

Posttraumatic amnesia : When a patient regains consciousness after TBI, he may be unable to record new information. Brooks[13] has defined the post traumatic amnesia as the interval between injury and regaining continuous day to day memory with intact orientation. In assessing the post traumatic amnesia in a retrospective way, one must be aware of two pitfalls. One is that the patient maybe unconscious for a while and will not have any memories of this period. The second pit fall is the " Island of Memory". An island of memory is said to be present when a patient reports a single trivial incident even though he may not have full memory. Presence of post traumatic amnesia also indicates the severity of injury. The relative inability to record events or information given to the patient is called as anterograde amnesia. The digit span is not a valid test of memory. Many TBI patients obtain near normal scores even in the presence of amnesias

Interventions for amnesia include internal strategies and external reminders. The internal strategies include rehearsal , mental retracing, visual imagery ,mnemonics and associations. The external reminders include: Time reminders: Alarm clock, telephone, organisers, diary, wall planner and calender, and Person reminders: Name tags, dressing patterns, uniforms, Place reminders: Labels, codes, colours and symbols and Other reminders: Lists, tape recorders, pagers and care givers and relatives.

Behavioural changes

Changes in personality and social behaviour are common among survivors of TBI. Injury to subfrontal and anterior temporal region produces disinhibition, lack of insight, childishness, apathy and inertia. Neurobehavioural impairment is usual in patients with moderate and severe TBI. Even in patients with moderate TBI and post traumatic amnesia exceeding 48 hours , irritability agitation and anger was reported at 12 months. In severe TBI the changes may remain permanently. The cognitive impairment and personality changes contribute to the negative social integration of the patient One of the late sequelae is social isolation.

The interventions for behavioural problems after TBI include search and treat precipitating and aggravating problems like pain, urinary retention and seizures, reduce drugs causing confusion, behavioural modification , Psychotherapy , antipsychotics and short acting tranquilizers. The behaviour modification include identification of target behaviour, reinforcement of the desired behaviour with rewards, token economy and supportive and non threatening therapy sessions. The Psychotherapy sessions attempt to improve awareness and insight , accept the disabilities , improve motivation , set realistic goals and regain self control. The drugs used for the treatment of aggression include anticonvulsants, antidepressants, beta blockers, lithium, neuroleptic, benzodiazepines, clonidine, verapamil, d-amphetamine, methyl phenadiate and pemoline.

Neuropharmacology

Drugs which are said to augment neurologic recovery, probably do so by improving attention and thereby learning a target oriented task. These drugs may also help resolve diaschisis and help activate latent cerebral circuitry. Some of the agents used with variable success include amphetamine , dopaminergic agents and gangliosides.On the other hand drugs which adversely influence functional outcome may do so by increasing transsynaptic neural cell death, by inhibiting sprouting of new path ways and neural connections[18].

Common drugs alleged to have adverse effects include benzodiazepines, barbiturates ,major tranquilizers and antihypertensives. Use of pharmacological manipulations of necrologic outcome is more of an art than science. Drugs which cause adverse outcome can be avoided or used only when really required. Use of pharmacological manipulations of necrologic outcome is more of an art than science. Drugs which cause adverse outcome can be avoided or used only when really required.

Conclusions

In spite of vast advances in understanding TBI , the mechanisms of recovery and outcome are still uncertain. Though the present picture is not dismal the best alternative remains prevention.

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Rehabilitation of Spinal cord injuries

B. P. Gardner

Introduction

Prior to World War II most spinal cord injured patients succumbed soon after injury. Those who survived were often institutionalised, their lives impoverished and frequently bedridden. The establishment of systems of comprehensive care, pioneered by the National Spinal Injuries Centre in Stoke Mandeville Hospital, dramatically altered this situation. [1,2].

Spinal cord injury affects every system of the body. Successful rehabilitation depends on the effective management of all aspects. Failure of care in any area results in unnecessary morbidity and morality. Following spinal cord injury there is a complex interaction within a multi-system disorder. The greatest threat to the successful rehabilitation of the patient is fragmentation of case. To avoid this, systems of care were developed, first in the United Kingdom and later in Australia, New Zealand, the United States of America and Canada, where all aspects of treatment are addressed. In the former three all facets of medical care following the Accident and Emergency Department phase are dealt with in one centre. In the latter the systems involve acute care in one hospital followed by subacute and chronic care elsewhere, resulting in some adverse consequences when compared with the unified system but overall resulting in much improved outcomes compared with care that is either fragmented or carried out by those lacing the required insights and training .[3,4]

In the United Kingdom of Great Britain and Northern Ireland the Clinical Service Specifications of Modern Spinal Cord have been laid out. They are listed in the Concluding Section of this Chapter. A multi-disciplinary approach is essential if the optimum rehabilitation outcome is to be achieved.

Definitions

A spinal cord injury is complete if there is no somatic motor or sensory function below the level of injury. If the arms are spared the patient has paraplegia. If they are involved he has tetraplegia. 5 The level of injury is the lowest intact spinal cord segment. 2 If

there is residual function several segments below this then the injury is incomplete and the patient has either paraparesis or tetrparesis. Use of the terms quadriplegia and quadriparesis should be avoided.

Epidemiology

The incidence of traumatic spinal cord injury varies in different countries and series between 10 and 50 per million in the population each year. [6] The figure in the UK is towards the lower end of this range.

Prevention

The two components relevant to rehabilitation are first the prevention of the injury itself and second the avoidance of the secondary deleterious effects that are the consequence of poor care.

Spinal cord injury is most commonly caused by motor vehicle accidents. Seat belts, both front and rear, side impact support systems and inflation bags reduce the incidence and severity of injury. Sports related injuries are uncommon but devastating. In the United Kingdom the commonest sporting causes of spinal injury are, in order of frequency, diving, rugby and horse-riding. Diving related injuries can be prevented in part by good education, appropriate pool design, adequate poolside signs and appropriate supervision. Pool attendants should be trained in safe methods of retrieval. Rugby injuries can be reduced by adherence to the rules of the game, the avoidance of participants playing out of position and ensuring that players are suitably fit.

Avoidance of the secondary deleterious effects that are a consequence of poor care is dependent first on the recognition that a spinal injury has, or may have, occurred and second on knowledge of an expertise in the correct actions to take. It is all too common for spinal injuries to be missed with consequent deleterious effects on the spinal cord and impaired rehabilitation outcome. Toscano showed that the State of Victoria in Australia, 26% of spinal cord injured patients sustained major neurological deterioration between the times of injury and of admission into the Austin Hospital Spinal Unit in Melbourne. The site of the major neurological deterioration was the accident site in 9.4%, the initial ambulance assessment and ambulance transport to the local hospital in 28.1%, the local hospital in 53.1% and the transport to the spinal unit in 6.3%.7 The more the spinal cord is damaged the less complete is its recovery and the worse the rehabilitation outcome.

Clinical management

Optimum rehabilitation outcome depends on good management of the many facets that affect the spinal cord injured person. Accurate diagnosis is essential. Rehabilitation outcome is adversely affected when spinal fractures are missed. Common sites where fractures are overlooked include the cervicodorsal junction and spinal fractures below the major one. Adequate radiological evaluation may not be possible immediately after the accident. If there are reasonable grounds for believing that the patient has sustained an unstable spinal injury then appropriate steps must be taken to immobilise the spine until such time as the diagnosis can be confirmed of refuted.[8].

1. Management of associated injuries

Associated injuries must be well treated ensure optimum rehabilitation outcome. Amongst the more important are the following:

a. Brain : Successful rehabilitation following spinal cord injury is dependent on the total involvement of the disabled person. Impairment of personality, memory, concentration and intellect can profoundly alter outcome. Good executive function is of particular importance in enabling the spinal cord injured person to lead a safe and well-integrated life. Relatively minor degrees of higher cerebral impairment can interact with the other problems associated with spinal cord injury to make safe independent living and successful employment much more difficult.

b. Limb joints and bones : Spinal cord damaged persons are more dependent on their arms than prior to injury. Joint damage, and to a lesser extent long bone fractures, can severely impair transfers and wheelchair skills. Contractures and frequently very disabling. Because arm joints, especially the shoulders, are put under stress by the routine activities of wheelchair life, problems commence in them at an earlier age. The onset of these difficulties is accelerated by damage sustained at the time injury.

c. Peripheral nerve injuries, especially brachial plexus : Peripheral nerve and brachial plexus injuries occur in association with the spinal cord injury. Paraplegics require both arms for most activities. The affected arm cannot cope so well with transfers and wheelchair control. The functional impact can be reduced by trick movements that may take years to develop.

d. Chest and abdominal : Chest and abdominal injuries, through life threatening at the time of the original event, are seldom important in rehabilitation terms as they do not often result in an increased requirement for care or equipment.

2. Neurology

The level, degree of completeness and pattern of the spinal cord injury are of central importance in determining rehabilitation outcome and prognosis.[9,10,11] There is no level of neurological disability, including ventilator dependency, that is incompatible with life in the community.[12]

Whilst poor spinal and general care can cause neurological deterioration, the only specific treatment for the spinal cord in the acute phase which may produce a positive rehabilitation outcome is methylprednisolone.[13] Opinion is divided as to whether or not the harmful effects of the very high doses of steroids required outweigh the relatively small benefits.

Incomplete spinal cord injuries are associated with the lessened risk to life. Preserved sensation enables the paralysed person to become aware of complications as they arise below the level of injury. Complete spinal cord injured persons also learn to recognise signals coming from the paralysed and denervated parts of the body but these are less precise. It is not just pressure sores but also other complications such as intra-abdominal events and long bone fractures which are recognised in this way.

Neurological level

The neurological level injury is the most important determinant of rehabilitation outcome. Each segmental level in the cervical region in particular is of vital importance. Patients with complete lesions at C3 and above require a greater or lesser degree of ventilatory support, such as intermittent positive pressure ventilation and phrenic nerve pacing. Less widely used techniques include intercosto-phrenic nerve anastamosis and artificial ventilation by mouth. C4 level patients can almost always breathe independently but are otherwise almost totally dependent. Electric wheelchair mobility and control of the environment via the Possum and other systems is achievable using retained head and neck control. C5 level patients have good shoulder control as well as elbow flexion. With aids, such as feeding straps, limited function is possible. Assistance is still required with every activity.

C6 level patients have good wrist dorsiflexion. Elbow extension can be achieved by means of trick movements. By locking the elbow, transfers are sometimes possible. Wrist dorsiflexion is associated with passive tenodesis of the fingers and the thumb. Upper limb reconstructive procedures can be of great benefit at this level of injury. Active elbow extension can be achieved by the Moberg posterior deltoid to triceps transfer operation. A stronger and more active key grip can be achieved by tendon transfers around the wrist, such as insertion of the extensor carpi radialis longus into flexor digitorum profundus and the brachio-radialis into flexor pollicis longus. These procedures do not usually increase transfer capability but they do improve upper limb control and so lead an improved quality of life. [14] C7 and C8 level patients lack fine intrinsic hand muscle control but have sufficient upper limb function to achieve some independence in transfers and activities of daily living.

Upper thoracic, T2 to T6, level patients lack the abdominal and lower paraspinal muscle control that is essential to achieve good truncal balance. Backwheel balance control and transfers are impaired as a result. Spontaneous spasms are likely to cause problems in transfers. Ambulation in long leg calipers is difficult. Braces that stabilise the upper body, such as the reciprocating and hip guidance orthoses, are usually required if ambulation is to achieved.

Lower thoracic, T7 to T12, patients have greater abdominal and paraspinal muscle control and hence better truncal balance. Higher kerbs can be negotiated because better backwheel balance are possible.

L1 level patients frequently achieve ambulation though this is seldom of functional benefit. The good quadriceps control of the mid-lumbar level person usually allows functional ambulation in younger patients.

Longer term neurological consequences.

In recent years it has become clear that the incidence of tertiary spinal cord change is much commoner than had previously been recognised. These changes continue to develop throughout the life of the spinal cord injured person. The most important is the spinal cord syrinx. The previously quoted incidence of syrnix formation of 2 and 4 per cent was largely based on clinical diagnosis. It is now clear that the incidence of syrnix is much greater than this because the majority do not have diagnostic clinical features. In a recent study in Stoke Mandeville Hospital of 153 patients whose spinal cord injury

was injured more than 20 years ago, the overall incidence of syrnix formation was 20 per cent. The longer the patient is injured the more likely he is to have syrnix.[15]

The presence of syrnix has important consequences for rehabilitation. If a person has a spinal cord syrnix then he should alter his lifestyle so as to avoid those abrupt stresses, strains and other events that could cause serious spinal cord deterioration. Falling from the wheelchair in a poorly executed transfer for example can be associated with loss of the use of a hand or an arm. A patient with a spinal cord syrnix needs a greater degree of care assistance because he must avoid the risks associated with syrnix deterioration, such as falls during transfers.

The aetiology and management of syrnixes remains controversial.[16] Surgery is not usually required continued review is essential a neurosurgeon with a specialist interest in the spinal cord injured is an essential member of the mutlidisciplinary team.

3. Spine

Following the acute event spinal problems are not usually an issue. Arthritis may occur at an earlier stage in the intact spinal joints above and below the injured segment. This can give rise to increased spinal pain and stiffness in older years. The contributes to the greater dependence that arises with aging. Deformities such as gibbus are seldom functionally important.

Long spinal fixations can be very disabling. A young person with paraplegia and a long fixation is usually totally independent in his younger years but when he is older his loss of truncal mobility cannot be so readily compensated for by increased movement in his hips. This brings forward the stage at which his dependence increases. Long fixations in the cervical region prevent the tetraplegic from looking around himself, making driving a care more difficult.Around 10 per cent of spinal injured patients have fractures at multiple levels. Those below the level of the main injury are important if they cause neurological damage or scoliosis. For example, a complete cervical spinal cord injured person with an L1 fracture must have the latter carefully treated if important reflex bladder, bowel and sexual functions are to be retained.

Progressive skeletal deformity is a particular problem in children. Regular careful review of their spinal position is required unit skeletal maturity. Whereas gibbus does not significantly increase disability, scoliosis can be a significant problem. Sitting posture,

the pattern of pressure on the ischial areas and transfers are all impaired by scoliosis. An orthopedic surgeon with a specialist interest in the spinal cord injured person is an essential member of the multidisciplinary team.

4. Pain

Musculo-skeletal and neruogenic pains are common following spinal cord injury. They can be intractably disabling. Treatment is frequently difficult.[17] Sometimes the pain makes it necessary for patients to shift from one position to another or to lie down at intervals during the day. Employment can be difficult for this reason and also because of the effect on concentration of the pain itself and its associated medication. An anaesthetist with a specialist interest in the spinal cord injured person is an essential member of the multidisciplinay team. Neurosurgical intervention is rarely required. Behavioural approaches usually offer the best prospect for the patient learning to cope with his pain.

5. Bladder

a. Lower Urinary Tract: Bladder sensation and control are impaired by spinal cord injury. The precise pattern of bladder management varies with the individual.[18] All methods of bladder care are associated with events that can be distressing and inconvenient. With intermittent self catheterisation there is incontinence and toilets are frequently inaccessible. With automatic drainage the urinary sheath occasionally comes of causing the patient to become soaked and minor penile problems prevent application of the sheath forcing the patient to remain in bed or to insert an indwelling catheter.

When the partial bladder control remains there is often a degree of urgency and frequency that seriously impairs the patient's quality of life, for example by forcing him to plan his journey according to the location of accessible toilets. Oxybutinin may help in these cases. Bladder management in females in particularly difficult. There are no satisfactory external collecting appliances. The risk of incontinence and the awareness that there may be a smell of urine impairs self-confidence and femininity.

A variety of urological procedures exist that benefit certain groups of patients. The more commonly used included augmentation cystoplasty, distal urethral sphincterotomy, the artificial urinary sphincter and the Brindley sacral anterior root stimulator. The latter is of particular benefit in females.[19]

Many patients elect to have indwelling suprapubic or urethral catheters. Although associated with increased risk because of the inevitable infection, the quality of life of the patients is often improved by this method. Indwelling bladder catheter associated problems include bladder stones, intravescial bladder changes, uretheral discharges and the problems associated with catheter blockage, especially autonomic dysreflexia.

b. Upper Urinary Tract: Continued vigilance of the upper urinary tract is required throughout the life of the paralysed person. [18] Asymptomatic upper tract problems such as calculi and dilatation can occur. The pattern of review that is required varies with the individual. An annual evaluation will usually suffice to ensure early diagnosis and treatment before problems arise. Improved urological techniques, such as percuatneous and whole body lithotripsy, have reduced the morbidity of upper tract stones.

Experienced nurses are important sources of advice and help with incontinence aids and appliances. A urological surgeon with a specialist interest in the spinal cord injured person is an essential member of the multidisciplinary team.

6. Bowels

Upper gastrointestinal problems are seldom significant. Faecal evacuation by contrast is usually a major problem. Most patients require suppositories or digital stimulation. Some require aperients. A disciplined pattern of bowel control is essential. Episodes of incontinence occur and can be very distressing. They are minimised by attention to discipline and the avoidance of precipitating factors such as hot curries and similar foods.

Most paraplegics are able to mange their bowels by transferring onto the toilet followed by suppository insertion of digital evacuation. The rectum needs to be checked after bowel emptying to ensure that no faeces remain. Most tetraplegic need a greater or lesser degree of assistance. Bowel evacuation whilst seated on a shower chair over the toilet and followed by a shower at the end of evacuation is a commonly adopted pattern. After the shower the patient dried. He then has his top half dressed whilst still seated on the shower chair and his bottom half dressed after onto the bed.
Bowel problems are common in chronic spinal cord injury. [20] Faecal evacuation may take a progressively longer time. Aperients become less effective. The life of the paralysed person can be greatly disrupted. Nurses are the key team members advising on bowel care following spinal cord injury. An active peripatetic nursing service that offers

telephone advice can be a welcome source of continued support to patients in this very difficult and under-researched area.

7. Joints

Wear and tear on upper limb joints is increased. As paraplegic patients get older, episodes of upper limb joint pain and stiffness occur with increasing frequency, especially in the shoulder girdle.

Heterotopic ossification can occur in the early stage following injury. [21] Hip mobility can be severely impaired. Transfers and activities of daily living become more difficult. The ossification process eventually becomes quiescent. Surgery is rarely required. It should only be undertaken after ascertaining that there is no residual bony activity. There is a small place for radiotherapy immediately following excision of the abnormal tissue.

Contractures interfere with independent living, effective mobility and transfers. They give rise to pain and disability. In tetraplegics, contractures of the shoulders, elbows and wrists are a particular problem. In paraplegics, lower limb contractures frequently prevent ambulation and interfere with transfers.

Therapy to joint is essential at all stages following spinal cord injury, and especially in the acute phase. Splinting of hands and correct positioning of shoulders and elbows can prevent unnecessary upper limb morbidity. Physiotherapy and occupational therapy staff should establish the treatments which can then be continued by the patient, carers and family.

In the chronic spinal cord injured persons long bone fractures can occur following relatively minor trauma, such as the leg twisting off the foot-plate of the wheelchair. Simple splinting, such as with a Robert-Jones bandage often suffices. Internal fixation often fails because of the inadequacy of the osteoporotic bones and the development of pressure sores that may prove very difficult to heal.

8. Spasms

Spasms and spasticity are usual accompaniments of spinal cord injury. They are sometimes helpful but more usually a hindrance. They cause embarrassment when out

of doors. They can be dangerous if they occur abruptly during a transfer or when driving. The sleep of both the paralysed person and the partner is disturbed. The spasms may throw the legs out of position in bed. Treatment of spasms includes eradication of any precipitating cause, in particular intravescial and bowel related pathology, good physiotherapy including standing, systemic drugs such as Baclofen and Dantrium, and, in rare circumstances, operative intervention such as insertion of the intrathecal Baclofen infusion system. [22]

Systemic medication for spasticity has adverse effects. Baclofen causes drowsiness and interferes with concentration. This has implications for quality of life and employment. The intrathecal drug delivery systems have potential complications that can be serious. Delivery tube dislodgment and kinking occurs necessitating revision. Pump replacement is sometimes necessary, in particular with the battery driven types. For the management of spasticity to be effective there must be expertise in the spinal cord injury centre in all relevant physiotherapeutic, medical and surgical techniques. Intrathecal pump insertions should only be carried out by those with experience.

9. Respiratory

Permanent ventilator dependent patients can live safely in the community provided that they have sufficient care. [12] A trained carer must at all times be "in-line-of-eye" of the ventilated person. This carer must be able to carry out tracheal suctioning, tracheostomy replacement, ventilator reconnection and lug bagging. Alarms to summon help immediately are required. With a portable ventilator, supplemented where appropriate by the phrenic pacemaker and other systems, free movement out of door and including aircraft travel can readily be achieved. High tetraplegic ventilated patients' value their lives even though these are impoverished in physical terms. [23]

Mid and low cervical patients have good diaphragmatic control but no intercostal or abdominal muscle function. Their cough is weak and may need to be assisted. Physiotherapy may be required during chest infections. Respiratory impairment is the most important increases risk to life in tetraplegics. Carers need to be carefully instructed in the relief of choking, the assisted cough, postural drainage of the chest and clearance of secretion. [24]Mid-thoracic paraplegics lack a good cough because their abdominal muscle control is absent. They require help with chest infections in their older years.

For the management of respiratory problems to be fully effective there must be a chest physician available who has a special interest in the respiratory problems of the spinal

cord injured. Expert anesthetic support is required to make domiciliary ventilation as safe as possible.

10. Cardiovascular

Postural hypotension is a common problem in the early stage following spinal cord injury. It is seldom disabling thereafter though tetraplegics may require occasional assistance with being tilted back when hypotension occurs.[25] Autonomic dysreflexia is a serious problem in all patients with injuries at T6 and above.[26] It can be precipitated by any stimulus arising below the level of injury. The most common are those from the bladder and the bowels. Some events, such as rectal electrostimulated semen emission and vibrator induced ejaculation, are particularly virulent stimuli. During autonomic dysreflexic episodes the arterial blood pressure can rise to dangerously high levels. Cardiac dysrhythmias may occur. Patients describe that their heads are bursting open with pain. Their sweating may be so profuse that a change of clothes or bedding in necessary. Because tetraplegics cannot deal with the factors that precipitate autonomic dysreflexia, care support needs to be available to ensure that should an attack occur it is dealt with promptly and safely. It is essential that staff, patients, family and carers are fully conversant with the diagnosis and treatment of this unpleasant and dangerous complication.

In spite of immobility and leg dependency, deep venous thrombosis and pulmonary emboli and uncommon except in the early stage following injury. Anticoagulation is rarely required following the acute stage.[27]Peripheral oedema and superficial vascular skin changes are common. Careful attention must to be paid to the feet so that cellulitis and other complications are avoided. Chiropody is sometimes helpful. Risk factors need to be regularly reviewed.

11. Skin

Immobility and loss of sensation contribute to the risk of pressure sores. Careful discipline and good care will largely prevent their development. The insensitive skin must be inspected morning and evening. The minor red marks and skin abrasion that occur during transfers are best treated by rest in bed until the skin has returned to normal.[28] With aging the skin and its underlying tissues becomes less resilient and the risk of pressure sores increases. Patients may go many years without a pressure sore and then develop a serious one. During the acute stage following injury two-hourly turns in bed are necessary. In the later stages such frequent turning is rarely required. Prone lying is

an excellent way of maintaining the hips, minimising spasticity and preventing pressure sores.

Paraplegics are usually able to turn in bed independently in their younger years. They require increasing help as they get older. Various aids such as monkey poles are helpful. Tetraplegics usually require assistance with turns from one or more persons. The required time-gap between turns in bed at night depends on the individual.

Whether one or two persons are required for turn depends on the patient. In the British Isles, European regulation must be applied. This often means that two carers are required for activities, turns or transfers where, prior to these EEC ruling, one person sufficed. The selection of the appropriate bed is important. The type required will change during the lifetime of the person concerned. Variable height beds help carers by making transfers easier. The ability to elevate the head of the bed is useful. Rotating beds are seldom popular. Most patients prefer double beds with double mattresses that they can share with their partners. Beds that appear normal are preferred to beds which, though functional, retain a hospital ambience.

An appropriate mattress will increase the gap between turn and hence the burden on the carers. Many different types are available. Some permit the patient to remain in one position for long period. Unfortunately the latter can also make turns more difficult. Many different types of cushions are available. The appropriate one for the individual must be selected. A spare cushion should always be to hand in case the main one is damaged. It must be borne in mind that in the prevention of pressure sores it is not just the cushion and its characteristics which are important but the whole posture and seating status of the patient. A Jay Back may be required to correct posture. The Jay Protector enables patients to go up and down steps on their bottoms and to travel more safely in vehicles when other methods of buttock support are not available.

The nursing staff of the Centre must be conversant with teaching patients, families and carers techniques for lifting and turning. A posture and seating clinic linked to a pressure clinic is essential to ensure that the optimum seating system for the patient is defined and that the risk of pressure sores is kept to a minimum.

12. Sexual Function

Sexuality is severely impaired following spinal cord injury. A spinal cord injured man sometimes feels incomplete because not only is normal sexual intercourse impossible

but also he cannot be a full husband, father and breadwinner, or be involved in masculine activities.[29]

Women can lose their self-respect. Wearing attractive clothes as skirts is limited by the leg-bag and the wheelchair. Urinary incontinence produce the sense of always being surrounded by a smell of urine.[30]

Although many approaches are available to achieve erections including implants, intracavernosal injection and external aids, the spontaneity, sensation and orgasm of normal intercourse are lost. Sildenafil, a phosphodiesterase inhibitor that prevents the breakdown of the cyclic GMP in penile corpus cavernosal smooth muscle, produces effective erections in approximately 70% of impotent males when taken orally. It is not yet available of general use.

Fertility in spinal cord injured men is usually severely impaired.[31] Obtaining semen is the first problem. Methods for achieving this include the penile vibrator, rectal electrostimulated semen emission, vascannulation, micro-epididymal sperm aspiration and the hypogastric plexus stimulator. The second and more important problem is oligoasthenospermia. It is usually necessary for the services of a fertility centre to be used if parenthood is to be achieved. This includes perpetration of the semen followed by various treatments of the female partner to increase her fertility. Of the Current techniques, intra-cyto-plasmic sperm injection (ICSI) has the highest success rate. With ICSI, sperm motility is no longer important. Men without any motile sperm, such as those who were pre-pubertal at the time of injury, may become fathers following testicular biopsy. Female intercourse is possible but passive. Orgasm does not occur except in women with lower levels of injury. Fertility is usually unimpaired.

Both male and female cord injured persons are unable to be parents in the full sense. They cannot take their children out to the park or play with them as previously. Relationships and marriages are under greater stress following spinal cord injury. The prospects for maintaining and developing firm, lasting relationships are reduced. This is particularly the case with young women.

The spinal cord injury service must have doctors, nurses and therapists who are knowledgeable in the profound sexual problems which follow spinal cord injury. Sexuality and fertility clinics are important. A gynecologist with a special interest in the particular problems of the spinal cord paralysed woman is essential. Close liaison with an advanced

fertility clinic is mandatory if reasonable take-home-baby rates are to be achieved for couples where the male partner is spinal cord injured.

13. Mobility

The wheelchair must be carefully selected. Expert occupational and physiotherapy assessment is required. Different wheelchairs are necessary for different purposes. For example, a sports wheelchair, a lightweight wheelchair and an outdoor electric wheelchair may all be required by the same persons for use at different times. The pattern of wheelchair requirement varies with the individual. It also changes with age. A young tetraplegic can cope with a lightweight manual wheelchair indoors on level surfaces and up shallow steps. In his older years he may require an electric wheelchair instead. The range and type of wheelchairs that are available is enormous and constantly changing. Before the appropriate wheelchair for an individual can be selected, it should be evaluated in a practical setting.

The most sophisticated wheelchairs, such as the Permobil, allow control of the environment using an infrared signalling system built into the wheelchair. These chairs can also take portable ventilators. Alternatively they can provide a stand-up or a reclining facility. The wheelchair must be integrated with an appropriate care for satisfactory mobility out of doors. The patient must be able either to get the wheelchair in and out of the car himself or to get into the vehicle whilst still seated in the wheelchair.

The selection of the appropriate vehicle and its control require careful assessment, sometimes in a specialised centre. The individual characteristics of the patient must be considered. For example, tall patients have a restricted range of vehicle that they can use whilst seated in their electric wheelchair. One adjunct that assists transfers in an out of the car is the swivel seat. Car telephones are important because if the car breaks down then the paraplegic person cannot easily get to a telephone. The care must well-maintained as the paralysed person is so dependent on it. If the spinal cord injured persons has not passed his driving test then it is mandatory that he does so. In general, tetraplegics at the level of C5 and below are able to drive. Those at C5 usually required a joystick control. Some at C6 and most at C7 and below can cope with vehicles with hand controls, automatic transmission, servo assisted brakes and power assisted steering.

Ambulation is seldom a functional form of mobility for paraplegics or tetraplegics but it can be useful as a form of exercise. For persons with poor truncal balance, such as low

tetraplegics and higher thoracic paraplegics, the reciprocating or hip guidance orthoses, which provide truncal support, are necessary. With lower levels of thoracic and with upper lumbar levels of injury, the knee-ankle-foot orthoses usually suffice. Those with good quadriceps control usually cope with ankle-foot orthoses alone. The majority of spinal cord injured patients who learn to ambulate soon cease to use their walking devices. Few regret having mastered the techinque.[11]

Public transports, such as overground and underground trains, and buses are difficult or impossible. Air travel is usually feasible. There are a number of recreational mobility vehicles, such as the three wheeler bicycle and the motorised quadbike. Children can be placed at the back of the three wheeler bicycles. Those involved in country pursuits need the quadbike for mobility over rough ground. A portable ramp is useful when visiting friends or other places where ramped access is not available.

14. Transfers

This refers to the ways in which a paralysed person gets from one position to another. Nearly all paraplegics become independent in level transfers. Most achieve the more difficult ones such as from the easy chair into the wheelchair and out of the bath. The most difficult transfers, such as getting from the floor into the wheelchair, and from the floor into the upright position having fallen in calipers, are achieved by only the most able.

There is great individual variation between paraplegics in their capability with transfers. Factors associated with reduced ability include increasing age, poor truncal balance, spasticity, spasms, obesity and upper limb problems such as muscle strains, nerve injury and joint contractures. Those with a low arm to trunk length ratio, for example achondroplastics, seldom achieve independent transfers. A few low level tetraplegics become totally independent in transfers, usually with the aid of a sliding board. Most require help. The minimum pattern of help required by each individual is best determined following a course of rehabilitation in a spinal cord injury unit. The key members of the team here are the occupational and physical therapists.[32]Hoits are important transfer aids. Portable hoists are versatile but ceiling mounted ones take up less room. Strengthening of the ceiling is required with the latter.

15. Activities of Daily Living

This refers to the normal activities of daily life which the able-bodied take for granted.

The occupational therapy department is vital in this area.[33] In general, paraplegics are independent whilst tetraplegics are partially dependent, usually in lower half activities such as washing, dressing and personal hygiene. Obesity, poor truncal balance, increasing age, upper limb musculo-skeletal problems, spasms, spasticity and short arms all reduce ADL ability.

Higher level tetraplegics benefit substantially from environmental control systems.[34] Provided that the person can voluntarily control in an accurate and predictable manner a single muscle then he can control his environment, such as opening and closing curtains and suing the telephone. An expert is required to advise each individual regarding which system is most appropriate to his needs. The optimum application is best determined at a home visit. The system should usually be in the bedroom, living room and study. Most paraplegics and tetraplegics benefit from a remote control door-opener. If a paraplegic is sitting in an easy chair and someone calls at the house, he does not have the time to transfer into his wheelchair to open the door. Paraplegics are usually able to manage the shower seat. Higher level paraplegics and low tetraplegics and low tetraplegics find the shower chair system more helpful, as described above. Most paraplegics can manage a normal bath whilst they are young. This becomes more difficult with aging. A bath-board may then help. Eventually a specialised bath may be required to relieve the carer.

16. Psychology

The effects of sudden paralysis, potential incontinence, impotence, infertility, loss of personal relationships and all the other manifestations of spinal cord damage, insinuate into every facet of the person's life. The impact can be devastating. In spite of this, depression is not a major consequence of spinal cord injury and suicide is not much commoner in the spinal cord injured population compared with the able-bodied. Most paraplegic and tetraplegic persons who have been through a spinal cord injury unit have learned to minimise the effect of their disability. they seldom concentrate on what they cannot do. Children in particular adapt well. Counselling may be helpful at various stages though it is usually resisted.

The psychologist in the multidisciplinary team has an important role not only in diagnosing and treating patients but also in assisting family and centre staff to deal with the enormous psychological and emotional strains that surround spinal cord injury. A psychiatrist with a special interest is essential both because psychiatric conditions such as depression and schizophrenia can result in traumatic spinal cord injury and because the threat of suicide may arise in the early months after injury.[34]

17. Family

The enormous impact of paralysis on the family including parents, siblings, spouses and children must be considered. Relationships can be destroyed. The old age of parents can be shattered by paralysis in their children. The ability of the spinal cord injured person to be a true father, wife, husband or mother is severely impaired. The adverse effects on the family rebound on the patient who sometimes feels guilty for the suffering cause. 35The view that family members should look after their spinal cord injured relative is no longer widely accepted. It is better for normal relationships to be retained. This will increase the likelihood of preserving the integrity of the family. In particular a wife should remain a wife, mother and lover rather become a nurse and carer. The National Spinal Injuries Centre in Stoke Mandeville holds regular teach-in days for families and carers. Chronic spinal cord injured patients and their families attend and provide invaluable insights based on experience to the families of those who have recently been injured.

18. Home

The accommodation which most spinal cord injured persons have at the time of injury is seldom suitable for the life in a wheelchair. Early housing assessment is required. Incomplete paraplegics who can ambulate and cope with stairs in their younger years find that this becomes increasingly difficult as they grow older. Many eventually become wheelchair dependent. Crutch and rollator walking takes up more space that normal ambulation. Doorways and corridors need to be wider to take account of this. Tetraplegics and complete paraplegics are safes in ground-floor wheelchair accessible accommodation. This is seldom achieved in the United Kingdom first because most houses are two storey and second most spinal cord injured persons like to remain in their won area. Accordingly, through-floor lifts and stair-lifts are usually provided instead. The precise housing requirements following spinal cord injury depend on the person concerned and the pattern of disability. [37] It is seldom possible in the United Kingdom for all needs to be met by statutory authorities so some element of compromise is almost always required.

Amongst the many housing aspect which must be considered are the following:

1. There should be a covered way for the car and from the car to the front door together with adequate space for the spinal cord injured person to get in and out of

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Amongst the many housing aspect which must be considered are the following:

1. There should be a covered way for the car and from the car to the front door together with adequate space for the spinal cord injured person to get in and out of

the vehicle under cover. This requirement is seldom met. Most have to accept that to get to and from their car they will be exposed to the elements.

2. There must be appropriate, usually ramped, access to the house. This is usually available, though initially only by way of temporary ramps.

3. Doorways and corridors should be of sufficient width to accommodate the wheelchair base and turning circle. This requirement is seldom achieved. Accordingly skirting boards and doorways are usually scuffed and damaged.

4. Adequate storage space should be available to avoid equipment cluttering up corridors and living space. This is seldom available.

5. The main bedroom should be of sufficient size for easy wheelchair mobility and with adequate storage space for catheters, urinary sheaths and other personal equipment.

6. There should be an en-suite toilet and bathroom to the main bedroom. This is necessary because the spinal cord injured person needs to get to and from the bathroom and toilet whilst seated in his wheelchair in a state of undress.

7. There must be appropriate hoists. These will be required occasionally when the spinal cord injured person is young and regularly when he is older.

8. For tetraplegics, and paraplegics in the last years of their lives, carer accommodation is required. To ensure regular recruitment of satisfactory carers their accommodation must be comfortable and attractive.

9. Paraplegic and tetraplegic persons are less able to maintain their body temperature. In the case of tetraplegics, temperature control is further compromised by altered sympathetic nervous control. Central heating is advised in all cases. Because tetraplegics can become overheated in hot weather it is desirable that at least one room in the house has air conditioning.

19. Recreation

Recreations possible before injury are seldom practicable afterwards. A home computer system is often helpful. High tetraplegics benefit from pager turners. Although some

paraplegics and tetraplegics enjoy wheelchair sports, the majority are no more sporting that the rest of the population. Access to place of public enjoyment such as theatres and cinemas is often difficult or impossible.[38]Regular holidays help maintain morale and family relationships. They are usually more expensive as the cheaper hotels are inaccessible to wheelchairs. Extra help is required. A regular visit from and Occupational Therapist is helpful in brining the persons up to date with modern developments in aids, equipment and retractions. Disabled clubs and societies also provide information. Skiing, scuba diving, piloting aeroplanes, abseiling and wheelchair rugby are a few examples of the type of recreations that are possible.

20. Employment

Sir Ludwig Guttmann would tell his patients "you are not rehabilitated until I see your first pay-slip". This most important rehabilitation goal is, regrettably, often not achieved. The opportunities for employment following spinal cord injury are greatly reduced. Retraining centres exist. The disablement resettlement officer can also advise. Many universities have facilities where spinal cord injured persons can study. Most succeed in getting good degrees. However there is a great difference between obtaining a qualification and achieving employment. In general, the wheelchair dependants are overlooked when there is competition.[39]. Those who had physical outdoor manual employment prior to injury and in particular those with poor academic backgrounds are at a great disadvantage following spinal cord injury. They usually remain unemployed. Academically capable patients and those who succeed in retraining clerically still face many problems. It takes longer for them to get up and get going in the morning. At work the care must be under cover and with access from it to the place of work. The latter must be wheelchair accessible. Getting from on floor to another and from one building to another is usually difficult and sometimes impossible. There must be facilities at work to allow for episodes of incontinence. Employers have to accept that complication such as red marks and urinary tract infections will result in time off work. Drugs such as Baclofen interfere with concentration and mental agility. Although many paraplegics and some tetraplegics achieve some form of employment, it is more likely to be part than full time, intermittent than continuous and to involve early retirement.

21. Post first admission medical care

In the chronic stage following spinal cord injury, complications can arise, such as pressure sores and urinary tract infections. Some can be successfully treated at home. When hospitalisation is required this should be prompt and into a spinal unit. Annual

comprehensive review in a spinal cord injury unit is required. This should include upper unitary tract assessment and a comprehensive clinical review.

22. Aging

Some of the effects of aging have been considered above. It is essential that these are taken into account when planning and spinal cord injury service. There is no stereotypical pattern for aging. Some people are intrinsically more able than others. Others have the effects of aging brought forward by problems such as contractures.[36]

23. Care Attendant Needs

Low level paraplegics are usually independent when young apart from needing help with domestic activities, shopping, certain obstacles out of doors, gardening, do-it-yourself work and home maintenance. They usually stand by assistance when ambulating in calipers or similar devices. Mid-level paraplegics may require assistance with getting into and out of the standing frame out of the bath, in and out of the care and with lifting the wheelchair in and out of the car. Spasticity, spasms, intrinsic ability, truncal balance and age are important. Some low level tetraplegics are almost independent. The majority require some assistance. For example, they can use a spoon for eating but not cut up meat. They can drive a car but not transfer into it or lift their wheelchair in and out of it independently. Because tetraplegics can get autonomic dysreflexia or choke on food someone should be at hand to deal with an emergency should the need arise. Notwithstanding this, many tetraplegics live on their own for substantial periods of time. This is a reflection of the inadequacy of resources available in the community rather than the particular needs of the tetraplegic person. As mentioned above, in general it is not appropriate for family members to be involved in the physical and personal care of their relations. Nevertheless, they frequently choose to do so and provide extremely good care. The patter and type of domiciliary physiotherapy required depends on the individual. In general, carers can carry out the straight forward physiotherapy activities of join range of motion and assisting patients into the standing frame. More specialised physiotherapy tasks require a Chartered Physiotherapist.

Conclusion

The successful rehabilitation of a spinal cord injured persons is critically dependent on a careful understanding of that particular individual including his past history, current

situation and further aspirations. Treatment should be carried out in a Spinal Cord Injury Centre.In the United Kingdom of Great Britain and Northern Ireland the Clinical Service Specification of a Modern Spinal Cord have been laid out. They incorporate the following:

1. Cooperation in the efficient retrieval and early admission of acute spinal cord injured patients for specialised care. This requires liaison with the Ambulance Service and with all Accident and Emergency and Acute/Orthopedic Units in the region. It includes the provision of guideline for acute care and transportation.

2. An admission system that has the support of a fully equipped Accident and Emergency Unit and Trauma Service to deal with the admission of patients both directly from the local accident scene and for those who have been transferred. On those occasions when the spinal cord traumatised person is too ill to be transferred immediately to the Spinal Cord Injury Centre, provision must exist for the patient to be visited at the referral hospital to ensure that optimum treatment is applied.

3. The accurate and rapid diagnosis of the spinal lesion using modern diagnostic aids. The service must have access to facilities for full diagnostic investigation including plain X-ray films, CT scans and MRI scans on a twenty-four hour basis with other modalities such as neuro-physiological assessment being available as appropriate.

4. Specialist management in the acute phase with the aim of optimising recovery and minimising complications. The service must have available the support of Orthopaedic Surgery, Neurosurgery, General Surgery and Anaesthesia. The hospital must have the capability of managing multiple injuries and patients requiring ventilatory support.

5. Physical and psychological rehabilitation to enable patients to reach their full potential for independent living. The service must have dedicated Physiotherapy and Occupational Therapy staff with on demand services from Speech Therapy and Dietetics. The team will include support from Clinical Psychology as routine. Psychiatric Services will available on demand.

6. Discharge of patients to appropriately modified domestic or residential facilities. The service must have close links with the Social Services and other community providers.

7. Participation in health related aspects of retraining to enable patients to pursue an economic carer or to develop activities within a sheltered work setting. This is not merely an extension of Occupational Therapy but also involves close links with local Colleges/Universities etc.

8. Provision of after care which encompasses Hospital Outreach Services. After care for spinal cord injured patients necessitates lifetime surveillance. The Unit must provide community liaison services with open access for consultation by patients, general practitioners and community nursing staff.

9. Provision of self-care teaching programmes for patients and carers and a programme for relatives.

10. Provision of specialised teaching programmes concerning spinal cord injury for health care professional trainees including prevention, diagnosis and management.

11. Provision of clinical audit of the process and outcome of care for acute spinal cord injured patients.

12. Provision for the readmission of spinal cord injured patients first for the treatment of life-threatening complications such as respiratory failure, intractable autonomic dysreflexia, septicaemia and widespread tissue necrosis with toxaemia and second for major surgery such as thoracotomy for phrenic nerve implant insertion, spinal canal exploration for the treatment of syringomyelias and intraspinal somatic and autonomic nerve implants, and laparotomies required for major reconstructions.

To ensure that no key areas are omitted, doctors are advised to apply a check similar to that shown in the table.

Table Rehabilitation of a Spinal Cord Injured Person.

01. Associated injuries
    Brain
    Limb/joints/bones
    Peripheral nerves/brachial plexus
    Chest/abdominal
02. Neurology - level, completeness: syrinx
03. Spine - deformities, arthritis
04. Pain

05. Bladder - upper and lower urinary tract
06. Bowels
07. Joints - heterotopic ossification
08. Spasms/spasticity
09. Respiratory
10. Cardiovascular - hypotension, autonomic dysreflexia
11. Skin - turns in bed, mattress, cushions, bed shower chair, bath
12. Sexual function - fertility, intercourse, sexuality
13. Mobility - wheelchair, car, orthoses, recreational mobility, ramps
14. Transfers - hoists
15. Activities of daily living - environmental control systems
16. Psychology - counselling
17. Family
18. Home
19. Recreation - holidays
20. Employment
21. Care Attendant Needs

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Rehabilitation of patients with neuromuscular disorders

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Introduction

The rehabilitation of patients with progressive neuro-muscular disorders involves a multi-disciplinary approach. The principles of rehabilitation in neuro-muscular disorders affecting children include : fostering optimal physical, mental and emotional development; maintaining or improving function whenever possible; and preventing secondary complications such as contractures, skin break down and malalignment of the spine [1]. The patient with neuro-muscular disease, must initially be evaluated by the neurologist, with detailed history, particular emphasis being given to the age of onset of symptoms, type of progression, (gradual, remitting - relapsing, rapid or static), and pedigree history. Family history should include a detailed pedigree chart spanning three generations and examination of family members (including those who are believed to be "unaffected"). This helps to determine the pattern of inheritance and is of utmost importance for genetic counselling and molecular genetic studies. A meticulous clinical examination reveals the pattern of muscle involvement, which is typical and characteristic of certain diseases for eg. facial and shoulder girdle involvement in facio-scapulo humeral dystrophy, calf muscle hypertrophy and proximal weakness initially of lower limbs in boys with Duchenne and Becker dystrophies, frontal baldness, cataract, ptosis, facial weakness, distal weakness and myotonia in myotonic dystrophy and fasciculations with wasting, weakness and brisk reflexes in motor neuron disease. Detailed clinical evaluation provides clinical differential diagnoses which are corroborated by laboratory investigations including estimation of muscle enzymes, electroneuromyography and muscle biopsy. The final diagnosis is established by correlating clinical, electrophysiological and histopathological information [2]. Accurate diagnosis is a pre-requisite for proper management. The clear differentiation between polymyositis and dystrophy, congenital myopathy and myasthenia, chronic inflammatory demyelinating polyneuropathy and hereditary motor sensory neuropathy has important therapeutic implications. The next step is one of imparting this information to the patient.

Informing the patient about the diagnosis

This requires considerable time and communication skills. The patient approaches the doctor in the hope of complete cure. When a progressive neuromuscular disease is

diagnosed, which does not have a complete cure, the treating team is faced with the responsibility of breaking this news in the best possible manner, so that it produces least emotional distress while simultaneously apprising the patient or family of the true nature of the disease so as to prevent medical shopping. Neuromuscular disorders like muscular dystrophies, motor neuron disease, spinal muscular atrophy and congenital myopathies are rare as compared to stroke. However, patients require life long care and with advancing age, there are changing needs. Pamela Shaw[3] in her experience with patients suffering from motor neuron disease recommends balancing information about the lack of specific treatment and cure, with the inherent variability in the clinical course of the disease and the palliative therapies aimed at alleviating distressing symptoms, which can do much to improve the quality of life for the patient during the course of the disease.

When once the patient and/or family are aware of the nature of the disease and its implications, the process of rehabilitation can commence. At NIMHANS, for the past seven years, a neuromuscular clinic is conducted once a month, where we attempt to provide comprehensive care to patients through a multi disciplinary approach. The team comprises of neurologists, social workers, orthopaedic surgeon, physiotherapists, occupational therapists, clinical geneticist, yoga therapists and Ayurvedic physicians. Aspects of rehabilitation pertaining to certain common disorders are outlined in this chapter.

Duchenne muscular dystrophy

This is the most common dystrophy we have encountered at the clinic. The absence of specific treatment makes it all the more important to prevent its physical, emotional, social and educational complications and to provide active support for the family throughout the course of the disease[4]. The clinician's first responsibility is to provide an unequivocal diagnosis and enough information and constructive suggestions to enable the family to formulate practical plans for the future. These must be given in relaxed sessions after the initial shock has passed[5]. Melinda Firth interviewed 69 parents of boys suffering from Duchenne dystrophy at home[6]. The interview explored the parents' experiences at the time of their sons' diagnosis. Many parents had experienced distressing delays (average 2.5 yrs) between the time they first became aware of the symptoms and the time of the diagnosis. Only on 18 occasions were both parents informed of the diagnosis together. One third of the parents were "not satisfied" with the way the diagnosis had been communicated. Though there is clearly no single best way to tell the

parents of a child that he has Duchenne muscular dystrophy, the suggestions of the parents who have been through this experience are summarized below :

(1) Most parents want to know if there is something wrong with their child as soon as possible. Evidence from parents of handicapped children shows that most have strong feelings that early telling is desirable.
(2) Most parents prefer to be told the diagnosis together
(3) Parents should be given some privacy when told the diagnosis. They also require some time to allow the release of emotions.
(4) Full and balanced information should be given. Parents have different opinions about how much of the prognosis should be explained in the initial session.
(5) As many parents will be unable to take in much of what they are told at the first notification of the diagnosis, a series of contacts should be planned.
(6) Social workers and other professionals who have a clear understanding of the disease and its implications could provide good follow up support to families who have recently learnt the diagnosis.
(7) Parents must be encouraged to ask questions and voice their worries and uncertainties.
(8) The offer of contact with another parent of a child with Duchenne dystrophy may help. The parents had strong feeling both for and against this form of support.

Firth, Gardner - Medwin and co-workers[6] conducted interviews of parents to determine problems faced by parents of boys with Duchenne dystrophy. 62% of the problems were "practical" related to lifting, bathing, toileting, dressing, feeding and finding suitable accommodation; 23% were "service" problems relating to difficulties in obtaining services aids and allowances or dissatisfaction with them, 15% were emotional problems such as the affected boy's depression, the parents' emotional problems in watching their son deteriorate, parental isolation and awareness of society's attitude to handicapped people. Once patients have been apprised of the diagnosis, they may be introduced to passive exercises.

Passive Stretching Exercises

Studies on the natural history of Duchenne dystrophy[7] indicate that boys begin to fall quite frequently and have increasing difficulty in climbing stairs between six to eight years of age. From 9 to 11 years of age, they cease climbing stairs completely and are no longer able to stand up from the floor. Part of this difficulty is caused by the muscular

weakness and part of it by contractures which develop in the hips, knees and ankles and make it impossible for the child to stand with the degree of hyper-extension necessary to maintain balance[8]. Contractures of the iliotibial bands and hip flexors are noted by four to five years of age and heel cord tightness follows shortly. Early therapy is therefore aimed at preventing them. Passive stretching of the hip flexors and iliotibial bands is an important part of management[4]. Prone lying is useful to prevent hip flexion contracture.

Vignos et al recommended passive stretching for at least 5 seconds, repeated 10 times, twice a day[9]. Passive stretching exercises should be performed daily by the family[8]. Stretching exercises done on a weekly basis are completely ineffective. In a study of therapy and management of contractures, the most effective treatment in the prevention of contractures was splinting. There was a high correlation between the use of night splints (plastic ankle foot orthoses) which maintain the foot in dorsiflexion during the night and preservation of movement at the ankle[7].

Active Exercises

Vignos and Watkins studied 211 patients with muscular dystrophy [10]. Maximum resistance exercise programme was instituted for one year. Improvement in muscle strength occurred in all patients throughout the first four months of exercise regardless of the type of dystrophy. Subsequently, a plateau occurred and was maintained throughout the period of observation. Improvement in functional abilities was less than the increase in muscle strength. Patients with adult onset dystrophies (limb girdle and facio scapulo humeral) derived the maximum benefit. de Lateur and Gianconi studied four children with Duchenne muscular dystrophy in whom they submaximally exercised one quadriceps using a Cybex isokinetic exerciser and recorder, with the contralateral quadriceps as control [11]. This was performed four or five days per week for six months. There was no statistically significant increase in strength during the exercise period. There was no evidence of over work weakness. Scott et al published a six month study comparing a group of boys with Duchenne dystrophy who performed manually resisted exercise with another group who performed exercises in response to oral commands for 15 minutes per day [12]. No statistical difference was found between the two regimens. Fowler and Taylor suggested that exercise training programmes in patients with neuromuscular disease should be started early in the course of the illness when muscle fibre degeneration and regeneration are minimal [13]. They emphasized submaximal exercise levels. There is still no satisfactory published trial of the effect of active exercise in Duchenne dystrophy but it is common experience that Duchenne boys benefit from

exercise and conversely, that rest is detrimental[14]. Bed rest for minor illness or trauma should be avoided whenever possible and regular walking, swimming and games, a little more than the boy really wants to do, should be encouraged. Older boys can do more formal and deliberate exercises, and in the late stages, these should be continued with emphasis on the upper limb and breathing exercises. At NIMHANS, we have encountered over 100 boys with Duchenne dystrophy at the neuro muscular clinic. They are taught active exercises and encouraged to practice them daily. Swimming is also advocated. They are also referred to experts in Naturopathy and yoga for training. Some of the families opt to spend a fortnight at a residential facility where they undergo intensive training in yoga. The mother is also taught yoga exercises, so as to enable her to supervise the child.

Respiratory Muscle Training Exercises

Since respiratory failure accounts for death in over 75% of Duchenne muscular dystrophy cases [14], specific respiratory muscle training exercises are often used to improve ventilatory endurance. Uncontrolled studies showed improvement in vital capacity after a breathing exercise programme [15,16]. Di Marco et al evaluated the effects of inspiratory resistive training on respiratory muscle function in patients with Duchenne (n=5), limb girdle (n=5) and facio scapulo - humeral (n=1) muscular dystrophies [17]. Following six weeks of training, there was remarkable improvement in ventilatory endurance. In six patients, who trained for an additional six week period, respiratory muscle endurance increased even further. The degree of improvement was positively correlated with baseline vital capacity and maximal inspiratory pressure, suggesting that those individuals with the largest mass of functional respiratory muscle and the largest proportion of relatively normal respiratory muscle fibers achieved the greatest improvement in endurance with training. They suggested that an exercise programme should be instituted early in the course of the disease when there is maximum amount of functioning muscle. Although there have been some suggestions that over activity may accelerate muscle breakdown and result in a deterioration in muscle performance, in this study there was no loss of respiratory muscle function in any of the patients. The authors had used a simple inexpensive technique which could be carried out at home with minimum supervision. They suggested that improvement in respiratory muscle endurance might delay the occurrence of pulmonary complications and the onset of respiratory failure. Martin noted improvement in endurance, but not in strength as a result of specific respiratory muscle training [18]. They suggested that this was the result of training type I fibers, preventing deterioration caused by disuse atrophy, the disease or both, Smith et

al reviewed the practical problems in the respiratory care of patients with Duchenne muscular dystrophy [19]. The long standing respiratory muscle weakness in this disease leads to serious secondary problems which include scoliosis, thoracic mechanical abnormalities, widespread microatelectasis with reduced lung compliance, weak cough with retained secretions and repeated infections, ventilation - perfusion imbalance and recurrent sleep related hypoxemia. Maximum inspiratory pressure is reduced at all lung volumes in patients with Duchenne muscular dystrophy. This occurs early in the clinical course of the disease and may antedate changes in vital capacity. Maximum static expiratory pressures are lower than are maximum inspiratory pressures in patients with Duchenne dystrophy. Maximum respiratory pressures give little information about the reduction in lung and chest wall compliance through fibrosis and shortening of dystrophic muscle. Normocapnic hypoxemia is common in patients with Duchenne dystrophy who have moderate or severe respiratory muscle weakness. In the absence of infection, hypercapnia occurs only as a pre-terminal event. These boys are at risk of clinically important sleep hypoxemia, severe cases may have REM related central sleep apnoea which may account for the tendency of these patients to die at night. Smith et al cautioned against the use of resistive training in advanced disease as the weak muscles are working against the reduced compliance of the lungs and chest wall and may already be close to their fatiguing threshold [19]. They concluded that the role of training remains controversial and more work is needed to clarify it.

Gardner-Medwin and Walton recommend regular breathing exercises and prevention of scoliosis [4]. Postural drainage and appropriate antibiotic treatment is important during respiratory infections.

Ventilatory Support

Chronic respiratory failure with nocturnal hypoxemia can produce anorexia, weight loss, apathy, depression, late-night insomnia, restless fearful sleep, morning headache, somnolence, inertia and sometimes morning cyanosis. Hypoxia can accelerate muscle weakness. The use at home of nocturnal positive-pressure ventilation, using a tightly fitting mask will relieve many of these symptoms and may substantially improve the quality of life [4]. Miller et al studied decision making, daily functioning and quality of life for patients with Duchenne dystrophy who were ventilator dependent [20]. They concluded that providing life extending technology to patients with progressive disease is controversial and much debated among health care professionals. Accepting or rejecting assisted ventilation is a decision to be made by patients and families. Health

care providers must abandon the role of decision makers and become information providers. Patients and their families must be given the necessary information and the option whether or not to choose assisted ventilation well in advance, so sufficient time is allowed for well thought out decisions. Some patients may refuse ventilation and allow the disease to take its natural course. Respiratory insufficiency is an anticipated event and should not be allowed to occur as an unexpected emergency.

Orthoses and Surgery

The time at which leg braces are used for ambulation and the type of brace used are critical to the success of braced ambulation[8]. The major functional weakness is that of quadriceps. Early in the course of the disease, children fall because they are hurrying and careless about their balance. Bracing at this stage will slow down the patient, who will find it unacceptable. The child finds it easier to tolerate bracing when falls occur during quiet standing or slow walking. Watt and Brooke use the following criteria for bracing; the child has several falls per week, can no longer climb stairs or only with difficulty, cannot rise from the floor and cannot extend the knee against gravity[8]. The long leg brace with knee support is most useful. The disadvantage of the plastic knee ankle foot orthosis (KAFO) is the tendency of the patient to throw the foot into equinovarus, resulting in calluses and abrasions on the outside of the foot. The metal double upright attached to a surgical boot is cosmetically less appealing, but can be more comfortable and holds the ankle in a more rigid position. If the limb is too contracted to allow the use of the long leg brace, percutaneous tenotomy may be performed with release of iliotibial bands, hip flexors, hamstrings and heel cords[21]. With the passage of time, it has become evident that a good physiotherapy programme instituted early, together with the use of ankle night splints, makes it possible to mobilize the boy in callipers without surgery[4]. If tenotomies are to be performed, the dangers of anaesthesia must be borne in mind and post-operative mobilization must be rapid. Prolongation of walking and standing delays the onset of kyphoscoliosis. Ultimately, on account of progressive weakness, the boy opts for a wheel chair which should initially be hand propelled, but later may be electrically propelled.

Progressive scoliosis sets in soon after patients become unable to walk. This may go on to cause severe thoracic distortion and respiratory impairment. Good posture in the wheel chair can prevent scoliosis. The use of spinal braces and moulded wheel chair inserts to prevent scoliosis is an area shrouded in controversy[9,22,23]. Spinal fusion with the insertion of a Harrington rod has been advocated as an alternative to bracing when

scoliosis is uncontrolled[24,25]. Most cautious surgeons claim no benefit from the operation in terms of life expectancy. Gardner - Medwin and Walton examine the spine every three months among boys who have lost ambulation and offer spinal fusion if the curve exceeds 20 degrees at a stage when the vital capacity is still sufficient for safe anaesthesia.

There are other primary muscle disorders like congenital myopathies and chronic polymyositis where weakness is predominantly proximal but severity and progression are much less than in Duchenne dystrophy. The general principles outlined above hold true for these disorders as well.

Motor neuron disease

In most instances, probably about 80% of cases, the diagnosis of motor neuron disease (MND) is easy, especially for the experienced neurologist. However, distress may be caused to the patient if there is no need for investigations and the diagnosis is given at the first consultation. Most neurologists therefore avoid doing this and many would admit the patient to hospital for investigation, even if this is not strictly essential. The early development of bulbar and pseudo-bulbar palsy is usually a sign of poor prognosis. Other factors that influence the prognosis are the patients' age, other medical diseases, emotional background, financial position, housing and partner's health. Focal forms of motor neuron disease described in India, like monomelic amyotrophy[26] and wasted leg syndrome[27] have a very good prognosis. Juvenile motor neuron disease, described from Madras, also has a good prognosis[28].

Telling the diagnosis

The patient and partner should be told the diagnosis once it is reasonably well established. It is well known that patients take in very little information after they have been told a very serious diagnosis. For this reason, only a small amount of information should be given at the first interview. A second interview two weeks later is usually desirable for more information[29].

The patient experiences a stage of deterioration without major disability for 12-18 months. During this period, advice about job and driving and information about research in motor neuron disease are usually requested by the patient. Subsequently, bulbar problems, speech difficulties and respiratory problems arise.

Salivary Drooling

The factors which contribute to salivary dribbling/drooling in motor neuron disease include loss of the automatic swallow reflex, weakness of lip closure, weakness and spasticity of the tongue, infection of the mouth especially oral thrush, and head position i.e. the head falling forwards. Exercises to improve the awareness of lip closure and strengthen lip seal may be of assistance. The patient may be encouraged to hold a tongue depressor between the lips as he watches television. Lip exercises such as closing the lips against resistance or producing bilabial sounds such as "p" or "b" may be used [30].

Spasticity of the tongue can be reduced by sucking ice, by external application of ice to the neck, or by the use of anti-spasticity drugs such as baclofen. Candida infection of the mouth should be treated with frequent mouth hygiene and antifungal medication. The use of a proper chair and collar can avoid neck tilt. Some patients produce very thick secretions. Anticholinergic drugs must be avoided in these patients.

Dysphagia

The act of swallowing can be arbitrarily divided into five phases : pre-buccal, early and late buccal, pharyngeal and esophageal, all of which may be affected in MND. A careful history about possible dysphagia should be obtained. The patient should be observed while drinking about 50 ml of water and eating a biscuit. Lip closure, tongue strength, palatal movement, strength of the cough, presence or absence of drooling and dysarthria need to be ascertained. Cine-videofluoroscopy is useful in some patients when there is hold up at the level of the cricopharyngeal sphincter.

The patient should have his meals in a relaxed environment. Head support may be beneficial. The food should be firm and smooth but not solid or unyielding. The consistency can be varied by the use of blenders/mixers. Most patients find that semisolids are easier to manage than fluids. A few experienced clinicians have used palatal splints to support a flaccid palate [31].

Cricopharyngeal myotomy (CPM) has been practised for many years. Langton Hewer and Enderby reviewed five published papers on this subject, which discussed the results of CPM in 106 patients with motor neuron disease [32]. The post operative mortality varied between 6% and 30%, the average being 14%. Benefit reported ranged from

64% to 100%. However, there had not been an objective assessment of benefit. The value of this operation is not definitely known and many experienced clinicians have abandoned the procedure [29].

Alternate forms of feeding should be considered when there is frequent choking and the patient is dehydrated. The nasogastric tube is cosmetically not very appealing. However, it does circumvent the problem of dysphagia. Narrow bore tubes are less obtrusive than wide bore ones. The tube does cause mucosal irritation with an increase in secretions. Percutaneous, endoscopic gastrostomy, using a small bore catheter inserted under local anaesthesia is a simple, safe and effective procedure for malnourished, dysphagic patients [33, 34, 35].

Dysarthria and communication problems

A combination of weakness, spasticity and in-coordination of movements result in dysarthria in 75% of patients with MND. Decreasing the speed of speech and using short phrases may improve intelligibility. Loose dentures may play a role in dysarthria and must be dealt with. Reduction of spasticity by using local ice or baclofen also helps. The patient can point to the first letter of each word on a letter board as he speaks thus providing visual clues. Communication aids like the Canon communicator and scanning aids like the Possum communicator are available in the United Kingdom. Speech syntheses are also available on a soft ware package that can run on a modern portable computer at moderate cost. Speech therapists are mainly involved in the management of dysarthria.

Constipation

This can be a major problem in motor neuron disease [36]. There are many precipitating factors including weakness of the abdominal muscles, spasticity of the pelvic floor muscles, immobility, lack of fibre in the diet, dehydration, and the use of anticholinergics and opiates. Constipation can be very distressing to the patient. Management comprises of increasing the dietary fibre, maintaining adequate fluid intake and avoiding anti-cholinergic drugs if possible. Patients are sometimes able to have a bowel action after relaxing in a warm bath. Bulk laxatives like ispaghula can be used, as also suppositories and enemas. If faecal impaction occurs, manual evacuation should be undertaken by a professional helper.

Pain :

Pain is common in motor neuron disease [36,37]. Saunders and colleagues studying 100 cases seen in a hospice found that 45% complained of pain, and this arose from stiff joints, muscle cramp and skin pressure [38]. Comfortable positioning, regular physiotherapy and treatment of spasticity can help in pain relief. Emotional lability sometimes occurs with inappropriate laughter and crying. This frequently responds to imipramine in the dose of 25 mg two or three times per day.

Wheel chairs

The choice of wheel chair depends upon an accurate assessment of patients' impairment, the circumstances of its use and the length of time spent in the chair. patients with neck weakness would benefit from a chair with reclining back and collar., side supports help truncal instability, leg elevators reduce leg swelling, special seating minimizes the risk of pressure sores and ball bearing arm supports are needed when there is arm weakness. These measures serve to alleviate suffering in patients with motor neuron disease .

Respiratory failure

The majority of deaths from MND are probably due to respiratory failure and/or bulbar palsy [37,39,40]. The general management of respiratory insufficiency includes the detection and prevention of aspiration pneumonia, proper positioning and chest physiotherapy and the judicious use of antibiotics. Patients should avoid contact with people who have significant upper respiratory tract infection.

Assisted ventilation

Assisted ventilation is not a reasonable option for the majority of patients [29]. It is to be considered only after full discussion of the implications with the family, neurologist and pulmonary physiology team. Non invasive negative pressure assisted ventilation may be acceptable when respiratory insufficiency limits patients' activities. Nine patients who used a cuirass ventilator during sleep derived considerable subjective improvement in sleep, mobility, exercise tolerance, morning headache and day time fatigue [40].

Spinal Muscular Atrophy

In acute infantile spinal muscular atrophy (SMA) nutritional and respiratory concerns are of primary importance in the first year. The use of nasogastric feeding may be necessary. Chronic assisted ventilation is probably not warranted because of the poor prognosis. In the less severe forms of SMA, attention should be directed towards prevention of complications and improvement of function.

Watt and Greenhill recommend a custom seating device at 6-8 months of age in order to mimic normal motor development. A special headrest can be used to support the neck[41]. At one year of age, a standing frame or A-frame is provided to promote axial loading and to stretch out contractures. Night splints for the feet are routinely used. Most of the children are unable to propel the standing frame, and a miniature wheel chair is provided. A spinal orthosis is used to correct early scoliosis. These children are usually very bright and hence, powered mobility can be provided very early.

Granata et al provided orthoses to seven children with the intermediate form of SMA[42]. They ranged in age between 1 year 8 months and 4 years, 4 months (average age 2 years, 8 months). All of them could sit unsupported but none of them had ever stood or walked before fitting the orthoses. The orthosis was constructed on a plaster cast using polyethylene and light weight metal bars with ischial support and releasing knee joint. Six of the seven children were able to stand with the orthosis and assisted ambulation was subsequently achieved in four. The children tolerated the orthoses fairly well. The authors suggest that it would be ideal to provide orthoses before the age of two years.

Besides, soft tissue releases to allow brace wearing, Watt and Brooke are not aggressive in the treatment of joint contractures. Most of the older non-ambulatory children, develop hip dislocation, which is usually painless[8].

Scoliosis of the collapsing paralytic type is a problem which is difficult to treat in SMA[43]. Patients usually wear spinal orthoses until the scoliosis progresses beyond 40°. Elective surgery with pre-operative pulmonary assessment and post operative. endotracheal ventilation is usually successful. Luque and Moseley spinal fusion has revolutionized the treatment of paralytic scoliosis[44].

Patients of SMA become wheel chair bound in their teens. Deep breathing exercises, use of inspirometer, chest physiotherapy and postural drainage help to improve pulmonary function. Chronic ventilation is a measure to be discussed seriously with care givers and

patients. Computers, adaptive equipment help to improve the quality of life of patients and care givers.

Hereditary motor sensory neuropathies.

This group of heterogeneous disorders is characterized by symmetrical, slowly progressive distal muscle weakness. Endurance training probably improves general cardiovascular fitness and prevents disuse atrophy. The most common need for an orthosis in the lower extremities is to compensate for paresis or paralysis of the dorsiflexor of the feet. A light weight spring wire brace may suffice. This is however, not durable enough for hard use and not recommended for children. If the foot tends to pronate or supinate, a double upright brace with or without a T strap can be considered[45]. Orthoses for the lower extremity that fit inside shoes have also been described. When there is anaesthesia of the foot, it is safer to have custom made orthosis. Functional neuromuscular stimulation for ambulation does not apply in lower motor neuron lesions because the muscle cannot be stimulated through innervating nerve.

Severe weakness of the quadriceps may make it necessary to brace the knee so as to prevent buckling. The standard brace for stabilization of the knee is fastened to the shoe, has a knee joint that can be unlocked when the patient wishes to sit down and extends up the thigh posteriorly almost as high as the ischial tuberosity. If the brace is too short, the patient may slump into it, promoting flexion contractures at the hip and knee and increasing the pressure on the skin of the posterior thigh and in front of the knee.

The surgical procedures which may be considered include osteotomy, fasciotomy, tendon lengthening, arthrodesis and tendon transfer.

Participation in outside activities should be encouraged. Vocational guidance and counselling help the patient find work that is suitable. Work in a sheltered workshop may represent the ultimate goal or be an intermediate phase wherein vocational potential can be developed.

Conclusion

Rehabilitation of patients with neuro-muscular disorders involves a multidisciplinary approach. The overall aim is to improve the quality of life, limit impairment, minimize disability and alleviate pain and distress.

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Cognitive Rehabilitation

Shobini L. Rao

Introduction

Brain injury often leads to residual deficits in the physical and psychological spheres. The physical deficits and its resulting limitations are obvious. It is easy for the patient and family members to accept that these are beyond the patient's control. Psychological deficits are in the nature of cognitive and emotional problems. They affect the functioning of the patient in the family, social and occupational spheres. However link between cognitive deficits and psycho social outcome is not clear to the patients or their family members. The patient may be held responsible for the problems he / she faces in the family, occupation or society. This misattribution further complicates the already difficult predicament of the patient. Neuropsychological rehabilitation is useful in improving psychological functioning of the patient. The improvement in turn reduces the difficulties faced by the patient in his everyday interactions in the family, occupation and society. Neuropsychological rehabilitation helps to restore the patient to his/her premorbid level of functioning.

Need for Cognitive Rehabilitation

Head Injury and Stroke are common causes of brain injury. The incidence of head injury in US is 295 per 100,000 to 400 per 100,000[1]. In India head injuries affect nearly 1,50,000 persons every year [2]. Head injury is a major cause of lifetime disability and impairment for young persons. Disability rates calculated on the basis of hospital admissions, survival rates and variable disability proportions showed that 33 / 100,000 or 83,000 persons are disabled in the US alone in 1990 3 due to head injury. Even among patients with mild head injury , 84% of patients had complaints at three months, 50% had poor memory and only 34% of the patients returned to work [4]. In the moderately injured group 96% had complaints and 90% had memory problems. Again only 31% had returned to work after three months[5]. The above studies indicate that cognitive deficits are present even in mild to moderate head injury. Cognitive functions have been recognised as an outcome measure of clinical improvement. The assessment of attention, memory, language, mental processing, motor functions and behaviour is advised in severe to moderate injury[6].

Stroke is another disorder wherein brain injury leads to cognitive deficits. Prevalence of stroke is between 84 and 144 per 100,000 in different countries. Residual disability is present in 30% to 50% of stroke victims and memory deficits in 30%[7]. At one year after stroke visuo - perceptual impairment including visual neglect was present in 31 - 41% and memory deficits in 30% of the stroke survivors[8]. Specific neurological deficits including organic cognitive deficits are important determinants of functional outcome after stroke. Psychosocial disability is more prevalent than physical disability in victims of stroke[9]. The nature of motor and perceptual functions predicted self-care status at two weeks with 70% accuracy[10]. The cognitive deficits have a twofold effect. First, the deficit of new learning or memory can impair the daily functioning of the patient. Second, cognitive deficits can hinder the patient from profiting through other rehabilitation services. Cognitive deficits have a strong bearing on psychosocial outcome such as efficiency in the vocation, interpersonal relationships, leisure time pursuits. Thus, cognitive deficits by virtue of their link with psychosocial disability have an important bearing on functional outcome of stroke.

The other common neurological disorder associated with cognitive deficits is epilepsy which affects 24 to 53 per 100,000 persons[11]. Memory deficit is a common, complaint in the epilepsy clinic. The etiology of cognitive deficits in epilepsy is multi factorial. Disease related variables such as high seizure frequency, early age of onset, longer duration, presence of EEG abnormalities and poly pharmacy are associated with cognitive deficits. These factors being enmeshed with the illness, the epileptic patient is likely to have cognitive impairment throughout the course of the illness. Even newly diagnosed epileptic patients report cognitive impairment[12]. Improvement of cognitive functioning in these patients would improve their over all functioning. In fact the twin goals of treatment for epileptic patients has been reduction of seizures and improvement of cognitive functioning[13]. Other patient groups who require neuropsychological rehabilitation are patients with brain damage due to infections of the brain, hypoxia, and toxicity. The psychological effects of brain injury amply demonstrate the need for neuropsychological rehabilitation. Neuropsychological rehabilitation is an essential component of neurological rehabilitation.

Nature of Cognitive Rehabilitation

Neuropsychological rehabilitation is the process through which cognitive functioning of brain injured patients are improved. Natural or spontaneous recovery initiates the process of restoration of function. The pace of natural recovery is fast up to three months and

gradually slows down, but usually lasts for a year. Neuropsychological rehabilitation facilitates the recovery in the early stages and mediates recovery in the later and chronic stages. The goal of neuropsychological rehabilitation is to restore the patient to the premorbid level of functioning to the extent possible. The aim is to improve the patient's cognitive functions in different areas. Improvement of cognitive functions is expected to generalise to everyday functioning of the patient. If the primary goal of restoring the patient to the fullest pre morbid level is not achieved, then secondary goals are set. This secondary goal would be to improve cognitive functioning at least to the extent of the patient becoming productive, and to be able to continue with family and social responsibilities to the extent possible. In case even this is not possible, the goal of neuropsychological rehabilitation is to provide cognitive orthosis or aids such as diaries and boards which would support the patient's day to day functioning. The overall aim is to help the patient to function optimally; to reduce the burden on the support system such that less assistance is needed; to develop skills and use environmental resources to overcome residual impairments [14]. In the past twenty years , cognitive rehabilitation has been used to reduce deficits in the areas of attention, memory, information processing, visuo spatial perception, visuo constructive abilities, planning and reasoning.

Methods

Neuropsychological rehabilitation techniques are based on the methods of restitution or restoration, reorganisation and compensation of the lost function [15]. The principle of restoration recognises that retraining of the damaged function, by either training it in its totality or training its component parts would improve the function. The principle of reorganisation targets at improving the function through alternate procedures. An example would be improving communication in aphasics by increasing the use of gestures. Compensation refers to the use of cognitive orthosis such as diaries as memory aids. Restoration of function should be tried as far as possible. Only when it is clear that further restoration is not possible, should compensation be provided.

Method of restoration

The method of restoration is based on the following principles: -

1) Brain plasticity as evidenced through neuronal plasticity and redundancy of functional neural systems mediates the recovery process. Recent functional brain imaging techniques on stroke patients have demonstrated alteration of functional representations mediating

in the recovery of motor functions [16]. Collateral axonal sprouting, disinhibition of silent synapses, denervation super sensitivity are some of the mechanisms for the recovery of function [17].

2) A componential analysis is required to understand brain behaviour relationships. The componential analysis implies that a complex psychological function such as attention, memory, language, visuo spatial perception can be broken into elementary processes or components [18]. These components are localised in different regions of the brain. Brain damage would impair some components. Consequently, the composite function is disturbed. Restoration of the damaged component would restore the impaired function. Hence a detailed neuropsychological assessment is required to arrive at a profile of dysfunctional functions and their components.

3) Once the composite psychological function is improved, the improvement would generalise across tasks to everyday behaviour. Hence improvement in real life is a natural consequence of improvement in the laboratory or hospital setting.

The procedures employed for restoration of function depend on the function damaged. The basic principle is to break down the composite dysfunction into as many components as possible. This is usually done through a theoretical analysis. The next step is to assess the components in order to find out which are the dysfunctional components. This is followed by first improving the dysfunctional component with the most pervasive effect, followed by improving other components. An example would illustrate this principle. A patient with head injury may present with the complaint of difficulty in concentration. Neuropsychological assessment would reveal attention deficits. Clinical analysis of the deficit and a theoretical analysis would reveal the attention components of alertness and focusing to be impaired. Alertness being a more pervasive function compared with focusing, alertness should be improved first. Focusing should be targeted after alertness has improved. Thus, sequencing is essential in neuropsychological rehabilitation. Sequencing implies that a lower level function is improved first. After the rate of improvement has reached a plateau a higher level function is targeted for improvement. Therefore, the therapist waits for maximum improvement of the lower level function before attempting to improve a higher level function. The sequencing of levels holds good within a function and across functions. If attention, memory and planning are impaired in a patient, attention would be targeted first followed by memory and then planning. Within the domain of attention, alertness would be targeted first followed by tasks to improve focused, sustained and divided attention in that order.

The method of saturation cuing is followed within each level of function. Difficulty of the task is matched to the patient's capacity. Task difficulty is progressively increased in step with the patient's improvement. In the initial levels cues are provided if necessary, which are gradually faded out[19].

Method of restoration of functional skills

Restoration of functional skills refers to restoration of the skills required in every day life. The traditional method has given retraining tasks in the laboratory with the expectation that the benefits of retraining would generalise to everyday life. Research has found that this generalisation of improvement does not always occur. Often restoration of function is specific to the laboratory setting. Therefore its clinical utility becomes questionable[20]. The functional approach to restoration uses the setting and tasks of the patient's life to restore function. The composite function that the patient has to perform is broken down into functional bits. Each part of the total skill is taught to the patient. An association between the practical loss and the theoretical brain function is not made. Hence a here and now approach is adapted. The patient's disability is corrected, rather than attempting to correct an impaired function. An example would be that if a student finds it difficult to concentrate on studies following brain damage, functional restoration would teach study skills; teach the patient to organise his / her day; encourage the patient to take frequent breaks, rehearse the material and to take notes. In the traditional computer based approach attentional training would be given with the hope that the improvement would generalise to the school situation. Restoration of functional skills has gained wide acceptance as a cost effective, practical and useful approach to neuropsychological rehabilitation[14,21].

Method of compensation

Compensation techniques are adapted when restoration of function is not possible. Compensation refers to those strategies which are external to the patient but help the patient in discharging the disabled function. The patient with memory deficits might keep notes of a meeting or note appointments in a diary. A patient with planning impairment could be taught to organise his / her day with the help of a relative, note it down in a diary and follow it. Patients with visuo spatial deficits can be taught to look for landmarks. Verbal labelling of the parts of a drawing is a good strategy to overcome visual memory impairment. Compensatory strategies have been used in the treatment of aphasia. One of these is the Intelligent Word Finder which is an expert system run on

a computer. It helped to find words on the basis of incomplete information using inference rules. If the anomic patient gave the first letter of the forgotten name and another word with which it is highly associated, it would output the likely names[22]. Bliss symbols are flash cards which contain symbols instead of words as the means of communication. Grammar and function words are also explained through symbols. Bliss symbols have been successful in the treatment of aphasics[23]. The bliss symbols were adapted to the micro computer. The language known as VIC flashed symbols on the computer screen. The patient interacted with others using these symbols. It has been successfully used in communicating with patients who had severe global aphasia[24]. Communication through VIC on a computer was successful enough to help a patient exchange jokes with the therapist[25]. COGORTH are computer program which help in the planning and execution of daily activities. There are instructional modules which assist the patient through step by step instruction and guidance. The daily activity is first analysed and broken down into small stages or steps. The program then builds an instructional module for each of the stages, which are strung together to execute the total task. This program was successful in helping a severely amnesic patient to cook[24].

Behavioural management principles of differential reinforcement is a compensatory mechanism for behaviour problems such as aggression or amotivation following brain damage. The method of compensation has a limited effect on the patient's recovery. Compensation does not improve the function but is a practical way of helping the patient to live with a residual deficit.

Indian Experience

Cognitive rehabilitation is implemented as a treatment program for patients with head injury since the past 14 years at NIMHANS. We started the program by attempting restoration of function. Information processing deficits in patients with head injury were helped by improving visual information processing [26]. Serial and parallel information processing and focused attention were improved. The program was computer based. Daily one hour sessions were required for a duration of 4-6 weeks. Our experience with over 200 patients has found this to be an effective program for improving post concussion syndrome, memory deficits, and even frontal lobe deficits following head injury [27, 28]. Subsequently we tried a program requiring very little instrumentation to treat attention deficits following head injury. Focused, sustained and divided attention were successfully improved through paper and pencil tasks [29]. Subsequently the areas of contextual encoding in memory benefitted by improving

automatic encoding of spatial, temporal and frequency information in patients with head injury [30]. Incorporating the functions of attention and memory we developed a home based cognitive remediation program [31]. The therapist would teach a task to a care giver on a weekly basis. The patient's relative or friend would administer these tasks to the patient at home. We have tried the method of restoration on detoxified alcoholics with residual memory deficits [32]. The patients showed improvement in their planning ability and in their memory. However the cognitive rehabilitation did not prevent a relapse. In children with attention deficit hyperactive disorder we have used cognitive rehabilitation to improve their functioning. The children's attention span increased, impulsivity reduced and their school performance also improved [33]. Currently studies are underway to test the efficacy of cognitive rehabilitation in the treatment of cognitive deficits in epilepsy. A controlled study is ongoing to test the efficacy of a program to improve cognitive deficits in the areas of attention, memory, information processing and regulation in patients with head injury. Our experience over the past 14 years has established cognitive rehabilitation as a routine treatment for clients with brain damage at NIMHANS.

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Aphasia Rehabilitation

N. Shivshankar

Introduction

Aphasia is an acquired language disorder caused by damage to language processing areas in cortical and subcortical structures. The etiology may be vascular, neoplastic, traumatic, post-surgical or infective in nature. The language deficits depend upon the site, type and extent of lesion in the areas of the brain responsible for language processing. The language processing includes both input (receptive) and output (expressive) modalities. The receptive modality includes predominantly auditory-verbal (auditory) and reading (visual) whereas, the expressive modality comprises predominantly of verbal and written mode. Thus, aphasia is an impairment involving individual's linguistic, communicative and language related cognitive systems irrespective of input or output modalities. Variety of speech and language dysfunctions can be seen involving these modalities either in unison or in combination.

Classification of aphasia is important for localizing the site of lesion as well as predicting the outcome and for planning the rehabilitation strategies. For a speech and language specialist the classification yielding information on localization and the nature of linguistic deficits become important to formulate aphasia rehabilitation and ofcourse, to predict the outcome of recovery. The Boston classificatory system based on the speech and language characteristic is being widely used (Table 1).

Aphasia rehabilitation is a team approach and the role of speech and language pathologist is of paramount importance. The nature of linguistic deficits seen in aphasics vary from mild deficits such as simple word finding difficulty to a very profound deficit in language processing. The type of linguistic deficits need to be assessed through formal language tests and informal observation of the language behavior. In the Indian context, generally used aphasia tests have been, Indian versions of Western Aphasia Battery (WAB) and the Linguistic profile test (LPT). The other tests of Indian versions such as Porch Index of Communication Abilities (PICA), Boston Diagnostic Aphasia Examination (BDAE) and Functional Communication Profile (FCP) are also being used though not widely.

Table - I : Speech-Language Characteristics of Aphasia Syndromes from the Boston Classification System

Aphasic Syndrome	Conversational Speech	Auditory Comprehension	Auditory Speech	Confrontation Naming	Reading		
Repetition					Aloud	Comprehension	writing
Anomic	Fluent, empty	Good to mild	Good abnormal	Severely abnormal	Good or abnormal	Good or abnormal	Good or abnormal
Broca's	Nonfluent	Good	Abnormal	Abnormal	Abnormal	Good or abnormal	Abnormal
Wernicke's	Fluent, paraphasic	Abnormal	Abnormal	Abnormal	Abnormal	Abnormal	Abnormal
Conduction	Fluent, paraphasic	Good	Abnormal	Usually good	Abnormal	Good	Abnormal
Transcortical motor	Nonfluent	Good	Good	Abnormal	Abnormal	Often good	Abnormal
Transcortical sensory	Fluent, paraphasic echolalic	Severely abnormal	Good	Abnormal	Abnormal	Abnormal	Abnormal
Mixed transcortical	Nonfluent with echolalia	Severely abnormal	Good	Severely abnormal	Abnormal	Abnormal	Abnormal

After Aphasia, Alexia, and Agraphia by Benson, D.F, 1979, New York : Churchill Livingstone, From "The Nature of Aphasia in Adults" by M.R. McNeil, in Speech, Language and Hearing : Vol. II. Pathologies of Speech and Language by N.J. Lass, L.V. McReynolds, J.L. Northern, and D.E. Yoder (Eds.), 1988, Philadelphia: W.B. Saunders, Reprinted with permission.

When to start therapy

Generally, the language evaluation and therapy should be initiated when once the medical status of the individual is stabilized. Till such time a bed side aphasia screening may be done to know the evolution of the aphasic syndrome. Thereafter, a complete assessment

involving linguistic functions should be administered to know the type and nature of linguistic deficit which would guide the clinician for planning the rehabilitation program. In the initial stages, as a part of the aphasia syndrome several catastrophic reactions are frequently expressed by the patients. These reactions are shocking for the family as well. Understanding of the patient's premorbid personality helps in counseling the patient and family members. Patient's psychological readiness is a crucial point for initiating the therapy program.

Variables contributing to the recovery

a) Spontaneous recovery

Neurolinguistic recovery occurring spontaneously following brain insult is attributed to regaining of functions by the structurally undamaged areas in the brain. Several studies indicate that this period ranges from two to six months. Despite the disagreement regarding the length of the period of spontaneous recovery, this phenomenon must be appreciated and should be accounted for in any aphasia treatment study. Controversies exist whether therapy during spontaneous recovery will enhance the recovery process. Number of research reports support the hypothesis that aphasia therapy definitely benefits individual if directed properly. Thus, therapy during this spontaneous recovery period may enhance the process of recovery. There is no doubt that substantial improve-ment may result from language therapy even though a ceiling is frequently reached and recovery remains incomplete.

b) Etiological factors and aphasia syndrome

It has been documented that aphasia caused by trauma resolves better than that caused by strokes. Within the stroke group aphasias caused by hemorrhages resolve better than those caused by thromboembolic disease. It has been found that different aphasia syndrome has differential recovery pattern. Anomic patients show best recovery. Patients with Wernicke's aphasia without jargon show better prognosis than those who present. Similarly, anarthria may be an hindering factor in the recovery process of expressive aphasia.

c) Other factors

Equivocal findings have been reported with regard to contrbutory effects of age of onset and educational level on aphasia recovery. It has been recognized that pre-morbid

behavior is an important factor in aphasia management. In a recent study it has been reported that increasing age, stroke severity and neuropsychological deficits significantly contribute to the recovery process [1]. It has been recognised that there is a need for studying the contributory effects of literacy, language and scripts structures, and other sociocultural factors, on brain-language relationship [2].

Medical management of aphasia

The medical management in aphasia has been met with limited success. Several neurobehavioral phenomena that may accompany aphasic syndrome such as, frontal lobe depression syndrome, catastrophic reactions or extreme anxiety or agitation are amenable to pharmacotherapy facilitating language therapy. These treatment should only be considered as a last resort when all other supportive and counseling services fail. With specific speech and language disorders associated with aphasia syndrome, no drug has been found to change the speech and language behavior significantly. Findings on bromocriptine therapy have been equivocal [3,4,5]. Despite the inconsistency reported in pharmacotherapy studies in aphasia, medical line of management of aphasia syndrome should be pursued. While drug therapy may not revolutionise the aphasia treatment, it may still be used as an adjunct to behavioral rehabilitation.

Role of speech and language pathologist

Since, the language is a learnt behavior and the brain areas have the innate ability to store this behavior, language remedial procedures in aphasics aid in either relearning or reorganizing the language behavior. Speech-language pathologists constitute an important member of the aphasia rehabilitation team, whose responsibility is to help aphasics to regain the language skills to the extent possible and to help the individuals develop strategies to compensate for deficient language skills. These are accomplished through individualized or group therapy programs either by adopting best suited language techniques or developing alternative mode of communication where verbal skills fail to reappear. Their treatment also focuses on helping families and other caregivers who are the key persons in any aphasia rehabilitation program, to make them communicate effectively with the patient.

Role of the community

The community has a major share in the management of aphasics like in any other areas of disability. The community should treat them with respect. Interaction with aphasics

may require the use of alternate modes of communication (gestural, writing). Certain modifications may become necessary for aphasics to perform their activities. They may require an alternate job placement according to the type of disability or barrier free environment for them to move freely. A humanistic approach is what is important for their rehabilitation. Both family and the patient require psychological and moral support from the community.

Efficacy of aphasia therapy

Whether aphasia therapy is efficacious or not is still the topic of debate in aphasia research. In a recent study the efficacy of aphasia therapy was evaluated as per the specifications laid down by the American Association of Neurology (AAN) 6. The study implied that language therapy does give substantial benefit to patients.

Aphasia treatment approaches

Two schools of thought exist with regard to aphasia rehabilitation. The school which believes in the hypothesis of "loss of language" incorporates re-education program in aphasia rehabilitation while the other believing in the hypothesis of "impaired access to linguistic function" incorporates stimulation techniques to reorganize the neural access to stored linguistic information. Generally, an eclectic approach would be ideal to reap the maximum benefits of rehabilitation.

General Approach

Schuell's stimulation approach : The premise for the development of this approach is in the belief that the language is neither lost nor destroyed in aphasia and thus do not believe in teaching or reteaching approaches. The approach lays a strong emphasis on auditory process as this is the process which is involved in the acquisition, processing and control of language [7]. Thus, the approach stresses on the maximum utilization of auditory mode. It employs strong, controlled and intensive auditory stimulation as the primary tool to facilitate and maximize the patient's reorganization and recovery of language. However strong the argument may be in stressing the importance of auditory modality in the therapy approach, a caution must be exercised for its judicial application. There are patients who demonstrate dispropor-tionately severe impairment of the auditory process (Wernicke's aphasia) who may not be a right candidate for this approach and may benefit when both auditory and visual modalities (gestural or written input) are

used in conjunction. Thus, auditory modality need not be used exclusively. One modality may be used to reinforce the other and combined auditory and visual modality may be especially appropriate. The auditory input may be optimized by speaking slowly, pausing at appropriate intervals, reducing the rate of phoneme production by prolonging the words and stressing on the key word.

General frame work of the tasks emphasizing auditory abilities include, pointing tasks, following directions, yes-no questions and sentence verification, response switching, repetition tasks, sentence and phrase completion, verbal association, answering wh-questions, connected utterances in response to simple words, retelling and conversational verbal tasks. The reading tasks include all that mentioned earlier in the written modality along with other tasks such as, matching of pictures to written words/phrases/sentences, reading in unison with the clinician, reading paragraphs silently followed by questions about the content, reading aloud and then retelling. Techniques for Writing deficits, can also incorporate many of the above mentioned strategies along with copying and dictation. The materials used with these techniques have to be graded in the order of difficulty and should be presented to the patient in that order. Hierarchical therapy plan should be developed for each patient depending on the level of their language function.

Cognitive domain model of treatment

It focuses on the utilization of strategies for divergent, convergent and evaluative thinking. Hierarchical presentation of the stimuli for eliciting the desired response have been stressed in this model. Methods such as specific cuing techniques, clustering, increased contact time with stimuli and organizational strategies have been suggested by various authors [8]. These procedures enhance the memory functions as well.

Language Oriented Treatment (LOT): The program is based on operant paradigm and the objective is to improve the language processing efficiency in both input and out put modalities [9]. The traetment plan focusses upon auditory and visual processing, oral and graphic expression, and gestural communication. The responses obtained from the patient for the stimulus provided by the clinician using any of the modalities mentioned, are monitored by the clinician to provide feed back. Learning of specific stimulus-response bonds is not the objective of LOT.

Functional approach

Functional communication treatment: This technique is considered as a supplement

or adjunct to traditional form of language treatment. The emphasis is to restore and recover important daily communicative skills. It aims to improve the patient's reception, processing and use of information that are important for conducting daily activities, social interactions and expressing psychological and physical needs. The major principle of this technique is to establish communication thus, underscores the importance of linguistic accuracy. It focuses upon the use of natural environment to facilitate communication and emphasizes on the utilization of patient's effective and efficient channel for building communication [10].

Visual Action Therapy (VAT) : Based on the findings that global aphasics do retain a rich conceptual system and some cognitive operations necessary for natural language lead to the development of VAT[11,12]. The VAT incorporates nonvocal-visual/gestural approach for re-establishing communication. The final version of VAT comprises of three sub-components viz., proximal limb VAT, distal limb VAT and bucco/facial VAT. The proximal and distal VAT procedures were developed based on the observation that proximal gestures are significantly easier to train than distal gestures [13]. The VAT procedures use real objects, line drawing of the real objects and pictures of the real objects for training, The training steps are hierarchically arranged from basic picture-object matching to the communicative tasks using self initiated gestures. The limb VAT is found to be effective in patients with severe degree of global aphasia associated with moderate to severe degree of intransitive and transitive limb apraxia. Similarly, bucco/facial VAT is effective in patients with severely limited verbal output while their auditory skills and limb praxis are moderately preserved or recovered.

Back to the drawing board program (BDB) : This program can be applied in patients whose verbal communication is severely impaired. The technique aims at improving aphasic communication through simple line drawing to express their feelings, needs and events. The patients are first trained to draw the line drawing of selected figures, actions and sequence of actions which are hierarchically introduced. Finally, the patients are encouraged to use this technique for everyday communications [14].

Promoting Aphasics Communicative Effectiveness (PACE) : PACE [15,16] is a pragmatically based therapy technique to promote communication through appropriate stimulation using both verbal and non-verbal strategies within the pragmatic context. The clinical observation supported by experimental evidence that aphasic individuals possess far greater communication abilities than what could be predicted from their performance on formal tests. They demonstrate adequate abilities to use contextual (linguistic and non-linguistic) information, processing of non-verbal communicative acts,

cuing by prosodic information, comprehension and production, and making inferences from the situational context [16].

The technique comprises of stimulus cards containing the drawing of everyday objects or action or words. In this technique both patient and the clinician are equal partners in the communication event. Each of them is expected to send messages across as they pick-up the stimulus card on turn basis. The stimuli is selected based on the functional level of the patient and the communication is reinforced. Linguistic accuracy is given importance.

Specific Approach

Voluntary control of involuntary utterances (VCIU) : The VCIU technique [17] is useful with severely nonfluent aphasics whose speech is limited to few stereotypic meaningful utterances. The technique takes the patient from automatic utterances level to a level where the patient could voluntary produce words. The technique incorporates all the words that patients produce them involuntarily and were shown again in the written format to be read by them. Those words which are not read properly are removed from the list and new words with some emotional loading are added to the list for the patients to read. During the sessions if patients produce any other involuntary meaningful utterances, they also should be incorporated into the reading list. Once the patients are able to read the words, they are shown the line drawings of pictures representing the words. The patients are encouraged to identify the drawings and if they failed, the words representing the pictures are repeated till they gain control over the voluntary production of words. Gradually, confrontation naming of pictures are introduced for voluntary verbal productions.

Melodic Intonation Therapy (MIT) : The research evidence of right hemispheric participation in music/melody processing lead to the development of MIT with the objective of enhancing the verbal reproduction in nonfluent aphasics whose speech is characterized by non-meaningful verbal stereotypes [18]. The MIT has been found to be an effective therapy tool with severe Broca's aphasia. The therapy requires preselected stimulus items with corresponding pictures. The stimuli should consists of high probability words with hierarchical control over the word selection with regard to phonological difficulty and the syllable length. The selection of phrases and sentences as stimuli should take language structure into account and be arranged hierarchically according to syntactic complexity (for example, imperative sentences are easier to begin with than any other

sentence type). With pragmatics in mind, the stimuli may consist of names of family members and simple sentences fulfilling functional communication. The words are first intoned slowly, with constant voicing, using high/low tones. Determining the rhythm, pitch and stress patterns before beginning the therapy session is important. As the syllable in the words/phrases/sentences are intoned, it should be associated parallely with tapping on the patient's hand. The procedures are arranged in an orderly fashion. The level I begins with humming followed by steps like unison singing, unison singing with fading, immediate repetition and answering questions. The level II starts with unison singing with fading, delayed repetition (the patient is asked to intone the target stimulus after a delay of six seconds following the presentation of the stimuli), and response to questions. The level III consists of delayed repetition, speechgesang (a technique in which the rhythm and the stress are accentuated while the intonational features are dropped and replaced by the constantly changing pitch of the normal speech) with fading, delayed verbal repetition (normal prosody items are presented) and response to questions.

Helm elicited program for syntax stimulation (HELPSS) : This hierarchically based technique is designed to train patients with agrammatism 19. Agrammatism is a characteristic feature of nonfluent aphasias (Broca,s, Transcortical Motor and Subcortical-anterior/putaminal). The task uses a story completion format to elicit 11 sentence types at two task levels - A & B, using multiple exemplars for each type accompanied by line drawing depicting the story. The hierarchical gradation of sentence types are in the order of imperative-intransitive, imperative-transitive, Wh-interrogative, declarative-transitive, declarative-intransitive, comparative, passive, yes/no questions, direct and indirect object, embedded sentences and future tense forms.

The treatment of aphasic perseveration (TAP) : Perseveration is a characteristic feature of the aphasic syndrome. It refers to an inappropriate continuation or recurrence of an earlier response presenting even after the withdrawal of the earlier stimulus. Recurrent perseveration is the most frequent type observed in aphasia (other two types being stuck-in-set and continuous). The TAP is designed for patients who exhibit atleast a moderate degree of perseveration on tests of confrontation naming [20]. The material for TAP consists of stimulus items which are either real or realistic substitutes. All items are accompanied with pictures. The selection of strategies for eliciting desired response and to inhibit the perseveratory behavior depends on the individual naming performance and responses to a particular stimulus. A time interval of five to ten seconds are allowed between the presentation of the stimulus and eliciting the response. During this period

the clinician should help the patient to inhibit any involuntary verbalization. The strategies used to elicit desired responses are; by providing - (1) Gestural cue of the object, (2) Tactile cue of the object by making the patient to physically manipulate the object, (3) Drawing cue - by drawing picture of the object or by making the patient to draw the picture, (4) Descriptive sentence cue - by describing the function of the object, (5) Sentence completion task, (6) Graphic cue - by writing the first letter or two of the word or by asking the patient to write the word and read aloud, (7) Phonemic cuing, (8) Oral reading - by reading the entire word for the patient, (9) Repetition, (10) Unison speech or singing (as in MIT).

Treatment for Wernicke's aphasia (TWA) : The technique is designed to improve auditory comprehension of aphasic patients. It is desirable to use this method in patients with moderate to severe degree of comprehension deficits accompanied by relatively good ability for word-picture matching and ability to read picturable words. In the beginning, the corpus of words as a base material for initiating the program are prepared on the basis of matching and reading tasks. The items are presented hierarchically beginning with word-picture matching followed by reading the word aloud, repetition of the word verbally presented, and spoken word-picture matching. Here, deblocking of auditory modality is achieved through the visual modality[21].

Controlled Auditory Signal Presentation Strategies (CASPS) : The technique aims at providing controlled auditory input to enhance auditory processing[22]. The technique has been tried in patients with Wernicke's aphasia. Since, the auditory linguistic signals often evoke irrelevant responses from the patients, this technique begins with the presentation of non-linguistic auditory signals as training stimuli. The training starts with matching of tapping interms of number of times the tap is heard as the clinician presents them sitting in front of the patient. Once the target behavior is achieved, the clinician withdraws the visual cues and presents the stimuli only through the auditory modality. The next step would be to train the patient to match the various pitch patterns hummed by the clinician. The patterns consists of combination of low and high pitch variations in a set of three signals. Example, low-high-low, high-high-low etc. Similarly, duration of signals are also varied in matching tasks. The duration pattern consists of clinician humming the signals by controlling the length and combining them in an unit of three signals. Example, long-short-long, short-short-short etc. This method is based on the pitch pattern and duration pattern tests used in central auditory test battery. Gradually linguistic units are introduced by the clinicians using various procedures described earlier.

Auditory Perceptual Training (APT) : This is an yet another technique being applied to improve auditory attention and perception in patients with finer deficits in auditory perception as seen in mild Wernicke's or conduction aphasia[23]. The procedure involves the auditory presentation of the target stimuli (foreground signal) to be identified by the patients in the presence of background distracting signal. The target stimuli include digits and words. The presentation is done via cassette tapes. The recording protocol is depicted in table 2. The background signal contains a passage on which the target stimuli are superimposed. In the beginning, to make the task simpler and easier, the background signal (a running passage) is recorded using a female (or male) speaker in the secondary language of the patient while the foreground signal (two digits series to begin with and later hierarchically extended upto four digits series) is read by a male (or female, in relation to background signal) speaker in the primary language of the patient. The patients are asked to identify the target signal after listening either by verbally uttering the signal or by writing down. As the training advances the task is made complex by gradually withdrawing the contrasts one-by-one. Firstly, the voice contrast is withdrawn and the signals are recorded by using single speaker. Later, the language contrast is removed and both the background and foreground signals are presented in the primary language of the patient. Finally, when once four digits series are reached for presentation and desired benefits are accrued, the digits are replaced by words and the same protocol is followed for recording and presentation. The responses from patients on this technique have been encouraging.

Other Approach

Reeducation Approach : It is a "bottom-up" approach to reteach or rebuild linguistic skills in the aphasic individuals. It involves extensive linguistic drilling from individual sound level to connecting sounds to form syllables and then to real words to sentences. Motor placement approach is practiced to make the patient to produce sounds/syllables/ words and eventually sentences. Programmed instruction approach is also adopted to teach syntax. In patients with naming difficulty retraining is done through the use of picture cards. The patients are made to hear, repeat, trace and read. The patients with comprehension deficits are approached through systematic drilling using both auditory and visual modalities. The patients who do not benefit from these traditional methods are taught alterna-tive form of communication such as manual sign language or artificial language.

Table 2: Protocol of recording for APT

Phase	Foreground signal	Background signal
I	The response eliciting stimuli (digits) are **spoken by a male speaker** in the primary language of the patient. stimuli are randomly superimposed on the background signal.	Distracting signal: a passage read by a **female speaker** in the secondary language of the patient.
II	The response eliciting stimuli (digits) are spoken by a male speaker in the primary language of the patient. stimuli are randomly superimposed on the background signal.	Distracting signal: a passage read by a male speaker in the secondary language of the patient. (Withdrawal of voice contrast)
III	The response eliciting stimuli (digits) are spoken by a male speaker in the primary language of the patient. stimuli are randomly superimposed on the background signal.	Distracting signal: a passage read by a male speaker in the primary language of the patient. (Withdrawal of language contrast)

Note: Voice contrasts are interchangeable.
To start with two digit series are used and later as training progresses three and four digits series are introduced in Phase I itself.

Digits are replaced by words (from bisyllabic to multisyllabic) and the same protocol is repeated.

Computer aided aphasia treatment : Rehabilitation of aphasia using computers have demonstrated its tremendous application. A global improvement with generalization to untrained items and untreated oral naming deficits has been reported using computerized

written naming program in patients with naming difficulty[24]. Using a computerized visual communication system in expressive aphasics has revealed a significant improvement in verb retrieval and production of correct tense forms with good generalization effect[25]. It has also been reported that computer aided rehabilitation program is effective even in patients with long standing severe communication disability, and regardless of type and severity of aphasia[26]. Computer based reading training with chronic aphasic adults has also shown to be effective[27]. However, controlled efficacy studies are required to document the effectiveness of using computer in aphasia treatment although data available are encouraging. This would pave way for the development of programs on sound theoretical principles and neuropsychological models[28].

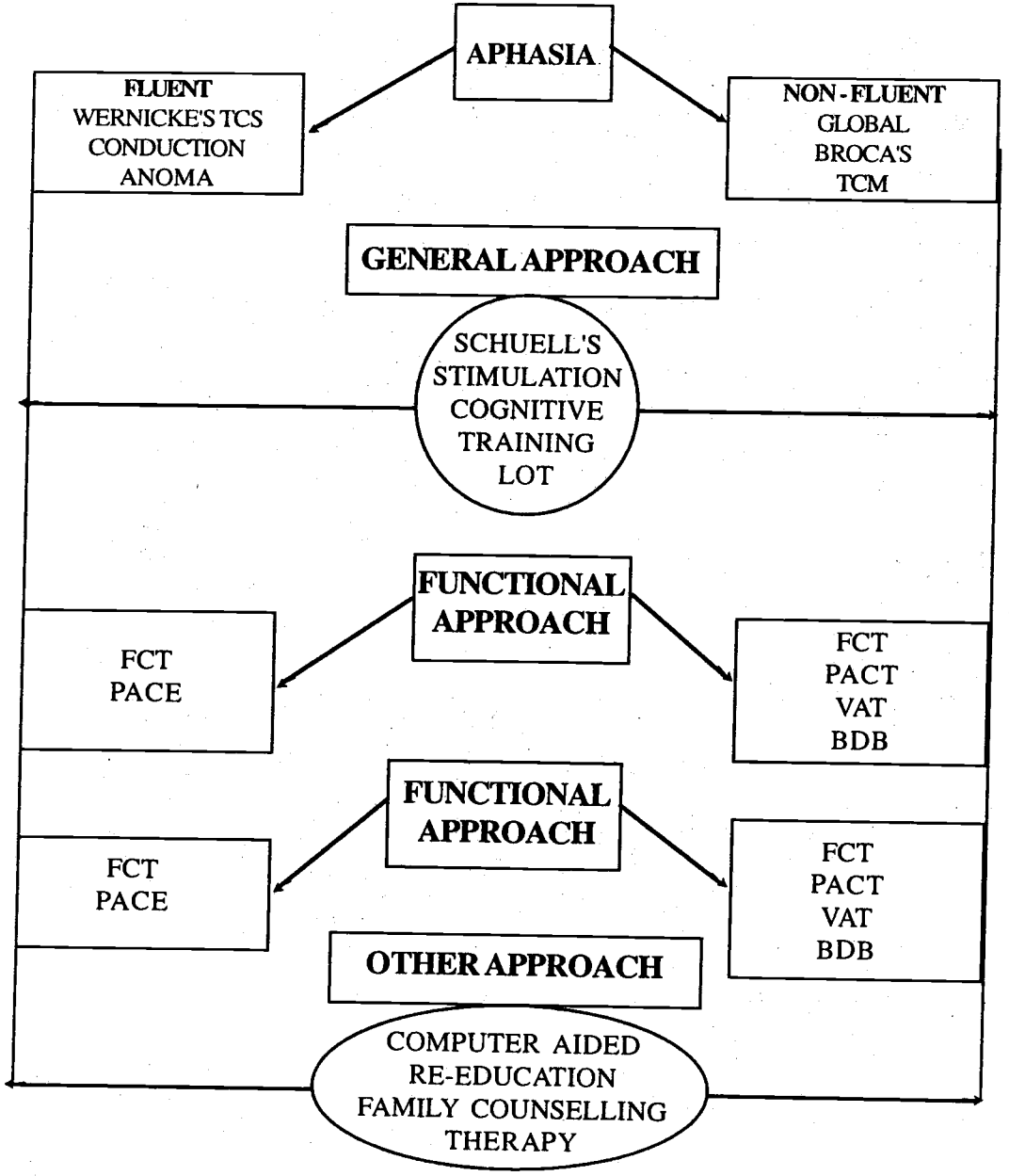
Family Counseling Therapy

Aphasia is a disability that disrupts the family organization and living with such a member is challenging for both the family and the affected individual. It is a sort of catastrophe for both the family as well as to the individual when once the deficits in the affected individual comes to the surface. Its impact is devastating and is multifaceted, more so on the economic front if the individual happens to be the bread winner. The communication deficits are so varied in them that the family is pushed into confusions and finds it difficult to cope up. Family therapy is important in aphasia rehabilitation as the prime concern is to provide stimulating and cohesive environment to the aphasic patient to help in regaining language functions. Thus, strong counseling is an integral part of the aphasia management program and is the initial step in the rehabilitation process to educate the family regarding status of the patient and the communication deficits. The family need to be taught how to communicate with the patient, to develop better interaction, to reduce catastrophic behaviors and to motivate the patient, and to keep the self of the patient in high esteem. The family members have a greater role to play and can influence the outcome of treatment services.

Concluding remarks

The purpose of this paper is to provide the basic information about the methods and strategies available for the rehabilitation of aphasics. The techniques that have been mentioned are not exhaustive. They have been drawn from various resources and from the author's own experience. For further reference to well designed therapeutic approaches , the reader is advised to refer to the article published by Holland et., al[6].

Figure 1. Aphasia Rehabilitation Technoques



P.S. Abbrevations in the diagram correspond to that used in the text.

A flow chart (Fig 1) is provided here as a ready reckoner to get an immediate idea of which technique may best suit what type of aphasic disorder (fluent or non-fluent). Many of the techniques mentioned here demand atleast a minimum level of education as they include reading and writing as input and output modalities. Thus, some of the techniques certainly does not suit our illiterate population as it is and may have to be modified for its best suitability. At times the clinician may have to develop an indigenous technique to suit the individual's need. As a general rule, a "child approach" of language training with adult aphasics should be avoided. Caution should be exercised while selecting the stimuli keeping the sociocultural, educational and professional background of the patient in mind. In some instances technique may have to be either dropped when it failed to demonstrate any change in the language behavior of the patient or may necessitate a change in the technique itself. As an end result we are interested in re-establishing communication in aphasics and hence "openness" in our approach is important. Our objective is to maximize the overall abilities of aphasics despite the disability.

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Psychosocial aspects of neurological rehabilitation

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Introduction

Psychosocial factors play a crucial role in rehabilitation of patients with neurological disabilities. These factors may contribute to initiation or exacerbation of illness and interfere with treatment and rehabilitation. Patients with neurological disabilities and their family are usually confronted with drastic changes in the life style. The patient may be unable to communicate, move or perceive and may lose control over bowel, bladder and sexual functions. Neuronal damage can cause cognitive and behavioural changes. All these can result in psychiatric morbidity in both the patient and family that require pharmacological or psychological intervention.

Psychosocial factors and Neurological disability

The psychosocial factors affect the course of neurological disorders in several ways. Habits like smoking, alcohol and substance abuse can cause neurological impairment. Psychological factors also influence how patients respond to their symptoms. Patients may neglect their illness because of depression, anxiety or personality problems. Lack of compliance, motivation, understanding, optimism and tolerance can interfere with treatment and rehabilitation. Living with neurological disabilities involves an element of psychological adjustment. Most people acknowledge the need to modify their lives by the demand of their illness.

Psychological factors affecting disability include age, personality, severity of the illness, expectations about outcome, attitudes of family and treating team, employment status and loss of control over environment and self[1]. Young patients with serious illness often react with resentment. They may regress to depressive state or object to the diagnosis. Elderly patients accept the medical problems but continue to fight their illness with stoicism. The negative attitude of the treating team influences the patient's reaction to illness. When the staff is calm, knowledgeable and reassuring, even the most anxious, resisting and angry patients can benefit. False hope of cure by the treating team lead to frustration and anger when the symptoms persist. Patients' emotional response to illness is influenced by the family concerns. Anxiety of the spouse is easily transmitted

to the patient. Complexities of the family dynamics affect the patient's behaviour The lack of privacy; fear of surgery or unfamiliar procedures and death in the same ward increase the problems. Job security and medical benefits available at the place of employment can help in reducing the psychological stress. Real or threatened loss of parts of body or body function, loss of social status, loss of future plans, home, job and occupation increase the psychological stress. Physical illness is particularly experienced as a loss when it interferes with social or occupational functioning, social status, impairment of particular skill and talent. Loss of love and approval, bowel and bladder control, sexual function and integrity of personality may also contribute for distress.

Psychiatric disorders in Neurologically disabled

Common psychiatric disorders seen in neurologically disabled patients include reactive depression (secondary depression), organic depression (endogenous depression), anxiety disorders, mania and psychosis. Reactive depression is a psychological reaction to the physical impairment. Organic depression can result from structural brain disease or from alterations of neurotransmitter mechanisms caused by drugs or biochemical disturbances. Clinically major depression is associated with longer duration of inpatient rehabilitation, deficient self-care and delay in resumption of premorbid social activities. It is found that the severity of depression does not appear to be related to severity of neurological impairment. Clinical features include: a) feeling sad and gloomy,b) loss of interest or pleasure in activities that are normally enjoyable, c) reduced energy leading to increased fatiguability, d) disturbed sleep and appetite, e) pessimistic view of the future, f) death wishes or suicidal ideation, g) crying spells h) non compliance with drugs or non adherence to treatment, i) lack of cooperation with treating team, j) reduced activity level and withdrawal, k) multiple body pain and i) self neglect.

Anxiety disorder is common in patients with neurological disability. It manifests as apprehension, worries about the future misfortune, feeling on edge, difficulty in concentration, motor tension, restlessness, fidgeting, tension headache, trembling, inability to relax and autonomic hyperactivity producing, sweating, tachycardia, tachypnoea, epigastric discomfort, dizziness, and dry mouth.

Mania is seen in patients with head injury , epilepsy and stroke. It manifests as: a) elevated mood or marked feeling of well-being out of keeping with the individuals circumstances, b) increased energy level c) increase in the quantity & speed of physical and mental activity, d) increased sociability, e) over talkativeness, f) overfamiliarity,

g)increased sexual energy h) loss of social inhibition, i) distractibility, j) grandiose or over-optimistic ideas and k) decreased need for sleep.

Psychosis is also seen after head injury and epilepsy. Clinical features of psychosis include a) delusions, b) hallucinations, c) irritability / anger outburst, d) thought disorders, e) disturbed sleep and f) loss of touch with reality.

Spinal cord injury

Immediate Psychological responses

Altered awareness : Only 50% of the patients were initially aware of a disability [2]. The initial awareness of the accident and it's sequelae are followed by a variable period in which individual is unaware of what occurs around him. Anaesthesia and shock may contribute to such unawareness apart from psychological defense.

Anxiety : It is the most common symptom because survival is the major concern for the patient and families. This is particularly so in patients with severe illness who require ventilatory support. Other common cause of anxiety is the abrupt loss of sensation over most of the body and inability to move body parts leading to panic and fear. Altered sensory input from the body, restriction of movements, isolation in the hospital, perceptual restriction results from monotony of the hospital routine and lack of intellectual activity leading to anxiety, tension, inability to concentrate and organize one's thoughts. More severe reactions are characterized by vivid sensory imagery and hallucinations.

Grief : Grief is the complex emotional response to loss. It may manifest as sadness, anger, guilt, anxiety and despair. According to Kubler-Ross[3] a typical grief reaction passes through the stages of 1) denial, 2) anger, 3) bargaining, 4) acceptance and 5) adjustment. Partial or total absence or distortion of the grief responses may predispose pathological grief reaction or depression.

Delayed responses

Depression : Depression is common in patients with paraplegia or quadriplegia. Some investigators believe depression is inevitable, some regard depression as a normal phenomenon as part of the phasic response to spinal cord injury. Sensory deprivation and insufficient arousal may result in lethargy and disinterest in the early post injury stay.

Few studies have systematically assessed the incidence of depression in spinal cord patients. Somasundaram et al[4] had found that approximately 70% of the patients had mild depression .In contrast to grief, depression is characterized by sustained lowered mood with sleep and appetite disturbances. Suicide and self neglect are not uncommon long term problems. Follow up studies have shown that 50% of spinal cord injury patients express suicidal thoughts. Maximum successful attempts were seen in the first five years of injury. Older and unmarried individuals are at greater risk. Wilcox and Strauffer[5] found that suicide or self neglect accounted for 43% of the 50 deaths recorded in patients with spinal cord injuries.

Alcohol and drug abuse may represent a maladaptive adjustment to neurological disabilities. Among patients with spinal cord injuries, 60% have a history of alcohol use disorders and 30%-60% have a history of drug abuse6. In addition, marijuana may decrease spasticity after spinal cord injury, thus reinforcing its use. It has been reported that 78% of patients undergoing rehabilitation who has a history of premorbid alcohol use, resume its use late in rehabilitation or few months after discharge[6]. Patients may use alcohol to alleviate the pain and to improve the sleep. Substance use may be a symptom of underlying depression.

Anxiety : The classic precipitant of anxiety includes helplessness, threat of impending injury, separation from secure environment, separation from loved one's, social disapproval and decreased self-esteem. Most of these factors are present when the person suffers from spinal cord injury. Anxiety may be expressed in the form of restlessness, hyperactivity, irritability, and verbosity. Sometimes anxiety builds to a level of panic with symptoms of hyperventilation, palpitation, tremulousness, cold and clammy hands and a variety of cardiac, pulmonary, and GIT symptoms. Somasundaram et al4 reported that 26% of spinal cord patients having severe anxiety.

Behavioural problems : Behavioural problems seen in these patients include aggression, yelling out and inappropriate sexual behaviour. These behaviours can be expression of depression, adjustment problems, anxiety, cognitive dysfunction or personality problems. In general only few patients of spinal cord injury suffer from these problems, but it disturbs the therapeutic alliances.

Pain : Chronic pain is a major sequelae to spinal cord injury. Christensen & Jenson[7] classified chronic pain into 6 types -root pain, segmental pain, phantom body pain, visceral pain, dysaesthesia and allodynia. Pain may occur, be prolonged or exacerbated

by psychological reasons. Psychological factors like anxiety and depression lead to muscle tension and result in pain. Less often pain may be due to hysterical conversion symptoms. Patients with chronic pain are often depressed. Psychological factors play a primary role in the etiology of pain in certain 'pain prone individuals'. Frequently they display marked hypochondriacal preoccupation and tend to deny life problems. Chronic pain is often reinforced by multiple secondary gains like care from family members, compensation and relief from responsibilities. Wagner Anke et al[8] studied the association between pain and quality of life, 46% experienced the pain of moderate to severe intensity and 70% scored high on GHQ (General Health Questionnaire)9 indicating psychological distress and reduced quality of life.

Body image disturbances : Body image is highly individualized, subjective and integrated sense of what one look and feel about self. Malformed bodies can trigger reactions including anxiety, depression, apprehension and obsessive ruminations. Bors[10] found distortion of body image in 100% of patients studied. These subjects had phantom limb phenomena involving cutaneous sensation , postural hallucinations and disturbance of proprioceptive body image. These disorders most often affect the lower limbs. Disordered perception of posture or movement, kinetic body image and disorder of perception of somatic size, bulk and continuity are also frequent. Patient often do not discuss these experiences and find them terrifying. The anatomical basis for disorders of body image following spinal cord injury is not clear.

Adjustment and coping with spinal cord injury

Spinal cord injury threatens the individual's well being at a number of levels. Apart from imposing physical and practical restrictions, it is a social stigma, it has sexual implications and it requires a radical revision in the person's self image. After injury he or she may pass through a number of phases, from shock to defensive retreat to acknowledgement and to adaptation. Full emotional adjustment may never be achieved for the minority of injured. One of the major problems that the spinal cord injured has to cope with is the attitude of other people. The disabled are often regarded not just as physically incapacitated but as ineffective in all ways. Some people have pity towards the disabled and disability gives them the licence to interfere even when help has not been requested.[11,12]

After injury people have to cope not only with the objective difficulties but also with their own feelings about their disability. They experience helplessness and frustration,

regret for what has been lost and anxiety about future. Individuals differ not just in the degree of impact of the disability experienced but in the way in which they qualitatively view this impact and attempt to minimise it.

Different patients use different defense mechanisms to cope with the injury. Many subjects cultivate an appearance of not caring, of being able to cope in order to hide their vulnerability from others. These patients use *suppression* as defence against showing their real feelings to anyone. In *denial* person refuses to acknowledge the fact of disability and its implications. They don't let themselves think too much about their feelings. They may be reluctant to associate with other disabled people or to be identified with the disability. They feel that they are the same as they were before disability and nothing has changed. Denial does not necessarily imply a distortion of reality. It can be a positive bid in the evaluation of situation and towards maintaining the status quo rather than making adjustment in the light of changed circumstances. Denial is a universal way of averting anxiety. *Acceptance* to disability means coming to terms with one's limitations. It means changing one's life style and limiting one's horizon's, learning patience and not wanting to do more than one can. This kind of attitude can bring equanimity and peace of mind.

Younger patients, women, satisfactory pre-injury life history and supportive interpersonal relationships, high ego strength, ability to delay gratification and conscientiousness are associated with good adjustment. Patients with little personal effort and ambition with psychopathic traits often cope poorly. Passive dependent personality predicts a need for longer term assistance with adjustment .Following appropriate grieving for loss of physical and associated social functioning, the spinal cord injured patient must develop a modified identify. Lack of motivation, dysphoric mood, death wishes and self neglect are suggestive of adjustment disorder.

Sexuality, Menstruation and pregnancy

Spinal cord injury affects the sexual functioning, with the nature and degree of effects depending upon the level and completeness of the lesion.[13,14,15,16] There may be problems in finding a comfortable and satisfactory position for intercourse. Spasm and incontinence may also interfere with sexual intercourse. Men may have difficulty in achieving and maintaining erection. Physical sensations will also be affected. Sex under these circumstances loses it's attraction. Few subjects withdraw from sexual activity totally. Those with loving and understanding partner with a spirit of compromise can maintain

satisfactory sexual relationships. The disabled person can focus upon those sensations and sources of pleasure which are still intact. In spite of the absence of genital sensation, other erogenous areas such as breasts, shoulders, neck and mouth may enhance the sexual experiences. There are reports of orgasm and phantom orgasm in paraplegics. The emotional fulfilment of the sexual act may be stressed rather than physical aspects. There may be greater feeling of involvement in ones partners' pleasure as opposed to the gratification of ones own need. For many people the greatest problem is the loss of confidence in their attractiveness as a partner and in their identity as sexual being as opposed to their ability to perform sexually. These self doubts and feelings of inadequacy can be more difficult to overcome than specific problems of sexual performance.

Women have been reported to have less impairment in sex role function and identification after disability than men. The adjustment may take several years. Sexual disability may lead to post injury divorce, further complicating adjustment. The woman's self concept and degree of perceived independence may have an impact on her participation as a sexual partner. Women must acknowledge that sexuality is an integral part of her being and be encouraged to experiment with their sexual expression. Orgasm may be described as similar to that of able bodied women or as a wide variety of psychological experiences such as pleasant, relaxful and glowing feelings. Vaginal lubrication may occur reflexely with lesion above T 9,not at all with lesions between T10 and T12 and psychogenic lubrication may occur in lesions below T12.

Amenorrhoea may occur following spinal cord injury although it's occurrence is not uniform and menstruation generally returns in 1 year. Reports of secondary amenorrhoea after spinal cord injury ranges from 50% to 60% . Women at or near the climacteric may become menopausal. Birth control is a complex issue after spinal cord injury. Intrauterine device usage is limited by the patients' lack of pain sensation. Oral contraceptives may further increase the risk of deep vein thrombosis. Pregnancy limits radiological evaluation and surgical treatment of spinal cord injury. Premature labour is more common. Breast feeding may precipitate autonomic dysreflexia.

Stroke

Psychosocial factors play a significant role in determining functional outcome following stroke. Educational status, acquired skills, premorbid vocational status, financial status, physical environment of home, type of community services available and family are some psychosocial factors determining the eventual functional outcome. The family should be guided to provide support, encouragement and realistic help to the subject

without over protectiveness or rejection. Since many problems are situational, interpersonal or related to family dynamics, psychosocial counsellors like psychiatrists, psychologists and psychiatric social workers are needed to provide help for victims of stroke and their families. The buffer to attenuate the patient's psychosocial distress may not be provided by the family alone There is a need for support from society to ensure that these patients' feel cared for, loved, valued and esteemed.

Depression : The prevalence of post stroke depression may vary from 2%[17] to 65%[18]. The peak incidence of depression is between 6 months to 2 years after stroke. The post-stroke depression may be due to organic alterations or a psychodynamic reaction to disability. There is a special relationship between stroke and depression. Stroke patients have higher incidence of depression than those with other medical conditions. Location and size of infarct influences depression. Left hemisphere lesions result in depression more often than right hemisphere or brain stem strokes.[19,20,21] Proximity of lesion to frontal pole has been found to be an important determinant of post stroke depression. This might be related to involvement of monoamine pathways. Serotonin receptor activation in uninjured right hemisphere may serve to ameliorate depression[22].

The factors important in determining the subject's reaction to stroke include age, sex, level of functional impairment, psychological defense, family environment, economic resources and social support. However, with the passage of time psycho-social factors become increasingly important and may obscure the effects of lesion per se on the mood. Post-stroke depression, apart from the classical clinical features of depression may also present as stoical attitudes, automatic patterns of behaviour, poor motivation for therapy and irritability. Management consist of anti-depressant drugs or psychotherapy or a combination of both. It is preferable to use the newer antidepressants which have less of anticholinergic side effects like fluoxetine or sertraline.

Cognitive impairments : Dementia usually occurs following multiple strokes whose deficits add up. Strokes within the dominant hemisphere are particularly liable to produce dementia. In multi-infarct dementia mental changes lag behind physical signs. Cognitive deficits may compromise attempts at rehabilitation. They cause difficulty in assessing the extent of impairment. Disturbance of language is a constant source of frustration for the patients and the therapists. Impaired comprehension, difficulties in communication, inattentiveness, perseveration and neglect due to disturbed awareness of self or of space may result in the failure of rehabilitation programme

Organic personality changes : Personality change secondary to stroke may persist or progress even after the physical sequelae improve. The changes outlined by Slater[23] include inability to adjust to new circumstances, becoming anxious or irritable over small matters, avoiding new experiences, stereotyped behaviour pattern, catastrophic reaction, irritability or abusiveness and heightened hypochondriacal concern. Constitutional predispositions may be accentuated and susceptible person may become suspicious or develop frank depression. These changes are important obstacles to rehabilitation and proper assessment and therapeutic interventions are warranted.

Psychosis : Hypomanic episodes have been reported after right hemispheric stroke.[24] Psychosis after stroke present with auditory and visual hallucinations, agitation, persecutory delusions and confusion. Levine and Finklestein[25] and Rabins et al[26] suggested that psychosis is associated with right sided stroke. Pure Hallucinatory phenomenon "peduncular hallucinosis of Lhermite." have been related to lesions in thalamus, midbrain and pons. Management would consist of appropriate anti-psychotic medications.

Other psychiatric disorders : Crying or occasionally laughing uncontrollably and without warning is an embarrassing and disabling aftermath of stroke. This is seen in 10-20% of stroke patients. The episodes are provoked by trivial emotional stimuli or on enquiry about the symptom. It is viewed as a form of disinhibition and is related to infarcts in anterior left hemisphere. Treatment includes anti-depressants or levodopa. Persistent agoraphobia and social withdrawal often associated with fear of looking conspicuous on account of disability has been reported.

Two types of behaviour might be seen in post stroke patients - unrealistic striving for independence and unrealistic dependency. Thus patient may deny his limitations or in his enthusiasm may interfere with progress. Sexual inadequacy, mainly impotence can be either a precipitant of depression or a sequelae of it. Marital discord secondary to constraints is another important sequelae and needs to be handled to ensure better quality of life.

Epilepsy

Psychiatric disorders seen in association with epilepsy include depression, anxiety, psychosis, personality changes and behavioural disorders.

Depression and anxiety : Depression and anxiety in their various forms are common in patients with epilepsy. A clear distinction between reactive and endogenous origins of anxiety and depression is difficult. In some patients both reactive and endogenous factors co-exists. The commonest causes for anxiety/depression are[27]:

1. anxiety and depressive reaction to acquiring the label of epilepsy,
2. anxiety and depressive reaction to social or family problems of epilepsy,
3. prodromal anxiety and depressive symptom before a fit,
4. anxiety and depressive symptom as aura,
5. depression/anxiety as an ictal experience,
6. post-ictal depressive/anxiety symptoms,
7. anxiety/depression occurring in association with epileptic psychoses,
8. true phobic anxiety related to seizures.

Psychosis : Psychosis in epilepsy may be ictal, post ictal or inter ictal in nature. Psychosis as a manifestation of seizure activity may be a continuous aura of a complex partial seizure. Post ictal psychosis is the most common form of psychosis in epileptic patients. Clinical features are aggressiveness and excitement. Hallucination are usually in auditory modality and delusions are usually paranoid in content. Post-ictal psychosis occurs with relative preservation of attention and orientation. Delusions and hallucinations are more structured and systematised. Mood alterations are common. There is typically an increase in seizure frequency, cessation of seizure with lucid interval of one to two days followed by psychosis. Spontaneous recovery is usual but episodes of psychosis often recur if seizure control lapses.

The prevalence of inter-ictal psychosis varies from 4.5 %[28] to 9.25%[29] . The psychosis is characterised by preservation of warm affect and personality with predominance of visual rather than auditory hallucinations. Formal thought disorder, incoherent thought, emotional withdrawal, catatonia and negative symptoms are less common. The possible risk factors are onset of epilepsy before 20 years, duration of epilepsy more than 10 years, antiepileptic polytherapy, high dose antiepileptics, left sided focus, temporal lobe focus and female sex.

Personality change : Subtle and more prominent personality changes have been frequently reported in people with epilepsy[30] . The clinical features include altered sexuality ,viscosity , hypergraphia, religiosity, obsessiveness, emotionality, impulsivity, anger, dependence, slowness and perseveration.

Aggressive behaviour : Aggressive behaviour in patients with epilepsy has been reported before, during or after seizures. The prodromal symptoms consists of irritability and verbal aggression. Ictal aggression is usually verbal or physical consisting of spontaneous, nondirected, stereotyped behaviour directed at objects or individuals. Aggressive acts during complex partial seizure can occur without provocation or as a reaction to an environmental stimulus. The post-ictal aggression occurs following generalized seizure or complex partial seizure. Physical restraint provokes the aggression which is usually terminated when restraint is withdrawn. Aggression also occurs with post-ictal psychosis especially in patients with paranoid delusions and threatening hallucination. Inter-ictal aggressive behaviour has often been reported in patients with temporal lobe epilepsy but remains controversial. Risk factors for aggression includes focal or diffuse neurologic lesions, cognitive impairment, medication such as barbiturates, male sex and violent behaviour as a child.

Family attitude towards patients with epilepsy: The family members response to the diagnosis of 'Epilepsy ' consists of denial, rejection or overprotection31. If parents use denial, they expect their child to be 'normal' in every way. This is often good as it encourages self-reliance and responsibility for managing their own treatment. But it can also be disastrous as child may not be able to compete equally with peers and fulfill parental expectations. Some families may use rejection. It leads to the exclusion of the patients by the family. He or she may be written off as unattainable or endurable, not worth wasting time on. Too low expectation of person's potential leads to problems at school and at work. At the other extreme the patient may be so watched over as to feel 'smothered'. Extreme reactions always imply intense anxiety on the part of the parents. Parental overprotection adversely affects the patients. They have undue dependence on family, reduced peer group interaction, limited development of social skills, problems on becoming independent as teenager and life long passive dependent attitude.

Social aspects of epilepsy: Epilepsy is often a secondary handicap. It is not the seizures but the attitude of society that cause great concern. The negative attitude of teachers results in difficulties for appropriate education. The negative attitude of peers results in difficulties in finding friends and leisure time activity. Public attitude towards epilepsy and misconceptions about this condition account for the difficulties experienced by the epileptic patients in getting employment[31].

Psychosocial handicaps of epilepsy: At an individual level the psychosocial handicaps of epilepsy includes low self-esteem, learned helplessness, external locus of control, dependent attitude, limited social skills, depression, pessimism and limited social orbit[31].

Education and epilepsy: A substantial portion of a child's life is spent at school and this environment will greatly influence the child's academic, emotional and social development. The majority of children with epilepsy are attending normal schools. According to National Child Development Study, UK, two thirds of affected children were educated in the normal school system. As a group, at age 11, their calculating, reading and general ability scores were slightly lower but within one SD of the mean of the study. This advocates that children even with special needs should be educated wherever possible in normal schools but there is no guarantee of educational success because academic difficulties can arise due to number of seizure related factors. An early age of onset and long duration of seizure is associated with poor educational progress. Children with C.P.S with the seizure arising from the dominant hemisphere perform poorly at studies. Frequent seizures have a detrimental effect on cognitive functions and scholastic performance. Occurrence of subclinical electric paroxysms as evidence of abnormal EEG impair the cognitive performance. Polytherapy regimens or high levels of antiepileptic medication, low expectations of parents and teachers, frequent and prolonged absences from school, low self-esteem, anxiety of the children and negative attitudes of the persons adversely affect the scholastic performance[32,33]. A minority of children with epilepsy may require some special educational input. The provision of specialized input will generally be made after conducting a multiprofessional assessment taking into account medical, educational, psychological and other factors including the view of parents. If the consensus is that the children has special educational needs, provision must be made to meet these needs .

Head injury

Psychiatric disorders and head injury: Head injury is associated with several psychiatric disorders34. The depression following head injury can be directly due to injury to the brain especially in the areas of left dorso lateral frontal and left basal ganglia lesion. It is of acute onset and tends to have a longer course Reactive depression secondary to sequelae of head injury is of late onset.Manic features have been found in association with right hemisphere injuries particularly with those involving basotemporal cortex or limbic structures. An increased prevalence of post traumatic epilepsy has been reported among patients with post traumatic mania,Patient has got high chances of developing psychosis whenever the period of coma extends beyond two days. It can occur when injury affects left frontal area, basal ganglia and both temporal and parietal areas. It may be difficult to differentiate from protracted delirium as both are characterised by hallucinations and fluctuating consciousness.

Head injury may cause an obsessional illness where patient experiences repetitive, uncontrollable, intrusive or irrational thoughts or doubts in clear consciousness.Persistent somatic symptoms like headache without any obvious cause can occur after recovery from head injury. Head injury also gives rise to emotional changes like irritability or depression and multiple somatic symptoms. Post traumatic stress disorder occurs due to excess and persistent anxiety after injury. There is reexperiencing of the accident, distress and avoidance of activity associated with head injury, difficulty in concentration and increased startle responses. Chronic agoraphobia, chronic paranoid delusional state and pseudodementia have been associated with head injury.

Behaviour and Head injury: Behavioral changes are most frequently seen when damage occurs to the frontal lobe. A combination of behavioural and cognitive changes can cause severe disability and adjustment problems.The personality changes can be egocentricity, loss of empathy, impatience, impulsivity, restlessness, impaired self control, silliness, irritability and altered sexual drive. Motivational disorder following head injury manifest as loss of initiation or extreme form of negative behaviour leading to a kind of drowsy, lethargic, passive and retarded appearance. The conceptual and behavioural rigidity is also a sequelae of head injury.[35]

Impairments are most frequently noted in social adjustments due to poor anger control. Patients tend to misjudge their deficits and try to resume their social and occupational activities prematurely. This will lead to embarrassment and frustration. Patients will be having problems in getting and keeping a job, making constructive use of one's own time and formation and maintenance of satisfying interpersonal relationships. They typically improve in the first 6-12 months after injury.In some patients these behaviours may persist. A head injured person should make social adjustments in the fields of activities of daily living, mobility, social relationship, leisure time activities and work.

Cognitive functions and Head injury: This is usually the most disabling sequelae of head injury. Certain domains of cognitive functions like memory are disproportionately affected by head injury. Memory is affected due to damage of neuronal structures like hippocampus and anterior temporal lobe involved in storage and retrieval of information. Recent memory is more affected than other forms of memory. Duration of the post traumatic amnesia in head injury is of prognostic significance. Recovery from post traumatic amnesia and ability to form new memories does not necessarily mean that patients learning and memory function has returned to normal levels.[36,37]

Profound attentional deficit is a common feature of head injury. It may represent a residual disturbance of consciousness and arousal. It can also remain as a long term sequelae. Vigilance and focused attention are affected after closed head injury. Impairment in the attention affects the speed of performance and complex decision taking abilities. Most common language disorder after head injury is anomia. Acute aphasic deficits due to primary left hemisphere damage have good prognosis. Putamen and internal capsule injury will give rise to post traumatic mutism. Right frontal lesion will result in tangential, socially inappropriate talk. Executive functions consists of abilities of initiation, planning and regulation of behaviour. Functions of information processing and motor speed are affected in head injured patients. They lack the mental flexibility to assess words, difficulty in generating their own models and ideas. Perseveration is a main feature.

Patients with minor head injury have impairment in memory and information processing. This improves in one to three months. Early cognitive impairment which is resolving is more suggestive of neurogenic origin. Chronic cognitive impairment is more likely to reflect depression and other psychological conditions. Repeated mild closed head injury may give rise to cumulative cognitive impairment[34].

The aim of Cognitive Rehabilitation is to train the patient in the use of strategies to improve performance despite impairments in cognitive functioning. External props like memory prosthesis, appointment books, reminder notes etc can be used to improve recall. Executive function deficits can be rectified by training to break down the tasks into simple component steps. Training social behaviour includes skills in topic maintenance, delivery of essential messages, and talking in balanced turns during conversation38.

Psychosocial interventions

Various psychosocial intervention techniques employed in neurological rehabilitation include pharmacotherapy, behavioural modification, family therapy, group therapy and psychotherapy.

Pharmacotherapy

When a patient receives multiple drugs for his neurological disorder addition of psychotropic drugs is known to cause interactions. The drugs used for psychiatric conditions should not interfere in the management of neurological disease. The drugs

used in the treatment of depression are tricyclic antidepressants (imipramine/amitriptiline) or selective serotonin reuptake inhibitors (fluoxetine/sertraline). The choice of drug depends on the side effect profile of the drug and the neurological symptoms

The drugs used in psychosis are either low potency neuroleptics (eg: - chlorpromazine) or high potency neuroleptics (eg:-haloperidol/resperidone). Extra pyramidal symptoms (EPS), dystonia and tardive dyskinesia (TD)are more common with high potency neuroleptics. In patients with brain disease, low potency neuroleptic are known to reduce the seizure threshold For anxiety the drugs used are benzodiazepines (alprazolam) or low dose tricyclic antidepressants.

Behavioural modification

Basic principles of behaviour modification are keep the environment simple, simple and concrete instructions, provide frequent and consistent positive feedback, avoid monotonous activities, breaking tasks into smaller steps,use the least restrictive setting and select appropriate target behaviour. Antecedent is the preceding event in the patient's environment that act as a cue to the individual.An antecedent event is followed by the occurrence of a behaviour. If the behaviour is chosen for modification either to increase or decrease, it is referred as target behaviour. It is categorised as: (a) excess (occurs too often) eg. anger outburst, impulsivity, socially inappropriate talk etc, (b) deficits (not occurring often enough) eg. communication deficits, (c) stimulus control disorders (behaviour at the wrong place/time/person) because of loss knowledge of the more abstract situation in which behaviour should occur. Behaviour followed by a consequent event that is going to affect the future rate, duration and intensity of the behaviour. Behaviour modification is done using differential reinforcement which consists of giving positive reinforcement for desired behaviour and withholding it in their absence.

Supportive and brief psychotherapy

Effect of stressors can be modified by the response adopted towards them. People acquire skills of mastery and adaptation during childhood and beyond. They are the personal and social resources to face a crisis. Responses that are directed at the reduction of stress or the resolution of a crisis are described as 'coping'. Brief supportive psychotherapy is helpful for patients in rehabilitation with physical disability. It helps the patient to explore different ways of coping with their disability and to select those that best meet their needs and resources.The objectives of psychotherapy are:

(a) promotion of the patient's best possible psychological and social functioning, thus enabling individuals to cope with psychological difficulties, (b) increasing the self esteem and self confidence, (c) minimising the impact of the threatening event and (d) transfer of the source of support from professionals to family and friends. The elements of supportive psychotherapy are reassurance, explanation, ventilation, guidance and suggestion.

Family and care givers

Family has to develop special coping mechanisms to deal with the acute and long term effects of neurological disorders[39,40]. During the acute phase family members experience the sense of relief if patient is out of danger. During the recovery phase the family must adjust to accepting the patient as permanently different. Care giver experiences burden and psychological morbidity, so attention to the caregiver's mood, function, health and family life is important. There is a direct relationship between the degree of caregiver's stress and the amount of help needed by the patients. The more dependent the patient more is the care giver stressed. Women are the predominant caregiver in India. They are expected to assume uncompensated care giving role to the dependent spouse, parents or children while continuing to perform regular home keeping.

In the general population, 25 % of the families function at a level that places them at risk for significant family problems if a family member becomes disabled. The care givers' stress is one of the major factors in institutionalisation of the elderly disabled. Families with disabled face changes in roles, leisure activities and health status of other members. Acknowledging that it is the family not just the patient who is disabled may produce anger and resentment. As many as 50% of the caregivers can be expected to develop major depression[38]. A large number of employed caregivers must quit their jobs to provide care. Families require education and follow up as knowledge regarding the illness will reduce anxiety resulting in better cooperation with the rehabilitation team. Little information is available regarding the effect of parental disability on children. If the children are less than 15 years old, they are likely to develop emotional problems and regressive behaviour.

In the first few days following the illness, families may experience a great sense of helplessness. Facilitating communication of the patients and family's need and promoting the ventilation of feelings may reduce the family's turmoil. The family should be encouraged to share feelings regarding the illness. The treating team should provide

explanation, understanding and support to the family.Education to the family members regarding the nature of illness and prognosis is essential.

Group therapy

Group therapy approaches used in rehabilitation settings include education group and family group. In group therapy patients and families have an opportunity to share their experiences, their sense of isolation and stigmatisation and educate themselves by learning from the experiences of other members of the group.Members of such groups respond to one another with a special sense of understanding and empathy because they share the unique experience of illness and its aftermath.

The concept of self help group for mental retarded and schizophrenic patients' families are already catching up in India, so it is important to develop self help group of the families of neurologically disabled. Further, this group can be a source of strength for programmes like community based rehabilitation because it is the time to take the rehabilitation of the neurologically disabled to the community at large.

Rehabilitation, outcome, and quality of life

King[41] mentions that 10 high ranked items for good quality of life (QOL) include health care, faith in god, spouse, family happiness, emotional support, friends, home, standard of living, children and family health. Several factors that modulate QOL are amenable to interventions like counselling.The techniques which promote healthy coping include discussion of perceptions of control, stress management and guidance in reappraising perceptions of being useful and ways of enjoying life. Family members play a critical role in promoting such behaviour change and must be included in interventions to facilitate healthy coping. Assessment of sexual concerns with appropriate education and counselling should be a included in the package. Since depression and social support are two most important predictors of QOL they must be attended to. Referral of patient to support groups and education of family members and the wider community of the importance of social support may help to strengthen support.

Other variables such as optimism, cognitive appraisal of the significance of disability, coping skills and family functioning are also predictive of QOL. A high ADL score on discharge is another variable indicating good QOL. Thus we find that psychosocial rehabilitation minimize disability, enhance likelihood of returning home and reduce social costs.

Conclusion

Advances in medical technology and treatment have extended the life expectancy of patients with neurological disorders. Quality of life is now an important issue for these patients. There are a growing number of patients who may need to adapt to severe and chronic disabilities. For many persons as their condition progresses or stabilizes, medical interventions have only a limited role. Long term management of the disability is likely to be carried out by the patient and care givers.Therefore, the patient's ability to deal with their illness at a psychosocial level becomes a critical issue. The members of the treating team need to be aware of these issues. In the Indian context psychosocial problems of the neurologically disabled has not been addressed. The family may find it difficult to give care due to psychological disturbance in patient and family, socio economic and environmental factors. A successful rehabilitation programme need to address these issues.In India Neurorehabilitation is in the stage of development. So far the focus was on medical rehabilitation and psychosocial aspects of disability were neglected.It is time to look into the psycho social factors for the successful rehabilitation of the neurologically disabled patient. Future strategies can be towards a community based approach, development of self help groups, empowerment of the community and development of a multidisciplinary approach to rehabilitation. It is necessary to sensitise various governmental and non governmental agencies and to ensure a good coordination among agencies. Public awareness on the possibilities of a long term rehabilitation for these disabled and provision of community supports are to be encouraged. With the present level of knowledge and simple intervention techniques in psychosocial rehabilitation, it is possible to alter the quality of life of atleast a portion of the neurologically disabled.

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Balance Rehabilitation

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Introduction

Balance problems are commonly seen in elderly individuals and patients with musculoskeletal and neurological disorders. The objective quantification of the balance has become more popular than the traditional subjective measures. For better balance rehabilitation, complex factors involved in maintenance of human balance are important.

Normal Balance

Balance control is an essential component for locomotion. It may be defined as ability to maintain function equilibrium. A body is said to be in a state of balance or equilibrium when the projection of Centre of Gravity (COG) falls within its base of support and when the resultant of all the forces acting on it is zero. Human balance is a complex process which involves the integration of sensory information from end organs to detect body position and utilization of this information by central nervous system (CNS) to produce adequate and proper motor output in the form of automatic postural responses. The sensory factors involved for the maintenance of balance are from visual[1], vestibular and somato-sensory (proprioception) inputs. The sensory information is integrated by CNS. The resultant discrete leg and trunk muscle responses maintain the COG over the base of support. Appropriate hip or ankle strategies are required to maintain balance depending upon the nature of perturbation of surface or sensory condition.

Biomechanically, a person has to move within " the cone of stability " to maintain balance. The cone of stability is an imaginary cone projecting upwards at an angle of 12.5 degree antero-posteriorly and 16 degree side to side. If the COG falls outside this imaginary cone of stability / limitation of stability (LOS) during upright standing or any other functional activities, there is a risk of fall or imbalance.

Common causes of balance disorders seen in rehabilitation practice are cerebrovascular accidents (CVA), Parkinsonism, cerebellar dysfunction, central or peripheral vestibular dysfunction, traumatic brain injury, orthopaedic trauma and old age[2]. In cases of total knee or hip replacement and amputation of lower limbs balance training is required to

facilitate proper weight bearing on the affected leg. In neurological diseases the equilibrium or saving reactions are often slowed or lost. Hence reeducation by facilitation techniques features predominantly in the rehabilitation process.

Standing balance: Upright stance control

Even during quiet standing, small adjustments are made especially around ankle joint to ensure maintenance of upright posture. These minute adjustments can be observed and measured using a force platform as postural sway [3]. The characteristics of standing balance in a subject are referred as postural sways. As already described, the maintenance of upright stance i.e. standing balance is an automatic active sensori-motor process which maintains the body's COG over its base of support. It is assumed that the triad of sensory inputs viz visual, vestibular, somatosensory form a multiloop control of postural stabilization 4 . However, it remains unclear whether the selection of these inputs occurs in a hierarchical way or not. It has been suggested that vestibular system is the most dominant of the three inputs in a normal subject and the visual system is responsible only for fine tuning of posture [5,6].

Assessment

Back ground of balance assessment

As early as 1853 Romberg reported measurement of postural sway. Non quantified assessment of balance were done in earlier times by observing displacement or alignment of segmental body parts and ability to maintain postural balance in progressively different conditions in sitting, standing, tilting in various direction.

Semiquantitative measures of stance were reported by Mithell & Lewis in 1886 by positioning patients in front of a grid pattern and observing sway. Hindsale in 1887 graphically record attaching a smoking paper to the top of subjects head (ataxia graph). In 1950, Miles used Ataxia meter to record sway on a kymograph using a pulley system attached to the subject's head. Hellebrandt in 1938 measured changes in COG in forward and lateral planes using a movable platform that recorded changes in the partial weight of the subject with a kymograph. Thomas & Whitney used a force platform in 1959 to record the horizontal forces of reaction at the feet and centre of foot pressure. Force platform have been used by various researchers. Quantitative measures of stance have been encouraged by the anticipation of the therapeutic measures having potential

effects upon the equilibrium of stance. Force platforms record the horizontal forces of reaction and these were basically developed to observe body sway.

Shumway Cook et al used static force plate system to examine postural sways in hemiplegic and normal subjects [7]. The effectiveness of centre of pressure (COP) biofeedback was compared to conventional rehabilitation therapeutic practices to establish stance stability in hemiplegic patients. The COP biofeedback was found to be more effective in reestablishing stance stability than traditional training methods. Stribely et al used a force platform and observed useful in providing quantitative measures stance [8]. They described steadiness scores, equivalent to the average absolute force in the forward and lateral direction. The increase in sway with the eyes closed recognised the importance of visual input in maintaining equilibrium.

Balance assessment in clinical practice

1. *Romberg test* - It was initially used to described qualitative information of visually detect oscillation of body segments in different in tabes dorsales by Romberg a German Neuro-pathologist, in 1853. The test is widely used in clinical neurology till today as "Romberg-test". It provides quantitative information by evaluating quiet standing with the eyes closed to differentiate sensory or cerebellar ataxia.

2. *Platform stability* - This technique can find out the quantitative measures of postural sway during quiet standing. Excursion of COP provides important information on sway characteristics. Force platform measure the vertical & horizontal components of forces acting on them. The point of application of the force on the platform is known as centre of pressure (COP). In a completely static situation the vertical projection of COG will be exactly equal to the position of COP. Human body is composed of many segments; The movement of these body parts produces accelerations measured as forces by the force platform.

3. *Computerized dynamic posturography (CDP)* - The CDP is the latest available system for objective evaluation of various components of balance in dynamic test conditions. Quantitative test results help us to identify the defective components that can be selectively used to facilitate balance rehabilitation and enhancement of compensatory mechanisms. Visual biofeedback training using volitional control of COG helps to develop automatic patterns. During training automatic control mechanism are provoked by working near the limits of stability [9].

Functional approach to balance rehabilitation

For the rehabilitation of balance problem it is important to understand the complex set of responses that are responsible for maintaining balance and interaction between various components. Balance disorders result from defective interaction of these components. For a successful balance rehabilitation training programme, it is mandatory to identify the defective components, primary and secondary effects of compensation and periodical monitoring of the effects of treatment on the patient's immediate performance needs. The various aspects of balance problems are: sensory problems, strategy selection, preparatory problems, sequencing and timing problem and scaling problem.

1. *Sensory problems :* Sensory problems for balance dysfunction may be because of vestibular (trauma/degenerative), proprioceptive (traumatic brain/spinal cord injury) or visual (as in elderly) systems. Loss of one sensory input may be compensated, but when two systems are faulty major balance problem arises. Management of sensory problems causing balance difficulty focuses on facilitation of damaged system and /or encouragement of remaining system. For example, proprioceptive system can be stimulated by standing eyes closed on a firm surface, shaking heads during walking. Similarly, narrow base standing, tandem walking, standing on rocker boards, walking on rough terrains and stairs help to promote visual and vestibular systems.

2. *Strategy selection :* Strategy selection problems as in Parkinsonism can also cause difficulty in balance. Such patients are unable to select proper ankle, hip or stepping strategies in response to environmental demands. In Parkinsonism the subjects have both ankle and hip strategies, but they are unable to use them in isolation. The timing, sequencing and amplitudes of strategy all appear normal. Management should try to concentrate on the strategy components i.e. hip and ankle strategies to develop control on the components separately at appropriate sensory input according to environmental demands (surface perturbations). To help patients formulate ankle strategy, subjects stand on board, firms surface, feet away from counter or wall on which he practices sway around ankle, joint, leaning forward/backward with hips and knee held in extension. For encouraging to stimulate hip strategy, training should be with the subject standing on a narrow or foam surface or over a slowly moving surface.

3. *Preparatory problems* - Preparatory problems arise when there is absence of normal anticipation/preparatory responses to a stimulus. Such patients (e.g. Parkinsonism) are treated by appropriate strategies (hip and ankle) by providing perturbation over the base of support.

4. *Sequencing and timing problems* - These problems arise when there is no normal sequential muscle contraction from distal to proximal around ankle joint in response to a perturbation. There is also no appropriate timing for muscle contraction i.e. unwanted co-contraction of muscle groups are seen e.g. in spastic hemiplegia, cerebral palsy etc. Treatment of sequencing problem includes practising appropriate strategies. The functional electrical stimulation can be used for sequencing of the muscle groups. The EMG biofeedback also can be used to encourage the patient to contract the muscle groups under training.

5. *Scaling problem:* The scaling problems arise when the amplitude of response does not fit to the stimulus. Cerebellar dysfunction (ataxia) patients have got the scaling problems with faulty amplitude of contraction leading to overshooting or undershooting of target. However, they have got adequate strategies and normal sequencing. Scaling problems are effectively treated with visual biofeedback training. When force platform or CDP is available, patient can be given training to control COG movements with visual feedback. Otherwise practising minimal motion while balancing over a rocker board may also be helpful to reduce large amplitude in scaling problems.

At present we have systems available for objective evaluation of various components of balance in dynamic test conditions. One of such system is posturography which is the science of recording change in body position as a means of assessing of change of body position for maintaining balance can be assessed with the help of moveable force plates to cause perturbation of body and resultant forces are recorded. CDP also allows assessment of effectiveness of training programme and modification of training programme, if required.

Advanced system of CDP has facility for sensory organisation testing (SOT). The SOT allows assessment of use of various sensory components of balance viz. visual, vestibular, proprioceptive by using disturbed sensory inputs. The SOT thus provides objective measures of COG control under varying sensory conditions. It is also possible to get composite scores and different test scores. It also allows sensory analysis and ratio which determines the use of somato-sensory, vestibular, visual inputs and use of inaccurate visual information. CDP allows sensory motor training by maximizing the use of sensory inputs. It also helps in stimulation of impaired sensory system and promotion of compensatory use of intact sensory system in cases of permanent impairment using visual biofeedback. Advantages of CDP for patients are visual feedback, clear and attainable goals, maintain attention and increase motivation. For

clinician instantaneous feedbacks of patient performances, objective measurement, customised treatment session and feedback on handling skills are advantageous.

Balance and mobility centre

There are already functioning units of balance and mobility centre in the Western countries. It consists of different specialities all coming under one roof: physiatry, otology, neurology, orthopaedics, geriatric medicine and occupational medicine. It has produced favourable outcome in the rehabilitation of many balance problems. The advantages are increased efficiency, reduced time for visiting several specialists and reduced cost of treatment, number of visits and test procedures etc. Balance and mobility centre tries to have a comprehensive assessment of all components of balance by integrating surgical, drug and rehabilitation management. The team is either led by a physiatrist or otologist for coordination of the team members. The importance of such centre in the prevention of falls in elderly population is already showing good results. With increasing geriatric population, morbidity following falls has been a problem affecting the quality of life in these population. With the introduction of prevention of fall programme on elderly incorporating balance rehabilitation concepts, there is reduction in morbidity from falls. Study of age and gender effects on postural control measures using CDP has confirmed moderate to good retest reliability by Hageman et al [2].The authors observed larger sway in older subjects with increased movement time, but did not find any gender effects on postural control.

Thus, recent advances in technology, particularly dynamic computerized posturography has lead to improvement in detection and quantification of balance problems. It provides information about contribution of various sensory inputs to imbalance in different neurological disorders. Further, this technique is also useful in the rehabilitation.

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Management of Spasticity

Michael P Barnes

Introduction

Spasticity is a major challenge to the rehabilitation team. Spasticity can prevent or hamper function, cause pain, disturb sleep, cause unnecessary complications and present major difficulties for care workers. This article reviews a variety of options available for the clinical management of spasticity. The need for clear treatment goals and robust outcome measures is emphasized. The initial management should focus on the alleviation of external exacerbating causes before specific treatment is considered. Physiotherapy is vital for correct positioning, seating, use of orthoses, splints and casts and for other antispastic measures such as use of heat and cold, ultrasound and electrical stimulation. The use of oral medication is discussed. Peripheral nerve blocks and botulinum toxin are two local treatments which are proving very useful and are under-used and under-valued techniques. In more severe cases intrathecal medication can be helpful. Surgical procedures such as rhizotomy and orthopaedic corrections may sometimes be necessary, but usually only for the most severe cases or in those who have been poorly managed in the earlier stages. Overall, the clinical management of spasticity often depends on a variety of different approaches necessitating the involvement of a comprehensive rehabilitation team.

Definition

Most physicians and therapists working with physically disabled people probably feel that they can recognize spasticity when they see or feel it. However defining it is much more difficult. Spasticity has been narrowly defined as a motor disorder characterized by velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks[1]. This narrow definition, however, does not do justice to the additional symptoms that there are often associated with increased muscle tone. Spasticity is usually accompanied by permanent voluntary muscle activation resulting in weakness and clumsiness of voluntary movements. The definition can be broadened further to include other positive symptoms such as flexor or extensor spasms and the "clasp-knife" phenomenon, exaggerated cutaneous reflexes and contractures[2]. Yet even these broader definitions do not give any flavour of the bewildering variety of spasticity that

can occur in different individuals and even in the same individual at the same time. The extent and type of spasticity can fluctuate widely according to position, fatigue, stress, drugs and even the weather. One limb may have one pattern of spasticity whilst another limb may have a different pattern. Spasticity is a very dynamic phenomenon and is a major challenge to the rehabilitation team.

Goals and Outcomes

The treatment of spasticity, like all rehabilitation processes, must start with the establishment of specific achievable goals and a carefully planned strategy to achieve those goals. The first question is, is it necessary to treatment the spasticity at all? Spasticity can be useful for the individual. For example, spasticity in a leg may serve as a brace to support the individual's weight for transferring or walking. People with spinal cord lesions can use flexor spasms to help them into a proper seating position or to act as an aid in dressing. Some consideration also needs to be given to the significant side effects of some treatments, particularly the weakness and fatigue induced by anti-spastic medication.

In general terms, there are three potential aims of treatment; to improve function, to reduce the risk of unnecessary complication and to alleviate pain. Occasionally, a justifiable aim is not specifically to help the patient, who may not perceive any problems with spasticity, but to make nursing easier for the main carers and to assist with the maintenance of hygiene, dressing and transferring.

Once a goal has been established then normally an outcome measure should be chosen that allows goal attainment and progress to be monitored. Most measures of spasticity are measures of impairment and not measures of disability or handicap. The clinical goal should have appropriate clinical outcome measures. For example, if the aim of treatment is to reduce pain, then little is achieved by monitoring the Ashworth scale. There are number of motor oriented disability scores that can be used to monitor functional effects of anti-spastic treatment programmes, from the broad based Barthel[3] to the more comprehensive FIM[4] to more specific walking or hand function tests[5].

Occasionally, specific assessment of spasticity is useful, particularly in a research environment. Unfortunately there are very few properly validated and reliable clinical assessments. The modified Ashworth scale[6] is the best known. However, there have been only two studies of reliability of this scale[6,7]. The original paper by Bohannon and

Smith6 was only concerned with the elbow joint and the second paper by Sloan and others[7] confirmed reliability for the elbow, but showed a rather poor interrater reliability for the knee flexors. Validity and reliability for this scale is not known for other joints. A variety of other assessment procedures are available. These tend to be either biomechanical and neurophysiological. In the former category, many attempts have been made to use mechanical techniques to measure torque/angle relationships with spastic joints during passive flexion and extension[8]. A valid technique for the measurement of spasticity of the knee is the pendulum test[9] which is a more sensitive measure of spasticity than the Ashworth scale. The pendulum test involves the appropriate joint, eg. knee, being held in extension and released and spasticity is measured as the rate of decay of oscillation of the limb. Obviously this is a difficult test to perform in smaller joints or in severe spasticity. Quantitative neurophysiological measurements of spasticity have largely been developed as research tools. Measurements mainly focus on characteristics of the H reflex, particularly suppression of the reflexes by vibration and by reciprocal inhibition. These techniques are well reviewed by Shahani and Cross[10].

The problem with biomechanical or neurophysiological measurements of spasticity is that such techniques are cumbersome and impractical in a clinical setting. They can also provide a false estimation of the functional effects of spasticity, as measurements are usually taken in static rather than dynamic situations. Practising clinicians, for the moment, will have to rely on the modified Ashworth scale in combination with practical, simple and preferably quick measures of disability related to the goals of treatment.

An approach to treatment

Alleviating and exacerbating factors: Spasticity should be seen as a symptom that has a variety of underlying causes. This is particularly important for people who are comatose, cognitively impaired or unable to communicate. Common causes for the onset or exacerbation of spasticity are urinary retention or infection, severe constipation, skin irritation such as pressure sores, or increased sensory stimuli from external causes such as ill fitting orthotic appliances and catheter leg bags. Occasionally, exacerbation of spasticity can indicate an underlying abdominal emergency or lower limb fracture, particularly in those who are unable to appreciate pain and not able to localise their problem.

Positioning and seating: Correct positioning, certainly for the immobile patient, is the most important aspect of management. Incorrect positioning in bed, particularly in the early stages after stroke or brain injury is a major cause of unnecessary spasticity. The supine position easily exacerbates extensor spasm by facilitation of the tonic labyrinthine supine reflex[11]. Similarly, a number of patients after brain injury exhibit an asymmetric tonic neck reflex which in the supine position will encourage a windswept posture characterised by asymmetric position of the pelvis with one hip assuming a flexed position in abduction and external rotation whilst the other hip assumes and abducted and internally rotated posture. This is a common cause of later orthopaedic problems, particularly subluxation of the hip on the abducted side[12]. It is often a matter of experimentation to find a posture that reduces spasticity. Side lying, sitting and standing can all be helpful in different circumstances with the primary aim of reducing spasticity and also producing stretch on the spastic muscles as well as facilitating the use of antagonistic muscle groups. Unfortunately, we do not know how long muscles need to be stretched in order to prevent contractures, although guidelines have been produced which suggest that each joint should be put through a full range of movement for at least two hours in every twenty four[13].

Proper seating is vital. The fundamental principle of seating is that the body should be contained in a balanced, symmetrical and stable posture which is both comfortable and maximises function. There are many different types of seating systems. All should have the ultimate aim of stabilisation of the pelvis without lateral tilt or rotation, but with a slight anterior tilt so the spine adopts a normal lumbar lordosis, thoracic kyphosis and cervical lordosis. The hip should be maintained at an angle of slightly more than 90° and this is often facilitated by a seat cushion with a slight backward slope. Knees and ankles should be at 90°. In people with severe spasticity, this posture may not be entirely possible or may require a variety of seating adjustments such as foot straps, knee blocks, adductor pommels, lumbar supports, lateral trunk supports and a variety of head and neck support systems. An adaptable and adjustable system is useful, particularly in people with complicated disabilities and in those with varied and changeable conditions such as multiple sclerosis[14].

Splinting and casting: The application of splints and casts can prevent the formation of contractures in the spastic limb and serial casting can improve the range of movement in a joint that is already contracted - a new cast being applied every few days as the range improves[15]. It is not known whether this is purely a mechanical effect or whether splinting actually reduces spasticity. There is conflicting EMG evidence [16,17]. Unfortunately, there is no clear agreement on the most appropriate design nor the length

of time a splint should be applied to give the desired effect. It is a field that requires much research.

Physiotherapy: A physiotherapist has a vital role to play in the assessment and management of positioning, seating, splinting and casting and the use of orthotic devices. However, do other physiotherapy techniques have an anti-spastic effect? Cold inhibits spastic muscles, but the effect is short lived, perhaps outlasting the application of the cold by about half an hour[18]. Paradoxically, heat is also used for relaxation of a spastic muscle[19]. Ultrasound is one way by which heat can be applied. Unfortunately, the anti-spastic effect is relatively short lived. Electrical stimulation has been used in some centres. Alfieri[20] found that ten minutes of stimulation to the finger extensors produced a decrease in spasticity and improved range of movement lasting for up to three hours. Scib and colleagues[21] have recently found that surface electrical stimulation of the tibialis anterior muscle has an antispastic effect that last for upto twenty four hours. Potissk and colleagues[22] have confirmed similar findings with the use of TENS machine, but the effects only persisted for up to forty-five minutes. Unfortunately, the role of electrical stimulation and other related techniques, such as EMG biofeedback and electrical vibration is still not clear. None of these appear to have much long term benefit but can have useful short term effects, particularly when used as an adjunctive treatment with other measures, such as the fitting of orthoses.

There are a number of different physiotherapy techniques such as Bobath[23], proprioceptive neuromuscular facilitation[24] and the Brunnstrom technique[25]. All claim an anti-spastic effect. There is, however, little evidence that any particular technique is better than the other for the management of spasticity. Larger scale prospective and controlled studies or single case studies are urgently needed to address this question.

Oral Medication: Oral anti-spastic medication has very limited use in the overall management of spasticity. All available agents are limited by their side effects, commonly drowsiness and weakness. The most widely used anti-spastic drug is baclofen, a GABA-B receptor agonist that probably has other pre-synaptic inhibitory effects on the release of excitatory neurotransmitters such as glutamate, aspartate and substance P. It is a commonly used drug, but it is interesting to note that there is in fact no convincing evidence of efficacy in spasticity disorders of cerebral origin[26]. It should be administered in divided doses as it has a short half-life. A total daily dosage of up to 80 mg is standard. There appears to be little benefit of increasing the daily dose beyond this level. In addition to the usual problems of drowsiness and weakness it can occasionally induce hallucinations.

An alternative agent is dantrolene sodium, which has a peripheral mode of action via a direct effect on suppression of release of calcium ions from the sarcoplasmic reticulum of muscle with consequent inhibition of excitation, contraction and coupling[27]. It is an effective anti-spastic agent if introduced in slow incremental stages up to a maximum of 400 mg daily in divided doses. As well as the usual problems of drowsiness, weakness and fatigue, there is the additional potential complication of impairment of liver function which necessitates monitoring liver function tests.

Diazepam is the earliest anti-spastic agent introduced and, whilst effective, it has serious problems of drowsiness and fatigue and is rarely of benefit to the patient[28]. A new agent, appears to take effect by preferential inhibition of poly-synaptic spinal excitatory pathways. The drug also has an effect on stimulation of alpha 2 receptors[29]. It is certainly comparable to baclofen as an anti-spastic agent and indeed may even be slightly superior[30]. 54 Whilst sedation, weakness and dry mouth can be problematic, tizanidine may exhibit slightly less side effects than alternative agents.

A number of other anti-spastic agents have been the subject of small scale studies. None have really stood the test of time, or at least have not been subjected to larger scale evaluation. Clonidine[31], glycine[32], threonine[33], tetrahydrocannabinol[34] and orphenadine[35] are a few examples of drugs that probably do have an anti-spastic effect, but whose place in the overall management of spasticity has yet to be determined.

Nerve blocks: Khalili and others[36] were the first to describe the use of phenol for selective peripheral nerve block, by a percutaneous approach. A surface electrode is normally used to locate the peripheral nerve. A needle with insulated shaft is then used as an exploratory electrode and the needle tip manipulated until a good muscle contractile response in time to the needle stimulus is observed. At this stage the phenol is injected. Any accessible peripheral nerve can be blocked in this manner. The obturator is probably the commonest and most accessible nerve and gives rise to very satisfactory reduction in abductor spasticity. The posterior tibial nerve is also a useful injection site for the relief of calf muscle spasticity and often abolishes troublesome clonus or facilitates the fitting of an ankle foot orthosis. Hamstring spasticity can be alleviated by blocking the sciatic nerve or possibly the branches to the hamstring muscles themselves. It is less easy to block the many branches of the femoral nerve to the quadriceps muscle and equally difficult to locate the nerve supply to the ileopsoas for the relief of hip flexor spasticity, although some authors have described a paravertebral approach using radiological control. It is also possible to block the median and ulnar nerves as well as

the musculo-cutaneous nerve for the relief of flexion spasticity at the elbow. The side effects of this technique depend on whether a mixed motor-sensory nerve is blocked with the consequent risk of dysaesthesia or whether the block is confined to motor end points. The most common problem is obviously loss of motor function but if there is any doubt as to the potential functional effect of the nerve block then bupivacaine should be used before definitive block with phenol or alcohol. The incidence of dysaesthesae is highly variably and reported from 3-32% but fortunately this complication usually consists only of a transient burning sensation lasting a few days although occasionally more persistent dysaesthetic pain can result. Damage to the local structures is possible and local pain, oedema and infection have been reported, but fortunately rarely. Overall, phenol and alcohol nerve blocks are useful for focal spasticity either as definitive procedures or as an adjunct to other technique[37,38].

Botulinum toxin: Botulinum toxin type A produces dose related weakness of skeletal muscle by impairing the release of acetylcholine at the neuromuscular junction. It is now an established first line treatment for focal dystonia. A number of studies have now shown that the botulinum is useful for the management of spasticity[39,40]. It is particularly helpful for spasticity in the leg adductors, calf muscles and the upper limb flexors. The technique is simple - botulinum is diluted in normal saline and is injected intramuscularly. In readily identifiable and palpable muscles no EMG identification of the muscle is normally required. The dose varies considerably according to the bulk of the muscle and the number of muscles to be injected. An average dose in unilateral leg adductors is approximately 500 Dysport units or about 150 Allergan units. Botulinum toxin injection is a safe and effective technique with very few side effects reported in the literature. Occasionally, weakness of the injected muscle or of neighbouring muscles can be problem, but no general systemic weakness has yet been reported. There are some disadvantages to the technique including the cost of the toxin, the need for repeat injections every two to three months and a risk, albeit small one, of developing antibodies. Fortunately, there are seven types of botulinum toxin and at least two other types that are now being developed for commercial use which should get round the latter of the problems.

Overall, botulinum toxin is now an established adjunctive treatment for the management for focal spasticity in the adult. It probably also has an important role to play in the management of spasticity in cerebral palsied children. It has been shown to improve gait and reduced the need for multiple surgical procedures[41].

Intrathecal techniques: There has been much interest in recent years in the use of intrathecal baclofen for the treatment of more resistant spasticity in the lower limbs. The technique was first described by Penn and Kroin in 1984[42]. Surgical details of the technique can vary but would normally involve implantation of a subcutaneous pump to allow programmable intrathecal delivery of baclofen via a silastic catheter. The baclofen can either be administered in regular doses or by continuous infusion. The daily dose needle adjustments according to clinical effect but would normally range from 50-1000 micrograms per day. Both short and long term efficacy have been confirmed in a number of studies. For example, a recent study[43] demonstrated complete abolition of spasticity in 28 patients who were previously unresponsive to oral baclofen and other anti-spastic medications. The follow-up period of this study was up to two years but averaged eight months. The only significant complications were related to technical problems with the pump device and included one pump failure and two catheter replacements. There is a risk of the pump delivering an overdose of baclofen. Tolerance is a possible but rare occurrence. A similar technique using intrathecal morphine injections has been described and this remains an alternative.

The first description of intrathecal injections for the relief of spasticity was kelly and Gautier-Snmith in 1959[44] who used phenol and glycerine injection. Although this technique is now largely unnecessary it is worth remembering for people with severe and resistant lower limb spasticity. It is effective but is likely to damage sacral nerves and should therefore be restricted to those who already have irreversible faecal and urinary incontinence. Sensation is also likely to be abolished with the consequent increased risk of pressure sores. However, for those already paraplegic and incontinent it can be a successful technique both to relieve spasticity and more particularly to relieve pain from spasticity.

Surgical and Orthopaedic Procedures: There is rarely a need to resort to surgical procedures for the management of spasticity, except in the occasional severe and resistant case and for the management of fixed contractures. A few techniques, however, are still useful and should be mentioned.

Anterior and posterior rhizotomy have been performed for many years for the treatment of severe and resistant spasticity. A more refined technique has been pioneered by Sindou and Jean Mondo[45] - a microsurgical lesion in the dorsal root entry zone (DREZ-otomy). The procedure can be used for both upper and lower limbs and the authors have reported consistently good results with minimal morbidity. The less invasive

technique of percutaneous radiofrequency rhizotomy[46] has also been described as a relatively simple procedure with an apparently high rate of efficacy but with a small risk of recurrence. The author has not had to resort to surgical advice for the management of spasticity for a number of years but these techniques should be borne in mind for the severe and resistant case.

Spinal cord and cerebellar stimulation have been reported to be effective in reducing spasticity but unfortunately the effect tends to be weak and relatively short lived. The treatment is also time consuming and expensive with some risk of equipment failure and electrode movement. However, it is a technique that should be recalled there is resistant pain as a result of spasticity[47].

Occasionally, surgical repositioning of joints and limbs can facilitate proper seating and ease positioning and the application of orthoses. One of the more common orthopaedic interventions is the use of one of the various Achilles tendon lengthening operations for a fixed equinus deformity often with associated correction of a varus deformity. Hindfoot varus is normally caused by spasticity of the tibialis posteior whilst mid-foot varus is normally caused by tibialis anterior spasticity. Often equino varus deformity needs the combination of Achilles tendon lengthening, tibialis posterior lengthening and split anterior tibials transfer procedures sometime in combination with lengthening of the toe flexors. Similar lengthening procedures can be undertaken on the hamstring muscles although some surgeons prefer hamstring tenotomy and transposition. Hip adduction deformities are relieved by obturator phenol nerve blocks or botulinum injections but sometimes obturator neurectomy or adductor tenotomy can be carried out in more resistant cases. Hip flexion deformities are a problem that are not readily amenable to non-invasive techniques and, although not always successful, iliopsoas procedures can be performed.

Conclusions

The management of spasticity is complex. Most individuals, even with quite severe spasticity, can be managed by a combination of physiotherapy and local nerve block or botulinum injection, sometimes combined with relatively low dose oral medication. The use of more advanced intrathecal and surgical techniques is rarely needed unless complications have arisen, often due to inappropriate early management. Spasticity requires the input of a full rehabilitation team, involving in particular a physician, orthotist and physiotherapist. Despite the complexities, the management of spasticity can often yield rewarding results and lead to major improvements in quality of life.

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Neurogenic bladder : Evaluation and management

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Introduction

Several neurological disorders cause urological symptoms and urinary problems aggravate neurological disability. Management of Neuro-Urological disorders require a proper understanding of physiology of micturition and effects of neurological lesions on urinary tract functions. A team consisting of neurologists, urologists, gynaecologists, incontinence counsellors and nurses are required for comprehensive Neuro-urological rehabilitation. Currently several accurate and sophisticated techniques are available for evaluation and management of Neuro-urological disorders.

Neurophysiology of Micturition

The function of lower urinary tract is to store and voluntarily release urine. The organs involved in this process are: bladder, the reservoir, and urethra and sphincters, the outlet. The functions of these organs are coordinated by neural circuits involving sympathetic, parasympathetic and somatic neurons. The preganglionic sympathetic neurons are located in the intermediolateral column of the spinal cord extending from first thoracic to second lumbar spinal segments. Their axons synapse with paravertebral and mesenteric sympathetic ganglia. The postganglionic fibres supply smooth muscles of detrusor and internal urethral sphincter. The beta adrenergic receptors are present in the bladder and alpha adrenergic receptors predominantly in the bladder neck and urethra. The sympathetic stimulation causes relaxation of the smooth muscles of the detrusor and contraction of the sphincters[1]. Relaxation of detrusor with contraction of the sphincters facilitates storage of urine at a low pressure.

The preganglionic parasympathetic neurons are in the intermediolateral column extending from second to fourth sacral spinal segments. These nerves travel through pelvic nerves to pelvic and intramural parasympathetic ganglion. The post ganglionic cholinergic fibres supply the detrusor and urethral sphincters. The cholinergic muscarinic receptors cause contraction of the detrusor and relaxation of the sphincters. The external urethral sphincter part of the somatic pelvic floor musculature is supplied by the pudendal nerve. The somatic motor neurons of pudendal nerve are located in the ventral horns of second to fourth sacral spinal segments.

(Onuf's nucleus). The external and internal urethral sphincters are tonically active and maintain continence.

Nociceptive and tension receptors are located in the bladder wall and urethra. Small myelinated A-delta fibres and unmyelinated C-fibres carry these sensations through pelvic and pudendal nerves to sacral cord. A small number of fibres also project through the hypogastric nerve to thoracolumbar spinal cord. The sensations of pain and tension are carried by A-delta fibres. The C-fibres are normally silent. They respond to stimuli only in patients with upper motor neuron lesions[2]. The afferent fibres from pelvic and pudendal nerves enter into Lissauer's tract and divide into medial and lateral collateral pathways. The lateral collaterals end in sacral parasympathetic neurons. The neurons in the medial collateral project to pontine micturition centre.

The micturition reflex has two phases: the filling phase and the voiding phase. During filling phase the intravesical pressure is low. This is due to the intrinsic compliance of the bladder, inhibition of the sacral parasympathetic neurons and stimulation of the sympathetic neurons. The activity of the sphincters also increases as filling progresses. When the bladder capacity reaches its threshold, the voiding phase begins. The A-delta fibres start firing. This in turn leads to relaxation of the sphincters and contraction of the detrusor. The flow of urine into the urethra further augments the detrusor reflex resulting in further increase in intravesical pressure. Relaxation of the sphincters in coordination with detrusor contractions results in voiding.

The process of micturition is basically a function of autonomic and somatic nervous systems. The different reflexes involved in this process are coordinated by various spinal and supraspinal centres. Pontine micturition centre (PMC)or Barrington's centre is located in dorsomedian pons. Virus tracing studies in rats have shown extensive labelling of PMC after injection into bladder, urethra and external sphincters[3]. The PMC receives inputs A-delta fibres from bladder and urethra. Fibres from PMC project to the sacral parasympathetic and Onuf's nucleus. Stimulation of PMC causes contraction of detrusor and relaxation of the sphincters. Lateral Pontine centre or storage centre increases the tone of the sphincters and decreases the activity of the detrusor[4]. The Pontine centres are inhibited by the neurons of the medial part of the frontal lobes. The striated muscles of pelvic floor is controlled by neurons of frontal motor cortex. Basal ganglion and Cerebellum also modulate the activities of pontine centres.

Bradley has described four neuronal loops which control the micturition. The first loop extends from medial frontal cortex, basal ganglion, thalamus and cerebellum to PMC.

This cortico-pontine-cortical loop is required for voluntary control of micturition. The loop 2 , the spino-bulbo-spinal loop, includes afferents from detrusor and sphincters to PMC and efferents from PMC to sacral parasympathetic and somatic motor neurons. This loop is responsible for coordinating detrusor contractions with sphincter relaxation. The PMC activates detrusor reflex of adequate duration to facilitate complete emptying. Loop 3 extends from detrusor to pudendal motor neurons. This loop causes a reflex inhibition of tonic urethral sphincter activity during detrusor contractions. Loop 4 A is the cortical -pudendal motor neuron loop. The pyramidal neurons from frontal motor cortex project through corticospinal tract to the sacral pudendal motor neurons. The proprioceptive afferents from the skeletal muscles project back to the sensory cortex. The receptors in the periurethral tissue project to the Pudendal motor neurons. Stimulation of this loop 4B result in contractions of the sphincters. The components of loop 4 are essential for the voluntary control of micturition[5].

Urological dysfunction in Neurological disorders

The process of micturition is coordinated and controlled by a neural network extending from frontal lobes to sacral cords. Lesions in any part of this circuit can result in urinary tract dysfunction. The urinary symptoms may be the presenting feature of a neurological disease or it may complicate an established neurological syndrome. The nature and the severity of the symptoms depend on duration, type, site and extend of the neuronal damage.

The urinary symptoms in patients with brain diseases may be due to associated cognitive, behavioural and communication problems or due to damage to specific areas of the brain involved in the control of micturition. In patients with frontal lobe lesions the incontinence may be part of a behavioural disorder or disinhibition. Subjects with aphasia may be unable to express the need to void resulting in incontinence. The incontinence due to cognitive, behavioural and communication problems is common in patients with stroke, head injury , dementia and cerebral palsy. Incontinence after stroke indicates a poor functional recovery[6]. Lesions involving cortico-pontine circuit release the PMC from voluntary control. This result in uncontrolled detrusor contractions with relaxation of sphincters. The symptoms include urgency, frequency, nocturnal enuresis and urge incontinence. Such patients will have insight and will be concerned about incontinence. Disorders of basal ganglia like Parkinson's disease and Huntington's chorea can cause urinary symptoms. Frequency, urgency, incontinence and hesitancy due to bradykinesia of the sphincters are common in Parkinson's disease. Huntington's disease can cause

detrusor hyperreflexia. Lesions involving PMC in dorsal tegmentum of pons result failure to initiate voiding and urinary retention due to lack of coordination between detrusor contractions and sphincter relaxations[7]. This is usually associated with Internuclear ophthalmoplegia. The common causes are multiple sclerosis, vertebro basilar stroke and brain stem glioma.

Urinary symptoms are common among patients with spinal cord disorders [2]. Immediately after the injury, due to acute spinal shock, the bladder will be flaccid. These patients suffer from retention with overflow incontinence. This may last up to six weeks after the insult. The spinal cord lesions above the sacral segments interrupt neural circuits connecting the sacral cord with PMC and other higher centres. The detrusor will be hypertonic and hyperreflexic and will contract without relaxation of sphincters. These patients suffer from incontinence, retention, frequency and incomplete voiding. Detrusor contractions against closed sphincters result in high intravesical pressures. This can lead to hypertrophy, trabeculation and diverticula formation in the bladder and hydrouretronephrosis and chronic renal failure. Presence of residual urine can cause recurrent urinary tract infections and stone formation. Damage to second to fourth sacral spinal segments or the spinal roots result in flaccid areflexia of detrusor without bladder sensations. These patients may suffer from painless urinary retention with overflow incontinence or continuous dribbling. This can lead to myogenic detrusor injury due to over distension and recurrent urinary tract infection.

Autonomic failure is a significant cause of bladder dysfunction. These patients suffer from poor detrusor contractions and loss of sphincter tone. They present with insensitive, large capacity bladder, poor flow and incomplete voiding. Disorders producing predominantly autonomic failure include diabetes mellitus, multisystem atrophy, pandysautonomia, paraneoplastic neuropathy and Riley-Day syndrome. Damage to pelvic and pudendal nerves due to prolonged labour, extensive pelvic surgeries, trauma, intrapelvic tumours and chronic constipation can lead to weakness of the pelvic floor muscles. These patients present with stress incontinence, double incontinence and genital prolapse [8].

Myasthenia gravis , a disorder of Neuromuscular transmission ,can produce detrusor weakness. Anticholinergic medications given in its treatment can lead to excessive detrusor contractions[9]. Smooth muscle dysfunction can also lead to poor detrusor contractions. Smooth muscle myopathies may be primary, secondary to chronic obstruction or part of a systemic myopathy like myotonic dystrophy. The primary smooth

muscle myopathies with involvement of bladder include hereditary and sporadic hollow visceral myopathies, degenerative leiomyopathy, dense body myopathy, polyglucan body myopathy and lipid inclusion myopathy [10]. Chronic bladder outlet obstruction can cause stretch injury to the muscle. Complex repetitive discharges from striated urethral sphincters can cause urinary retention. An abnormal muscle to muscle ephatic transmission causes continuous spasm of the sphincters. This disorder is reported only in women. An as yet unidentified hormonal imbalance is postulated to be responsible[11]. Hypertrophied bladder neck muscle with dyssynergia can cause incomplete emptying or retention in men[12].

Evaluation

A proper Neuro-urological evaluation is essential for understanding the pathophysiology of the urinary symptoms and planning the management. It is important to exclude the common local causes in these subjects before attributing the symptoms to neurological disease. Initial assessments include clinical examination and frequency -volume charts. These simple and objective techniques provide valuable information that is useful in planning further tests.

Pad testing : This is a simple noninvasive method of assessing the incontinence. In this test patient wears a pad and performs a schedule of activities for one hour. The pad is weighed before and after the test. An increase in the weight of the pad by more than one gram suggests incontinence[13]. The scheduled activities are walking and stair climbing for 30 minutes, standing up from sitting 10 times, coughing vigorously 10 times, running on the spot for one minute, bending to pick a small object from the floor five times and wash hands in running tap water for one minute. The pad test has a high false negative rate and the results may not correlate with severity of the incontinence[14].

Uroflowmetry : This is a physiological, quick, noninvasive screening test. The voided volume, maximum flow rate, acceleration rate, average flow rate, time to maximum voiding and flow time are assessed. The techniques include weight measurement, momentum exchange, carbon dioxide flow and electromagnetic method[15]. The urine flow rate (UFR) varies significantly with age, sex and volume voided. Normal adult male UFR is about 15 ml per second. A low UFR is seen in detrusor sphincter dyssynergia, poor detrusor contractions and mechanical outlet obstruction.

Post voided residual urine : Measurement of post voided residual urine volume is a simple and objective test of the ability of the bladder to empty. The volume of urine left

in the bladder is measured after voluntary voiding. This can be done either by post void catheterisation or with a simple hand held ultrasound scanner. Residual volume of more than 100 ml or 20% of voided volume requires intervention.

Cystometry : Cystometry is the measurement of the pressure - volume relation of the urinary bladder. Fluid or gas is infused into the bladder at a constant rate. Simultaneously intravesical and intraabdominal pressures are measured with transducers kept inside the bladder and rectum. Normal bladder capacity is more than 400 millilitres. Infusion of normal saline at the rate of 100 ml per minute increases the bladder pressure by [15] cm of H2O. An excessive rise in the intravesical pressure for the volume infused suggests low compliance. The volume at which first appreciation of fullness, desire to void starts and detrusor contraction occurs is noted. Excessive uninhibited detrusor contractions of pressure more than [15] cm of H2O during filling phase is suggestive of detrusor hyperreflexia[16]. The Bethenacol supersensitivity test and Bor's ice water test are two commonly used provocative tests during cystometry. Bethenacol, a parasympathomimetic agent induces detrusor contractions in patients with dennervated bladder. Thirty minutes after a subcutaneous injection of 2.5 mg of Bethenacol the cystometry is repeated. The rise in the detrusor pressure to 15 cm of water at 100 ml indicates dennervation supersensitivity. Rapid infusion of 100 ml of normal saline at 00C induces detrusor contractions in patients with upper motor neuron lesions. Test is negative in normals and those with lower motor neuron lesions This is a simple and rapid test that helps in identifying the type of detrusor instability [17].

Videocystourethrography : Videocystourethrography is the fluoroscopic examination of contrast filled lower urinary tract with cystometry. The purpose of this test is to image the bladder and urethra with simultaneous monitoring of intravesical pressure. This test is useful in the diagnosis of detrusor sphincter dyssynergia and its complications like ureteric reflux and urethral diverticula[18].

Ultrasound scanning : This is a simple noninvasive, less expensive alternative to Videocystourethrography. Vesical neck located behind the symphisis pubis is difficult to visualize with transabdominal scanning. Vaginal, transrectal and perineal probes have been used with different degrees of success [16].

Urethral pressure profilometry : Urethral pressure should always exceed intravesical pressure except during voiding. Recording of urethral pressure was first done by Victor Bonney[19]. Currently urethral pressures can be accurately measured with catheter

mounted microtransducers20 The recordings are done with subject in supine position with 250 ml of saline in the bladder. The catheters with dual transducers are introduced into the bladder and withdrawn slowly at 2.5 mm per second. As the transducers passes through the urethra it records the resting urethral pressure. The test is repeated with patient coughing. The recordings are also repeated at the point of maximal urethral pressure for two to three minutes. The urethral pressure profile helps in assessing the outflow obstruction, detrusor sphincter dyssynergia and functioning urethral length. The pressure profile during micturition helps to differentiate between detrusor external sphincter dyssynergia and detrusor internal sphincter dyssynergia.

Urethral electrical conductance : The electrical conductance of urothelium is less than that of urine or saline. Hence amplitude of current passing between two electrodes placed in the urethra will increase when urine or saline leaks into the urethra. Small urine leaks missed with conventional urodynamic studies can be detected with this technique[16].

Ambulatory urodynamics : In conventional urodynamic studies saline is retrogradely infused at a fast rate into the bladder in a controlled environment. Hence the study may not reflect the changes taking place in the bladder during routine daily life. Ambulatory urodynamic studies enable natural bladder filling to occur. Patients engage in normal daily activities that may precipitate the symptoms. After voiding and defecation, microtip catheters are introduced into the bladder and rectum. Patient is asked to drink 500 ml of water. A record of events like urgency, voiding and incontinence is kept. This test is usually done for twenty four hours and is indicated in symptomatic subjects with negative urodynamic studies, postural incontinence and sleep enuresis[21].

Electromyography of sphincters : Abramson first used electromyography (EMG)in urological evaluation[19]. The sphincter EMG is used for recording the activity of urethral sphincter during filling and voiding phases and to examine the innervation of striated muscles of pelvic floor. In women direct recording from urethral sphincters is possible with surface electrodes. Catheter mounted vaginal electrodes are also used for recording urethral sphincter activity. Direct recording from urethral sphincter is difficult in men. The anal sphincter behaves similarly to urethral sphincter. Hence it may be used for evaluation. The anal sphincter EMG can be recorded with surface electrodes applied to either sides of anal orifice or anal plug electrodes inserted into the anal canal. However, detailed EMG evaluation requires use of needle electrodes. Synchronous EMG and pressure flow studies are useful in evaluating incontinence. The sphincters are tonically active. Normally during detrusor contractions the sphincter EMG activity will cease.

Ongoing sphincter EMG activity while detrusor is contracting is seen in detrusor sphincter dyssynergia. Fowler and Kirby[11] recorded complex repetitive discharges with low jitter from urethral sphincters of women with " psychogenic "urinary retention. The single fibre EMG of pelvic floor muscles is useful in diagnosis of multisystem atrophy and cauda equina lesions[22].

Sacral reflexes : Sacral reflexes are contractions of parts of pelvic floor in response to stimuli applied to perineum, genitalia or the mucosa of lower urinary tract. The sacral reflexes that can be recorded include bulbocavernosus reflex (BCR), pudendoanal reflex, vesicourethral reflex and vesicoanal reflex[23]. The afferent and efferent pathways involved are second to fourth sacral roots. The BCR can be recorded with surface or needle electrodes after stimulating dorsal nerve of penis. This reflex has two components: early response at 25 - 45 milliseconds and late response at 60 - 70 milliseconds. The afferents are small-diameter myelinated fibres and the motor efferent are large diameter myelinated fibres. The early response is an oligo synaptic spinal reflex[24] and late response is a polysynaptic spinal pathway. The motor responses from urethral and anal sphincters can be recorded with bladder stimulation using catheter mounted electrodes. The latencies of these vesicourethral and vesicoanal reflexes are longer than that of BCR (59 ms Sd-8 ms)[25]. The sacral reflexes are prolonged or absent in subjects with cauda equina lesions[23]. Sethi[26] reported that BCR was inhibited during normal voluntary micturition and this voluntary inhibition was lost in subjects with supra sacral spinal cord lesions. The sacral reflexes may be normal in subjects with partial cauda equina lesions and don't test the autonomic nerves[23].

Pudendal evoked potentials : Pudendal evoked potentials are used for evaluating the neural pathways from sacral cord to cerebral cortex[27]. The dorsal nerve of penis is stimulated with ring electrodes. The 200 to 400 responses are averaged from Cz' (2 cm behind Cz) with reference at Fz. The latency of Pudendal evoked potentials (40 ms) is longer than that of the sensory evoked potentials from posterior tibial nerve. This is due to the slower conduction velocity of the spinal pathways of pudendal evoked potentials[28]. This test may be useful in evaluating impotence and voiding dysfunction. The value of this test in routine clinical practice is not yet clear. Cortical evoked potentials can also be recorded after electrical stimulation of urethra and bladder with catheter mounted electrodes[29,30] a small potential of latency 55 milliseconds can be recorded from the midline.

Pudendal motor conduction studies Due to its anatomical position the motor conduction study of pudendal nerve is difficult. Kiff and Swash[31] used perrectal stimulation with a

bipolar electrode fixed to tip a gloved finger of the examiner. The recording electrode was also fixed to the base of the same finger. The nerve was stimulated at the ischial spine and contractions from anal sphincter were recorded. The urethral contractions can be recorded with a catheter mounted recording electrode. This test is useful in identifying pudendal nerve damage particularly in patients with stress incontinence.

Central motor conduction studies : The motor responses of pelvic floor muscles to cortical or cauda equina stimulation can be recorded. The central conduction time can be calculated by subtracting the latency of sacral response from that of cortical response[32]. Potentials of these tests are still not clear.

Thermal threshold testing : Unmyelinated afferent fibres that carry temperature sensation are also involved in the transmission of autonomic impulses. Thus, measurement of thermal threshold of genitalia is an indirect method of assessing autonomic function. This technique was used for evaluation of patients with diabetic neuropathy. The only draw back is that it is a subjective test[33, 34].

Sympathetic skin response : Sympathetic skin response (SSR) is a simple electrophysiological test of sympathetic sudomotor function. Electrical, biological and noxious stimuli can be used to evoke these responses[35]. The SSR from perineum may be useful in the evaluation of impotence[36].

Classification of Neuro-urological disorders

There are several classification systems for voiding dysfunction. These systems are based on the type and location of the neurological lesion, type and location of the lower urinary tract pathology effect of the neurological lesion on the urinary tract, urodynamic observations, and clinical signs and symptoms. Lapides classified the neuro-urological disorders based on clinical and cystometric findings. The neurogenic bladder was classified into sensory neurogenic bladder, motor paralytic bladder, autonomous neurogenic bladder, uninhibited neurogenic bladder and reflex neurogenic bladder 5. Patients with sensory neurogenic bladder fail to appreciate bladder sensations. This is due to interruption of sensory fibres from the bladder to the spinal cord or afferent tracts to brain. Motor paralytic bladder is a large capacity bladder with painful retention. Cystometrogram shows absence of bladder contractions. This is due to destruction of the sacral motor neurons or motor nerves to bladder. In autonomic neurogenic bladder there is complete separation of bladder from the sacral spinal cord. The patient has no

bladder sensations or contractions. There is painless urinary retention with overflow incontinence. Disruption of cortical regulation of PMC results in uninhibited neurogenic bladder. This causes involuntary bladder contractions with co ordinated relaxation of the sphincters. These patients suffer from incontinence without residual urine. The reflex neurogenic bladder is due to disruption of the pathways between the sacral centres and brain stem centres. Bladder sensations may be preserved. There are involuntary detrusor contractions without coordinated relaxation of the sphincters. This results in incontinence with significant residual urine.

Krane and Siroky[5] devised a scheme to describe the urodynamic observations from combined cystomery and sphincter EMG studies (Table 1). This is the first classification system based on the functions of the detrusor and the sphincters. It provides an objective terminology for describing neurourological findings. The Krane and Siroky classification describes the functional effect of neurological lesion on bladder and sphincter function. Thus, it helps in selecting an appropriate therapeutic plan. This system requires results of urodynamic study. It is difficult to apply this system to non-neurogenic bladder problems.

Table 1 Krane and Siroky Classification of Neurogenic voiding dysfunction [5]

Detrusor hyperreflexia /Normoreflexia
Coordinated sphincters
Striated sphincter dyssynergia
smooth muscle sphincter dyssynergia
Non-relaxing smooth muscle sphincter
Detrusor areflexia
Coordinated sphincters
Non-relaxing striated sphincter
Dennervated striated sphincter
Non-relaxing smooth muscle sphincter

Management

Proper management of urinary symptoms are essential for functional recovery and rehabilitation of neurologically disabled clients. The mode of treatment depends on site of the lesion and pathophysiology of bladder dysfunction (Table 2). The nature of bladder symptoms may change with the course of the primary neurological problem. The nature and severity vary from patient to patient, from time to time. Hence the urological diagnosis and treatment need to be revised periodically. Associated cognitive and behavioural problems, sensory loss, motor disabilities spasticity and drugs may affect the urinary functions. The neuro-urological evaluation and management is a complex and dynamic process.

Urinary incontinence

Incontinence is the involuntary leakage of urine which is a social or hygienic problem and is objectively demonstrable[37]. This can be due to cognitive and behavioural problem, defective communication, uninhibited detrusor contractions, and defective sphincter closure. Incontinence secondary to cognitive and behavioural disturbances can be managed with scheduled voiding, bladder drill and behavioural therapy. Bladder drill is a regime of timed voiding during which the subject is taken to toilet or given a commode. Patient is given positive reinforcement if he is dry for a fixed period, uses commode without prompting or empties the bladder with reminders. The rewards are withheld if soiled. External collecting devices like condom can be used for patients who do not respond. Patients with defective communication can be helped with timed scheduled voiding, timed reminders and communication aids.

Detrusor instability

International continence society has defined detrusor instability as a condition in which the detrusor is objectively shown to contract either spontaneously or on provocation during bladder filling while subject is attempting to inhibit micturition[37]. This is seen in patients with lesions above sacral spinal segments. The symptoms include urgency, frequency, urge incontinence and nocturnal enuresis. The diagnosis is based on demonstration of uninhibited detrusor contractions during Cystometry. Mild detrusor instability may be managed with fluid regulation, avoidance of alcohol and caffeine and behavioural therapy. Further options are pharmacotherapy, maximal electrical stimulation, acupuncture, intravesical capsaicin and surgery.

Table 2 Management of neurogenic voiding dysfunction

Site of the lesion	Type of dysfunction	Management
Brain	Communication problems	Timed voiding Communication aids
	Lack of concern	Timed voiding Behavioural therapy
	Loss of voluntary control	Timed voiding Reflex emptying
Spinal cord	Detrusor Hyperreflexia	Drugs: Anticholinergic agents, Musclerelaxants, Calcium channel blockers. Electrical stimulation Bio feedback Acupuncture Capsaicin Surgery
	Non relaxing smooth muscle sphincter	Alpha adrenergic blockers: Prazosin, Alfuzosin CIC
	Non relaxing striated sphincter	Banzodiazepines, Botulinum toxin, External sphincterotomy, CIC.
Autonomic nervous system Cauda equina Peripheral nerves	Detrusor areflexia	Drugs Bethanecol, Carbachol Crede'smaneuver CIC
	Incompetence of sphincters	Kegel's exercise Drugs Alpha agonists: Ephedrin, and Psuedoephedrin Estrogen Local injection with Teflon Artificial sphincters Surgery Incontinence appliances
Smooth muscle	Poor Detrusor contractions	Crede's manoeuvre CIC

CIC-Clean intermittent catheterisation.

Pharmacotherapy : The hyperactivity of detrusor can be managed with anticholinergic drugs, smooth muscle relaxants, calcium channel blockers, prostaglandin inhibitors, potassium channel openers and beta adrenergic agonist. Anticholinergic drugs block bladder contractions induced by parasympathetic neurons. They do not increase the outlet resistance. A significant proportion of the bladder contractions are mediated by non cholinergic and non adrenergic neurotransmitters. Hence the anticholinergic drugs cause only partial inhibition of bladder contractions[38]. Commonly used anticholinergic drugs include propantheline, glycopyrolate, atropine, isopropamide and hyoscine. All of them block muscarinic receptors. Side effects of these drugs include dryness of mouth, blurring of vision, tachycardia, drowsiness and constipation. Musculotropic smooth muscle relaxants are the drugs that act directly on smooth muscles and cause relaxation. Oxybutinin is a smooth muscle relaxant with additional anticholinergic activity. The oral dose is 2.5 to 5 mg every 8th hourly. Intravesical instillation of Oxybutinin is also useful in the treatment of detrusor hyperreflexia. Flavoxate is a smooth muscle relaxant with local analgesic action. It reduces detrusor instability at the doses of 100 to 200mg 8th hourly. Calcium channel blockers also inhibit smooth muscle contractions. Terodiline a calcium channel blocker with anticholinergic activity was widely used previously in the treatment of detrusor hyperreflexia. This drug was withdrawn from market following repots of adverse cardiac side effects. Bodner etal [39] reported that verapamil augments the muscle relaxant properties of oxybutinin.

Tricyclic antidepressants (TCA), especially imipramine is useful in the treatment of detrusor instability. It also increases the outlet resistance. The TCAs have a central as well as peripheral anticholinergic activity. They also block the reuptake of noradenaline and serotonin. The imipramine has a direct smooth muscle relaxant effect on the detrusor. Doxepine, a TCA with muscle relaxant properties was also used to treat detrusor instability[38]. The side effects of TCA are mainly due to the systemic anticholinergic activity. Skin rashes, hepatic dysfunctions, obstructive jaundice and agranulocytosis have been reported rarely. They can also produce fatigue, tremors, parkinsonian features, psychosis, postural hypotension and sedation. All TCA have a direct myocardial depressant action. Abrupt discontinuation can cause abdominal distension, nausea, vomiting, headache, lethargy and irritability. Hence all TCAs should be tapered and stopped. They are contraindicated in patients already on monoamine oxidase inhibitors[38].

Other drugs tried in the treatment of detrusor instability include potassium channel openers, prostaglandin inhibitors and antidiuretic hormone. Potassium efflux causes hyperpolarisation of the smooth muscle membranes and prevents the contractions.

Prostaglandins mediate part of noncholinergic nonadrenergic contraction. Indomethacin, 50 to 200 mg may reduce the bladder hyperactivity[40]. 1-desaminocystine 8-D-arginine vasopressin (DDAVP) has been used for symptomatic relief of nocturnal enuresis. The drug administered as an intranasal spray suppress the urine production for 7 to 10 hours[38].

Electrical stimulation : Electrical stimulation may activate various inhibitory reflexes and facilitate continence. The sites stimulated include rectum, vagina, pelvic floor, common peroneal nerve and posterior tibial nerve. Several studies have demonstrated detrusor inhibition after repetitive electrical stimulation at these sites. Even persistent remissions of symptoms have been reported. Draw backs include unpleasant sensation during stimulation, poor acceptability, mucosal irritation, lack of standard equipment and lack of controlled studies[41].

Biofeedback : The detrusor contractions are converted into auditory and visual signals. The client is made aware of detrusor contractions through these signals and taught to control them. This method is very time-consuming and relapse rates are high[42].

Acupuncture : This traditional Chinese method of treatment has been tried in reducing the detrusor hyperactivity. The stimulation of acupuncture sites results in release of endogenous opiods. These opiods inhibit detrusor contractions. This method has been found to be useful in attaining bladder control following Spinal cord injuries[43].

Intravesical Capsaicin: Intravesical instillation of capsaicin has been tried in the treatment of detrusor instability. They act on C fibres containing substance P and inactivate them. As these C fibres are not involved in normal micturition reflex, normal detrusor contractility will be maintained. Wairt etal[44] recently confirmed the efficacy of capsaicin instillation in a double blind controlled trial.

Surgical interventions: Only affective surgical intervention for detrusor instability is augmentation cystoplasty. Bladder is bisected in the coronal plane. A segment of terminal ileum is used to increase the capacity. The surgical option should be considered only after exhausting all attempts at conservative management[45].

Incompetence of the urethral sphincters.

Genuine stress incontinence (GSI)is the most common cause of urinary incontinence women. The GSI is the urinary leakage on raising intraabdominal pressure in the absence

of detrusor contraction. Diagnosis is made with history, clinical examination and videocystourethrography. The GSI can be classified into two groups: Hyper mobility of the urethra and intrinsic sphincter deficiency. The single Fibre EMG of pubococcygeus muscle had showed significant denervation of pelvic floor muscle[46]. Treatments of GSI include pelvic floor exercises, vaginal cones, electrical stimulation, drugs and surgery. Vaginal cones are a cheap, effective and acceptable method of treating GSI. Electrostimulation of pelvic floor also helps in the treatment. The drugs used in the patients with GSI include alpha adrenergic agonist and estrogens.

Alpha adrenergic agonist: Bladder neck and proximal urethra have alpha adreno receptors which on stimulation cause smooth muscle contraction. Various alpha adrenergic stimulants increase the maximum urethral pressure and urethral closure pressure. Side effects include hyper tension, anxiety, insomnia, headache, tremors, weakness, palpitation, cardiac dysrrhythmias and respiratory difficulties. Ephedrine 25 - 50 mg 6th hourly release noradrenaline and directly stimulate the urethra. Psuedoephedrine and phenylpropanolamine also have similar action. Beta adrenergic antagonist potentiate alpha receptors and increase urethral pressure. Side effects include cardiac failure and asthma[38].

Estrogens : The urethra and trigone are embryonically related to uterus. Estrogenic hormones have significant effect on lower urinary tract. They increase the sensitivity of urethra to alpha adrenergic stimulation. Estrogens are useful in the treatment of post menopausal stress incontinence. They have an addictive effect when combined with alpha adrenergic stimulants. The proposed mechanism of action of estrogens include changes in autonomic innervation, increase in alpha receptor content, increase in metabolism of smooth muscle, changes in estrogen binding sites, effect on non muscular elements of urethral wall and better mucosal seal mechanism[38].

Injectable agents : Various substances like Teflon and glutaraldehyde cross linked with bovine collagen can be injected around bladder neck to produce an artificial increase in outlet resistance[45].

Artificial sphincter : Artificial sphincter is a fluid filled cuff placed around the bladder neck. The cuff is connected to a deflation device. On activating this device, the fluid from the cuff is pumped into a reservoir. This reduces the out let pressure and facilitates micturition. After two - three minutes fluid gets automatically pumped back in the cuff. This device is expensive and is associated with various complications like infection and

erosion into the urethra. This technique should be reserved for those failed to respond to all other interventions[42].

Surgical treatment : Surgery aims to elevate the bladder neck and proximal urethra into an intra abdominal position. The various operations described include anterior colporrhaphy, endoscopic bladder neck suspension, Burch's colpo suspension and urethral sling procedure. Hypermobility of urethra can be corrected by bladder neck suspension. Patients with intractable incontinence will need diversion, procedures like uretrosigmoidostomy, urethral closure with permanent suprapubic catheterisation, ileal conduit and continent urinary diversion procedures[45].

Incontinence appliances: Despite best treatment several patients remain in continent. They require incontinence appliances like pants, pads and collecting devices. The need for these appliances depends on severity of incontinence, mobility and manual dexterity of the patient, care giver and life style and sexual activity of the client[47]. The incontinence pants should be well fitting, not too tight and should not damage the skin. Absorbent body-worn, pads are available in different sizes and shapes. Super absorbers are polymers capable of absorbing 50 times their own weight of urine under some degree of pressure. Absorbent rolls are useful only for patients with light incontinence. Pads with protective plastic backing and can be worn with normal underwear are available. They also have self adhesive straps to hold in place. Pouch pads are designed for use with marsupial pants. Their size depends on the pant pouch and the degree of incontinence. Re-usable washable polyester pads have a water proof backing. They can be worn with normal undergarments. All-in-one pants can be worn without additional pants. They have easy side fastenings that can be worn by disabled clients. Bed protectors are water proof sheets that can be kept below the incontinent patients and can be changed easily. Badly creased or placed bed protector can result in pressure sores.

Penile sheaths and condom catheter that can be secured around the shaft of the penis are preferable to an indwelling catheter. They are non-invasive, do not carry risk of infection and safe. The collecting bags can be fastened around legs of mobile patients and to bed. Urine collection devices are not suitable for female anatomy. Currently female urinary pouches that can be attached to vulva and drain into a collection bag are available. But leaking of the seal and skin irritation are significant problems.

Urinary retention

Urinary retention can occur due to poor contractions of detrusor muscle or failure of the sphincters to relax. The goals of management of urinary retention are prevention of renal failure, avoidance of infection, continence, independence and Intravesical pressure of less than 20 cm of water. In Crede's manoeuvre gentle pressure is applied over abdominal wall and urine is expressed out of the bladder. This procedure is useful especially in patients with atonic bladder and dennervated sphincters. It is not useful in patients with abnormal sphincter EMG, as the increase intraabdominal pressure may result in excessive firing. This may cause increase in intravesical pressure, stretching and damage to bladder wall.

Several patients with detrusor contractions and coordinated sphincters can be trained to use reflex bladder contractions for voiding. The micturition reflex can be elicited by washing hands in running water, pulling supra pubic hair and stroking the inner aspects of the thighs. Detrusor, sphincter dyssynergia interferes with effective reflex voiding. The reflex may occur spontaneously resulting in incontinence. A urodynamic study is essential before attempting reflex voiding. It is important to monitor upper urinary tract regularly in these patients since high intravesical pressures can damage the kidneys.

Pharmacotherapy : Underactivity of detrusor muscle can lead to urinary retention, over distension and over flow incontinence. Drugs used in the treatment of urinary retention include agents to increase detrusor contractions, relax the urethral sphincters. Cholinergic drugs like bethanechol and carbachol may be useful to elicit detrusor contractions. Anticholineesterase distigmine bromide was also used for this purpose. The contractions of the internal urethral sphincters are produced by alpha adreno receptors in the bladder neck. Alpha 1 adrenergic blocking agents relax the smooth muscles and decrease the outlet resistance. Alpha blockers used for this purpose include phentolamine, phenoxbybenzamine, prazosin, terazosin, alfuzosin, nicergoline, thymoxamine [48,49,59]. The non-relaxing, striated external sphincter can also lead to retention. Anti spasticity medications like diazepam and baclofen are used to reduce the muscle tone. Injection of botulinum toxin is a new option available for these patients. It produces a non surgical reversible relaxation of the external sphincter. It is suited for patients who cannot perform intermittent catheterisation, and can wear external collecting devices [51].

Clean intermittent catheterization: This was popularised by Sir Ludwig Guttman during world war II[52]. It is a safe, effective and preferred alternative to long term

indwelling catheterisation. It is the best option for patients with hyporeflexic detrusor and urinary retention. In subjects with detrusor hyperrflexia with sphincter dyssynergia, additional anticholinergic medications may be needed to reduce the detrusor contractions. The patients should be taught to maintain the bladder volume around 300ml. Initially sterile techniques were advised for intermittent catheterisation. Subsequent studies have shown that clean intermittent technique is as safe as the sterile techniques for home setting. In hospital setting the role of sterile catheterisation compared to clean catheterisation is still controversial[53]. Upper urinary tract function is well maintained in these patients[54]. However the procedure is more labour intensive and time consuming than continuous indwelling catheter.

Continuous catheterisation : This was the only solution for urinary retention in the past. The indwelling catheter is always associated with bacteriuria. The complications of the continuous catheter include cystitis, urethritis, prostatitis, abscess, fistulae, strictures, epididymoorchitis and erosion of the urethra. Reflex bladder contractions can lead to significant pericatheter leak and intractable incontinence. This procedure is best reserved for dealing with complications in the short term. The aim should be to remove the catheter at the earliest. In patients with indwelling catheter good catheter care including regular changing of the catheter and collecting devices is essential for prevention of complications. Regular irrigation may reduce the accumulation of debris and blocking of catheters[55].

Suprapubic catheterization: This procedure avoids some problems with long term urethral catheter. It is of value in patients with severely damaged urethra secondary to infection or instrumentation or in women with dilated urethra. In some patients urethra needs to be surgically closed to regain continence.

External sphincterotomy: This procedure reduces the resistance of the external urethral sphincter. It is recommended for patients with intractable detrusor sphincter dyssynergia. It also protects upper urinary tract from increased pressure. But it produces incontinence requiring an external collecting device. About a third of the patients also complains of impotence.

Sacral anterior root stimulation: . Electrical stimulation of sacral anterior roots is an effective method of treating patients with supra sacral spinal cord injury. This is usually combined with division of the posterior sacral roots. This sacral deafferentation produces a large, areflexic bladder capable of storing large volumes of urine at a low pressure.

Thus it protects the upper tracts, prevents incontinence and abolishes autonomic dysreflexia triggered by bladder. The electrical stimulation of sacral anterior roots produces contraction of detrusor and urethral sphincter. The problem is due to simultaneous sphincter contraction can be avoided by giving stimulation and brief pulses. Detrusor smooth muscle take a long time to relax. During the interval between two pulses the sphincters relax but detrusor contractions will be maintained. Thus bladder is capable of emptying in bursts. Urinary infection and incontinence are significantly reduced in subjects using this device. There is no need for continuously carrying a collecting device. The upper urinary tract damage is also rare. No long term side effects have been reported[56].

Summary

The aims of management of patients suffering from neurogenic bladder are facilitation of adequate storage of urine, prevention of incontinence and over distension and protection of kidneys and urinary tract from complications. The knowledge of neuro-physiology of continence has increased tremendously during recent years. Rapid advances have been made in the investigations and treatment of neuro-urological disorders. Now it is possible to elucidate the pathophysiology of voiding dysfunction in most of patients with neurogenic bladder. This has made possible proper management of these problems. Currently care of these patients tended to be fragmented between neurologists, urologist and gynaecologists. An integrated multi-disciplinary approach is required for the practical management of this common problem.

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Pain relief in Neurorehabilitation

U. Nandakumaran Nair

Introduction

Among the vital concerns of modern medicine today is its tryst with pain experience. Physicians interested in pain relief would at once confess how frustrating it is to manage a chronic pain patient, and how challenging it is to be in a close encounter with pain sickness.

Definition

Pain is considered to be a psychic orientation towards stimuli that endangers its integrity or causes damage to an organism. Seen this way, pain is an important acquisition to animal evolution. Painful sensations often correlate very well to pathological processes and could forerun external signs of diseases. As an emotional experience pain is depressing and distressing serving as stimulus for various defence behaviour strategies. Pain sensations are also subject to higher mental functions related to cortical activity and are influenced by orientations, beliefs, and culture. Thus pain is entirely a conceptualized subjective phenomenon where memories of experience and experiences themselves do play a part. The nearest definition has come from the Taxonomy Committee of the International Association for the Study of Pain (IASP)[1]. Accordingly pain is "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage." Further understanding is through a five-axis taxonomy that can describe any patient's pain syndrome. These contributions no doubt help in a large way clinical and epidemiological studies and research.

Disability

Two factors can confound a pain situation: the potentials to become intractable and chronic. In either event, disability ensues, and can lead to handicap. Should these occur, the patient might suffer disabilities and handicap, similar to one affected by stroke or cord trauma. For optimal functional restoration, any policy on management makes rehabilitation not only desirable, but essential as well. In the context of health experience, a disability is any lack or restriction (resulting from impairment) of ability to perform an

activity in the manner or within the range considered normal for a human being [2]. Targeting disability is one effective means of ensuring better pain relief and hence rehabilitation. Vasudevan suggests the following scheme for disability evaluation in these patients[3]:

1. causation of injury and relationship of injury to impairment
2. impairment - anatomical, physiological and psychological
3. functional limitation imposed by impairment
4. relationship of functional limitation to work and recreational activity
5. suggestions for future treatment and rehabilitation, and
6. permanency of impairment and statement regarding whether impairment is expected to last twelve months or more

This understanding assumes significance in neurorehabilitation as more than one unrelated impairment can contribute to the occurrence of the chronic pain state. Many physicians spend time searching for the elusive tissue specific pathology as a cause for their patient's condition, indifferent to the foundations laid by IASP and to our knowledge about the impairment - pain disability continuum.

Fortunately the realization that a physical illness need not be the sole perpetrating factor in pain is getting currency. Many physicians now accept the importance of physical, behavioural and subjective testing as well as monitoring to be the best possible form of evaluation. Reading strongly recommends assessment of pain as a prerequisite to pain management strategies[4]. Numerous measuring scales are now available for use at the office level; a discussion of those methods is beyond the scope of this chapter. However mention has to be made of the Minnesota Multiphasic Personality Inventory which still offers an excellent method for a proper pain evaluation[5]. It carries 566 questions with true or false responses and is extremely useful as a screening tool for identifying depression, hypochondriasis, or a hysterical personality. However, it is not useful in making an opinion on the extent of organic involvement in an individual's pain syndrome nor is it sensitive enough to be put to prognostic and predictive use. In little children, scoring systems based on observation of factors like expression, posture, vocalisation do exist as in Infant Pain Behaviour Rating Scale (IPBRS), and in older children, Procedural Behaviour Rating Scale - revised (PBRS - r) and Children's Hospital of Eastern Ontario Pain Scale (CHEOPS). Probably, the Visual Analogue Scale (VSA) is simple as well as effective[6].

Pain in Peripheral Nervous System

Although theoretically motor, sensory, or autonomic features can occur with any peripheral neuropathy, pain, as a sensory symptom is not its common association. When it is present, along with specific treatment directed to the underlying pathology pain control measures will be quite appropriate. Most patients with painful neuropathy have distal sensory abnormalities that can be attributed to small myelinated and unmyelinated fibres. These fibres have increased impulse generation. Reportedly there is myelinated fibre reduction to a substantial degree in painful diabetic neuropathy and the relatively normal content of unmyelinated fibres is thought to represent recovery from previous injury. This presumed state of hyperexcitability induces axonal membrane changes related to ionic conduction and is seen in toxic, metabolic or compressive states. Substance P transit is blocked around sites of peripheral compression leading to consequential segmental vasoconstriction. Among the causes of painful peripheral neuropathy diabetes heads the list.

Neuromuscular involvement can occur during the course of HIV disease; among persons with AIDS (PWA), such an incidence is about 15%. Various presentations are possible and six identified subtypes are the following: (i) acute Guillain - Barre syndrome (ii) distal axonopathy (iii) progressive inflammatory polyradiculopathy (iv) chronic inflammatory demyelinating polyneuropathy (v) mononeuritis multiplex and (vi) ganglioneuronitis[7,8,9]. Symptoms range from paraesthesia and tingling to dysesthesias, lancinating pains, myalgia and cramps. Other sensory and autonomic disturbances do exist along with these syndromes but from the point of view of pain the important clinical situation is the distal axonopathy presumably owing to toxic, nutritional or metabolic reasons, characterized by distal axonal degeneration of primary sensory neurons of the lumbosacral dorsal root ganglia. The condition is very painful.

Among the less common varieties of painful neuropathies are those associated with abnormal proteins. They have several systemic symptoms as their concomitants and can be diagnosed by the presence of specific abnormalities in serum or urinary protein. cryoglobulinaemia, macroglobulinaemia, multiple myeloma, and amyloidosis are examples. Neoplasms also produce pain and other sensory disturbances. A recent report on the stiff leg syndrome suggests a possible neurogenic involvement[10].

Apart from the specific treatment directed to the illness, adjuvant management for controlling pain will draw favourable response. Since any of these conditions can produce

a chronic pain state with its attendant disablement efforts at pain management is always welcome.

In the coming years the Indian physician interested in neurorehabilitation is likely to come across persons with post polio residual paresis who complain of various renewed and late onset disabilities. Since India has launched polio eradication strategies [11] one would see less and less of poliomyelitis, as more states enter the mopping up phase. Singularly enough, one would be confronted with its late complications, a condition popularly known as post polio syndrome. Though its pathophysiological features are not settled at this time single fibre electromyographic results are indicative of a progressive neuromuscular transmission dysfunction [12,13,14]. About 70% of patients report pain as a major distressing feature, manifesting as either arthralgia or myalgia in partially affected regions, in the shoulders of a crutch ambulator, or a normal contralateral knee. Any pain causing functional problems in a residual polio patient accompanied by fatigue should alert one to that possibility [15]. It also appears that the post polio patient is at a higher risk of compressive neuropathy [16]. Once properly diagnosed the late effects have a benign course and are generally amenable to judiciously planned rest, exercises, orthotic correction, and lifestyle modifications alongside transcutaneous electrical nerve stimulation (TENS), heat, and symptomatic analgesics. The proposed therapy with substances acting on dopaminergic receptors like bromocriptine needs further validation.

Central Pain

Pain can follow central nervous system lesions, although rarely. Lesions in the spinal cord account for most of the CNS pain. Apart from cases where definitive surgical lesions are the source of pain, results of treatment are mostly frustrating. Among the CNS pains arising from the brain, thalamic pain is the prototype. Almost any thalamic lesion can produce pain though infarctions are believed to be the major cause. It is now known that lesions at sites other than thalamus also result in thalamic like pain. Anticonvulsants, barbiturates, and tricyclic antidepressants singly or in combination give good results. But treatment on patients in whom untoward effects precede therapeutically beneficial result remains unsatisfactory.

Reflex Sympathetic Dystrophy

The general opinion that reflex sympathetic dystrophy (RSD) is eminently curable or is satisfactorily manageable remains too optimistic. RSD is a symptom complex that has

led to difficulties in definition and classification, and more importantly, different clinicians follow different inclusion criteria. Payne considers causalgia to be different from RSD in its clinical profile [17]. The taxonomy committee of the IASP has proposed change of nomenclature of RSD and causalgia to Complex Regional Pain Syndromes I and II respectively. Causalgia results from trauma to the proximal region of a nerve trunk due to missile or cut injuries and bears no quantifiable relation with surrounding soft tissue damage. Causalgic response commences as early as several hours after the trauma producing most spectacular variations in pain profile ranging from a persistent moderate or severe pain to sudden bursts of acute, intense, and deep pain. Roberts [18] in his brilliant concept paper recounts the sequence of events leading to RSD and causalgia. Accordingly the spontaneous, high and unchecked activity levels in the multi-receptive neurons produce pain sensations which are anyhow sensitized by various nociceptor responses. While in causalgia, signs and symptoms fall mostly under the category Sympathetically Maintained Pain (SMP), in RSD at least some of the symptom complex are Sympathetically Independent Pain (SIP). Three stages can be often made out in RSD designated as the inflammatory, dystrophic and atrophic stages with distinct prognostic tags attached to each. Pain, tenderness, temperature changes and swelling of an extremity point to RSD, and presence of intolerance to sudden temperature changes favours the diagnosis. At other times diagnosis may not be easy, when some of these features are absent and hence a diagnostic criteria is sought [17].

From the treatment point of view, it is worthwhile making a distinction between causalgia and RSD, due to a possible frustrating outcome with the former. Sympathectomy has been a choice procedure for causalgia almost by instinct, but the results are so varied that some workers find it hopeless and others the contrary. The lack of strict criteria separating RSD from causalgia might have contributed to some of the successes. However, a case is made out for a contralateral sympathectomy as well especially when an ipsilateral procedure had failed. Phenoxybenzamine, which blocks alpha - 1 receptors at postsynaptic and alpha - 2 at presynaptic levels gave good results in one study [19]. It is also reported that clonazepam offers beneficial results, but the drug tends to taper off its efficacy in a couple of months and revision of dosage becomes necessary. RSD on the other hand responds much better to sympathectomy. Many of its symptoms like dysaesthesias, touch and temperature intolerance; vasomotor changes get controlled to varying degrees. Calcium channel blockers, corticosteroids, and Bier block have their role. Amitryptaline, which modulates serotonin activity at nerve terminals, has useful effects. Exercises, heat and splints maintain the limb in good function, and significant pain reduction can occur with TENS, and tender spot infiltration.

Phantom Pain

Among the pain caused by deafferentation, phantom pain and postherpetic neuralgia are the most important. Phantom pain can be of any quality intensity and duration. It appears abruptly and passes off after a while; at other times it is persistent and disturbs the individual all through the day. Why it occurs in some and not in others, why it is not seen in the congenitally deficient limb or in little children are matters of speculation. The central theories stress on the effects of tissue destruction on the higher centres. Melzack and Loeser propose a pattern generating mechanism for persistent deafferentation pain[20]. With the reticular activating system's (RAS) declining inhibitory control, the somatosensory projection areas develop self sustaining neural activity at multiple levels. Other theories focus on thalamic, subthalamic or even cortical involvement. Peripheral theories concentrate more on the spontaneous activity going on at the site of severance of the peripheral nerves, on the reverberatory circuits that are built up and on the possible misinterpretation by the brain on the origin of these inputs. There are other postulations explaining the syndrome on a psychological basis.

It is felt that a good preoperative preparation of the patient and subsequent management of his responses to loss of limb can help control phantom pain. Education of the patient on the impact of his loss and on the possible coping up strategies including prosthetic fitment will be helpful. Surgery for excision of neuroma or a revision is not likely to be of help. Regular analgesics might help, if NSAID is not useful, narcotic analgesic should not be with held. Quite often patients do respond to alternative drugs like

carbamazepine, tricyclic anti depressants, propranalol, anticonvulsants, and even baclofen. Other modalities include TENS, local anaesthetics, nerve blocks and combination of deep brain stimulation and TENS. Acupuncture might be useful. These measures have been successful in half to two thirds of cases. Judiciously prescribed physical therapy is helpful, to which ultrasound therapy, massage, heat and ice application can be safely added to produce optimum results.

Muscle Pains

Though existence of tender nodules in skeletal muscles was known to produce pain, it was with the publication of the 'Trigger Point Manual' by Travell and Simons in 1983, that the subject attracted the attention it richly deserved. The 'Manual' continues even today as the best guide to the discerning clinician interested in pain medicine. It recognizes

the fact that muscle can be the seat of intense, disabling pain lasting over a long period. This rather elusive type of pain is being called Myofascial Pain Dysfunction Syndrome (MPDS). It affects men, women, and children alike and spares practically no muscle. A tender nodule that has a specific pattern of referred pain characterizes the involved muscle; thus diagnosis is suggested by the regional distribution of pain. There are other clinical features like, restriction of range of motion and local twitch response. Sensitization of local innervating fibres of the group III and IV afferents are implicated in the formation of the tender nodule or band. This band is popularly called a trigger point (TP). Many inquiries have been made into the exact pathology of a TP. The tender muscles of non-articular rheumatism show electronmicroscopic evidence of 'moth eaten' I - bands, suggesting degeneration of actin due to metabolic distress. In certain situations apart from the above finding, ragged red fibres were present as also fat accumulation known by the term 'fat dusting' over the affected muscles [21,22]. These and other findings were interpreted as evidence of impaired perfusion and hypoxia.

The great clinical significance of MPDS stems from the following:
1 It is a treatable condition

2 It mimics other potentially dangerous conditions

3 When multiple, it gives clue to other diagnostic differentials.

A few examples are listed below [23].

1 Trapezius muscle is a fovourite location of the TP. When it occurs in the upperfibres,pain is felt on the posterolateral aspect of neck, and in the temporal region. These may be mistaken as a unilateral headache.

2 Masseer muscle pain refers to corresponding side of face , upper and lower molars and over eyebrows.

3 Infraspinatus muscle pain is refered to shoulder and anterolateral upper arm, forearm and hand.

Gluteus muscles are another common location for TP. The maximus muscle lesion refers pain mainly over the sacrum and inferior aspect of the buttock. The TP in the medius scatters vague pain over the back of thigh with particularly intense pain over sacrum and iliac crest. Referral pattern for the minimus is towards lateral side of thigh and the buttock if the TP is placed anteriorly in the muscle and to the lower limb on the posterior side if the TP is so placed. This mimics sciatica.

Several management policies can be adopted. Stretch and spray technique involves keeping the muscle in a stretched position, while the subject is kept comfortable, and a

steady stream of vapocoolant spray is administered in sweeps in the recommended directions. TP is also amenable to injections of local anaesthetic, steroid, or saline. Holding the TP between fingers, and infiltrating the entire area are important to the success of the procedure. Other techniques like ultrasound therapy, interferential therapy, TENS, diadynamic current, etc may be successful.

While limited TP disease has strong tendency to respond to specific treatment, when the number is large the condition shows no such inclination. Generalised muscle disease is multifactorial in onset and perpetration: these factors are to be removed for optimum results. Literature suggests that many chronic infections, including giardiasis, and amoebiasis can be a source of nagging MPDS. Similarly, any nutritional inadequacy, impaired glucose metabolism, marginal hypothyroidism and certain states of hypervitaminoses can lead to persistence of MPDS.

Closely related to MPDS are two other conditions, viz., fibromyalgia and chronic fatigue syndrome. The tenth edition of I C D has listed fibromyalgia as a separate entity and diagnostic criteria have been drawn up by the American College of Rheumatology in 1990 [24]. A major requirement for diagnosis is the finding of tenderness at specific points of the body and regions. It may be associated with systemic concomitants such as sleep disorder, a point where chronic fatigue syndrome tends to overlap. Though the oft-described spasm of fibromyalgic muscles has not been demonstrated kinesiologically or electromyographically, there is increasing evidence pointing to an organic basis for it. Ig G deposition below epidermis and alteration in collagen have been noticed [25]. Discrepancies in arousal noticed in fibromyalgic persons are thought to cause inefficiency in aerobic activity making affected muscles susceptible to microtrauma. Unfortunately very little can be done to help the victims of these conditions at the present time.

Microtrauma sustained over a period can lead to pain related disability. Classically many conditions are believed to be the consequence of chronic minor repetitive injury eg. carpal tunnel syndrome in typists and pianists. Guyon's type compressive neuropathy in persons manipulating the mouse of a computer, spondylolysis in young adults engaged in physical work or athletics, scapular pain in children having to carry heavy school bags. There are several other less popular types of microtrauma related diseases as multiple repetitions of an ordinary movement, or from having to work in a maladapting workplace. Housewives complaining of right-sided persistent rotator cuff pain in shoulder, scapular pains in administrative personnel using uncomfortable but standard furniture, cervicobrachialgia over the dominant side of dental surgeons, are examples. The author

and PGC Nair had a series of persons employed on a casual basis as prawn peelers who had to squat over long periods of time during their work reporting with diffuse pain and discomfort on the back and sides of legs (unpublished data). Some cases of chronic repetitive trauma can indeed produce features of RSD and sympathetically maintained pain, which does not as a rule respond to simple sympathetic blockade [17]. Its significance is not fully understood. The only way to diagnose these problems is to have a high index of suspicion and to extract a detailed occupational history. Lifestyle modification, workplace adaptation, splints, physical therapy and supportive analgesics or adjuvant medication are of immense help.

Some patients with dysthymia and alexithymia have pain disease [26]. Both ICD 10 and DSM-IV classify dysthymia as mood disorder associated with somatic features but at an intensity that does not attract a diagnosis of hypomania or depressive episode. The somatic preoccupation of a dysthymic patient can be pain, when it is so it is sustained, and has a prolonged course. Incidence is believed to be 2.2% for males and 4.2% for females, but the frequency of pain disorder within the group is not accurately known. The patient with alexithymia has difficulty to describe and express feelings, confuses emotional states with bodily sensations. In the context of pain disorder, these patients have problem in accepting their emotional difficulties and consider organic pathology behind all their symptoms. Surely a multi-pronged management would be essential in these categories of patients.

Thermography

In order that a proper evaluation is made, pain patients ought to receive well- planned and conceived set of investigations. An account of investigative methods and their importance can be had from other sources. One investigation that showed promise two decades ago but lost popularity since then is thermography. Of late it is being increasingly recognized as a useful tool for evaluating autonomic system disorders. With modern infrared telithermography system equipment smaller areas of the body can be studied with better accuracy. No doubt the system demands experience in interpretation, but in radiculopathy [27], RSD, fibromyalgia, MPDS, and low backache, thermography offers good results. It is also the author's experience that thermographic studies correlate very well with per-operative findings of intervertebral disc prolapse (IVDP). The attempts to use it to diagnose pain, however has to be deprecated. It probably has no role in settling disputes of compensation claim within a courtroom. We still have to learn much more about autonomic system to be able to read what the thermography pictures appear to say especially as its specificity is not yet established.

Therapeutic Options

Various treatment options are available to the physician within a neuro-rehabilitation setting. The plethora of techniques proves that no single measure has undisputed track record, this puts a great responsibility on the physician to choose from the different modalities at his disposal.

Spinal epidural injections have been praised and criticized. There are important studies that suggest good results with epidural steroids if patient selection is appropriate. Benzon reports that in IVDP with root involvement, it is therapeutic. It is useful in annulus tear and sometimes in chronic degenerative pain. Other backaches might not respond and it would be a good policy to try out better options. Sympathetic blocks are a popular choice in any neurorehabilitation facility. Their obvious indications are those conditions most likely to cause SMP. Of the several procedures in vogue, cervical sympathetic chain blockade is the prototype, and is indicated in a host of conditions including RSD, peripheral vascular disease, progressive systemic sclerosis, phantom limb pain, and malignancy. Responses to the block vary and there is no accurate predictor to aid patient selection. The procedure is carried out through a posteriorly directed needle a little over an inch above sternoclavicular junction, depositing a solution of phenol or alcohol at the level of the C-6 tubercle. Lumbar sympathetic block has rather ambiguous indications; its analgesic potential is believed to be partial, helping to supplement other therapies. It should be considered in cancer pain and post-herpetic neuralgia. Other sympathetic blocking procedures are less often practiced. When spinal apophysial joints generate pain, facet joint blocks can easily control them. Unfortunately diagnostic difficulties limit their use. Facet syndromes mimic any other backache that is not accompanied by typical radicular symptoms. Blocking the facet joint can be accomplished by injections or radio frequency lesioning under fluoroscopic control.

Electro analgesia has attracted considerable interest. The survival of several electro analgesic modalities despite the controversies about their efficacy is indicative of the confidence they have received from physicians and patients. The most notable among them TENS, whose neurophysiological basis is extensively discussed in literature [28,29,30,31]. New versions of TENS stress on Burst Frequency probably relying on the work by Sjolund and Eriksson [32]. The burst frequency is 2- Hertz; each lasting for 70 ms., and containing 7 pulses per burst, thus giving an internal frequency of 100 Hz. It is believed to release endorphins at the best levels. There are other conventional modes of application of TENS that are being followed. One advantage of TENS is its usefulness

as a patient controlled analgesia measure. Since prescription specifications on frequency, intensity, electrode placement etc, are not standardized, results of therapy do vary. Cold Laser is another technique that deserves mention. Its nonthermal beneficial results are believed to be due to its action on mitochondrial membrane, on superoxide scavenging through improved activity of superoxide transmutase, and probably on increased serotonin level.

Electromyographic Biofeedback (EMGB) offers a chance to the patient to participate in his treatment actively by following contraction and relaxation of his muscle groups and by willing pain reduction strategies. Migraine, low backache, cervicobrachialgia and MPDS are to different degrees amenable to EMGB. Even Relaxation techniques can relieve pain through a similar manner, and when combined they appear to complement each other [33,34]. The Cognitive - Behaviour Therapy (CBT) approach considers modification of coping strategies in the pain patient in order to obviate maladaptations in cognition. Its principles among others include (a) help the patient identify what the problem is, - not, what it is not, (b) acknowledge the existence of symptoms. (c) Give relevant information and (d) Collaborate with the patient and do not convert the sessions into combative ones [35].

Transdermal application of analgesics is becoming very practical - from Eutectic Mixture of Local Anaesthetic (EMLA) to several other potential drug delivery systems. Morphine reportedly can be given transdermally. Fentanyl, which was successful as a postoperative patient controlled analgesic, is available in a transdermal delivery system. It is considered to be the most appropriate drug for this purpose as it carries a molecular weight of less than 1000, and has good hydrophilicity and lipophilicity [36]. Transdermal fentanyl is used in cancer and postoperative pains. In the light of new information much of the old models on the role of the spinal cord in central sensitization are being reviewed. The cord is thought to be a centre having four stimulus processing states where impulses from the periphery can be qualitatively altered to an extent that what is perceived by the brain might be quite different from what happens distally. Munglani, Hunt, and Jones [37] review this 'black box' model of the cord in their article. The authors throw open exciting new possibilities in the present and future pain control therapies. Thus, Substance P and Neurokinin A activation might help and Vasoactive Intestinal Peptide antagonist be useful in treatment. Use of Calcium antagonist's synergism with opioids in the cord, intrathecal NSAID and antisense oligonucleotide to c-fos might be realized in future. There is renewed interest pre-emptive analgesia and its relevance to chronic pain prevention.

A great deal of interest has been generated on the placebo effect. Depending on the conditions under which it is tested placebo incidence can vary between 35% to 85%[38]. It is possible for a patient to experience multiple side effects with placeboes, and an occasional patient to have negative placebo response, the so called nocebo effect. With so many treatment options available for certain conditions, it is in fact not possible to rule out placebo effect anyway. How it works in the individual patient is far from understood. Claims that it is endorphin mediated or that it is dependent on patient's expectation remain unconfirmed. There will be left finally many patients with pain in whom treatment is largely unsuccessful. These persons could be reassured and might be informed that specific therapy might become available later and that until then with concerted action and help from family, attending physicians and other professionals a strategy of behavioural approach to tertiary prevention can be begun. Individuals will be encouraged to lead as normal a life as possible, and work hardening measures will be given their logical significance[39]. This will ensure productivity at whatever level possible, and will lead the person through structured programmes to improve efficiency and quality.

Summary

The above account is a general presentation of the pain diseases bearing a relevance to neurorehabilitation setting. It is not exhaustive, considering that each regional pain situation has separate modes of approach in management. A physician needs great skill and empathy to be able to manage the various dimensions of pain spectrum. Application of active rehabilitation principles acts as positive catalyst to ultimate favourable outcome[40], as demonstrated in the document released in the U.K. by the Clinical Standard Advisory Committee on back pain. For a physician interested in pain medicine, rehabilitation is a guiding principle.

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Possibility of use of Functional Electrical stimulation of extremities in neurorehabilitation

Martin Stefancic

Introduction

The method of applying electrical stimulation for obtaining functional movements, called by Liberson et al.[1] as "Functional Electrotherapy" is now known as "Functional Electrical Stimulation" (FES)[2]. It is a form of low frequency electrotherapy. FES is electrical stimulation of muscles, deprived of nervous control, to evoke contractions producing a functional, useful movement[3]. It is used in the treatment of patients with disorders of motor functions, for control and modifications of motor output and movements. Neuromuscular electrical stimulation can be applied to different organs and parts of the body[5,6]. In this article the experiences of Ljubljana multidisciplinary research and rehabilitation engineering team in the use of FES for external control of movements of paralysed or paretic extremities is described .

Principles

The structure stimulated by FES of extremities are the so-called motor units, or more precisely the axons of peripheral nerves. If we apply a series of electrical impulses of the intensity above the stimulation threshold on the motor nerve at 20-30 Hz frequency, a smooth tetanic contraction of the corresponding muscles occur. In this fashion it is possible to obtain desired movements of various muscles and muscle groups with adequate parameters of electrical impulses and positioning of stimulating electrodes.

FES of extremities is applicable in patients with lesion of upper (central) motor neurons, and not lower (peripheral)motor neurons. With respect to FES, the most important difference between the two types of lesions is that in upper motor neuron lesions the electrical excitability of the lower motor neurons is preserved. while it is reduced or completely absent if the lower motor neurons themselves have suffered a damage[3]. In LMN lesions muscle contraction could only be obtained by much stronger electrical impulses. With few exceptions, no clinically efficient FES systems have so far being developed for patients with lesions of peripheral nerves. Thus, the use of FES is not indicated in lower motor neuron lesions, but only in paralysis resulting from upper i.e.

central motor neuron lesions but not in lower motor neuron lesions. The candidates for FES of extremities are mostly patients with the following clinical pictures:

- patients with cerebral lesions (adult patients with hemiplegia or hemiparesis due to stroke, tumour, injury, inflammation etc cerebral palsy children with hemiparesis, diparesis, monoparesis etc.)
- patients with lesions of the spinal cord (spastic paraplegia or paresis).

Even among these patients with upper motor neuron lesion further selection for FES treatment is often necessary, considering the efficacy of muscle contractions obtained.

Depending on the types of electrodes three kinds of electrical stimulation are possible: transcutaneous stimulation with surface electrodes, percutaneous stimulation with wire electrodes and subcutaneous stimulation with implanted electrodes. Percutaneous stimulation with wire electrodes is rarely used, except in experimental conditions. Subcutaneous stimulation with implanted systems and radio frequency transmission of signals is till now not widely used, mostly because of technical problems. So, in clinical practice, usually transcutaneous FES is performed with surface electrodes (metal plate electrodes coated with several layers of gauze, soaked with tap water or conductive rubber electrodes with conductive jelly). The electrodes are connected by wires to the stimulator with electronics and commands. In Transcutaneous FES mono or biphasic impulses of 0.1 - 0.3 ms duration, frequency 20 - 40 Hz and intensity individually adjusted to get the suitable response of the muscles are used. FES can be performed with single channel (one pair of electrodes), or - multichannel stimulation.(two or more pairs of electrodes).

In selected patients with upper motor neuron lesions (hemiplegia or hemiparesis and paraplegia or paraparesis) we apply FES treatment with the following aims:

- to facilitate voluntary control of paretic muscles
- to perform artificial contraction of completely paralysed muscles
- to restrengthen weak atrophied muscles
- to modify the motor output and movements.

The therapy by FES is usually initiated once the acute phase of the disease or injury has passed and the state of the patients has been stabilized

FES in patients with cerebral lesions

In spastic hemiplegia there is loss of voluntary control of one half of the body. The severity vary from a complete hemiplegic state to a slight hemiparesis. The the patients often observed in rehabilitation centres are those with moderate hemiparesis. In these cases FES treatment can be used to improve function of affected extremities. In hemiplegic patients, unable to stand and to walk, FES can be used in sitting or lying position to facilitate different movements of the upper and lower extremity.

In hemiparetics, the hand opening i.e. unclenching the fist, is often difficult to perform as spasticity prevails in the flexors of the fingers and the wrist. The FES of the finger and wrist extensors with careful positioning of the electrodes over the extensor muscle group on the forearm results in hand opening. This may serve as a functional movement, in the beginning of grasping an object.

The walking pattern of the hemiparetic patients is quite characteristic. The foot is usually in an equinovarus position. While walking, the patient's toes and the outer foot margin rub against the ground. The patient is faced with an imminent danger of sprains and other injuries at the ankle. In the swing phase of the step a deficient knee flexion is compensated by a pronounced circumduction of the lower extremity as a whole. The equinovarus position is usually corrected by passive mechanic braces .The electrical stimulation of the peroneal nerve behind head of the fibula causes contraction of peroneal and anterior tibial muscle groups resulting in active correction of dorsiflexion and eversion of the foot.

One-channel electrical stimulator, facilitating both hand opening and correction of the equinovarus position of the foot are commercially available. While the systems intended for the hand are far from satisfactory and consequently seldom prescribed, the spastic equinovarus correction by means of FES is a fairly efficient and frequently used method. The so called FEPA (Functional Electronic Peroneal Apparatus) is available in several embodiments. It is an orthotic aid which can be prescribed for permanent use. Multichannel systems for surface FES are also developed for hemiparetic patients, but their use is usually limited to the hospital environment for therapeutic training. Nevertheless, it must be noted that in hemiplegic and severe hemiparetic patients FES alone is not sufficient to enable the patients to walk and other techniques for locomotor training must be tried. FES is one of many approaches in the treatment of these patients.

In children suffering from cerebral palsy, the application of FES is based on similar principles as in adults. Considering the type of affliction, stimulation can be performed both in the hand and the leg. A frequent need for a bilateral stimulation of the peroneal nerve is stated not only in children with diparesis but also in those with hemiparesis[8]. A small child, however, can only be coaxed into allowing electrical stimulation through playing. We usually do not use FES in children before third year of age. Electrical stimulation should never be painful or unpleasant - rule to be observed also in the case of adult patients, otherwise, there is no indication to apply it.

FES in patients with spinal cord lesions

In patients with an incomplete lesion of the spinal cord, gait is often characterised by deficient bilateral dorsiflexion of the feet. In such cases, external control of the pretibial muscles with FES of the peroneal nerve, applied uni- or bilaterally is indicated. Sometimes one additional channel of stimulation of ankle plantar flexors can be added for better push-off phase of the foot[9]. If the deficit is corrected by FES, patients are prescribed a suitable stimulator for permanent use.

Complete paraplegics represent a much harder rehabilitation problem. In younger patients with lower lesions, in good psychophysical condition, the goal can be achieved by mechanical braces for the lower extremities and walkers or crutches. Most of the patients, however, are bound to a wheelchair which they also use for moving around. An alternative to such a solution is external movement control by means of FES. Back in 1963, Kantrowitz described the possibility of strengthening completely paralysed muscles in paraplegic patients by electrical stimulation[10]. Adequately strengthened muscles, especially thigh quadricepses, are a prerequisite for using FES to get up and maintain the upright position for a certain period of time. Standing in a paraplegic subject can be achieved by bilateral stimulation of femoral nerve branches to cause contractions of knee extensors. Since the performance of a step implies activation of several muscle groups gait requires multichannel stimulation. A relatively simple and efficient solution has been described by Ljubljana research team for FES in paraplegia. A flexor response of the lower extremity during the swing phase is achieved by a single stimulation channel above the peroneal nerve - a combination of efferent and afferent stimulation. The stance phase of the lower extremity is maintained by means of efferent stimulation of the knee extensors in the same way as at standing[11,12]. A well trained patient can, all by himself, trigger stimulation trains to one or the other lower extremity by switches incorporated into the handles of the crutches or walker. Only rare paraplegics,

mostly younger patients with lesion in the mid thoracic segments and in good general condition succeed to walk with FES at least for short distances. Otherwise, the main kind of locomotion in paraplegic patients is still the use of wheelchairs.

In quadriparetic and quadriplegic patients yet an another problem is restoring the function of affected upper extremities. Complicated movements, particularly those of hands, require programmed multichannel stimulation. This is made all the more difficult in patients with lesions of the cervical spinal cord. The paralysis of their upper extremities is not only of the central but frequently of the lower motor type. Hence it is difficult to obtain tetanic contractions of proper strength. In the current stage of technology, satisfactory functional movements of the hand can exceptionally be obtained in rare quadriplegic patients.

Safety measures

Regarding the use of FES in practice, attention should be paid to the safety measures taken in the choice of the electrodes, the parameters of electric impulses and the technique of applying it onto the patient. Improper application of electric current is a possible electric hazard. The density of electric current with respect of the type, size and position of electrodes must be carefully considered to prevent burns and any other electric danger. Each electromedical device together with its parts and accessories should be always faultless [13].

Low frequency electrical stimulation is not always suitable for every patient with the above mentioned diagnoses. There are also some contraindications, which must be taken into account. In cardiac patients, also in those with cardiac pace-maker and in pregnant women FES must not be used. In patients with myasthenia gravis neuromuscular stimulation can cause exacerbation of the disease. Only the patients in good general condition without any severe concurrent disease are candidates for FES. Electrical stimulation is not applicable in very small children and in persons, whose psychical state prevent them from collaborating in the treatment. Candidates for FES should be also without contracture, severe osteoporosis, any signs of local and/or general inflammations, infections, malignant tumours and other complications. All other safety measures for low frequency electrotherapy must be also taken into consideration.

The proper positioning of stimulating electrodes is also very important to prevent some disturbing side effects of electric current. Incorrect application of FES neglecting some

contraindications considerably increases electrical hazard . For the use of FES it is not sufficient to buy the stimulator and to apply it to the patient. Special educational seminars for application of FES are necessary to therapists. Careful instructions and the training under the control of experienced therapists must be given to the patients, to enable them for the proper use. Specialised service for repairs must also be at disposal for long-term use of electrical stimulators.

Conclusions

We must say, that there are limitations of the use of FES regarding the present technology. We are able to carry out relatively simple movements of extremities for functional purposes with the current equipment . But on the other side, there are also advantages of the treatment with FES, in comparison to classic orthotic appliances commonly used to improve function of paralysed extremities. FES enables active use of patients' own muscles, even if artificially stimulated with some beneficial effects 14. We consider FES is one of various additional methods used to help the patients to achieve better rehabilitation.

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Integrating traumatic brain injury rehabilitation into multidisability community based rehabilitation programmes

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The paper attempts to provide some guidelines to evolve a national policy for the rehabilitation of the people with traumatic brain injury (TBI) in India, within the framework of the existing rehabilitation policies in the country. Since the emphasis of the paper is on rehabilitation, issues related to acute care of TBI are not dealt with. What is attempted is the possible integration of the needs of people with TBI into the ongoing community based rehabilitation (CBR) plans and programmes in the country, keeping in mind the need for realistic planning, effective coverage and affordable costs.

According to one global estimate of the causes of disability[1], trauma and injury account for 15% of people with moderate and severe disability. In the United States, it is reported that 5,00,000 to 1.5 million people suffer head injuries each year, with 50,000-70,000 persons suffering moderate to severe injuries[2]. The peak incidence is between the ages of 15 and 24, and it is 2-3 times more in males than females. In the United Kingdom, the reported prevalence of significant disability is about 0.3%[3]. World-wide, the available epidemiological data relating to TBI seems to be inadequate. In the U.K., noational planning to address neurological rehabilitation began to be addressed as late as 19923. In the U.S., more attention was focused on rehabilitation of TBI form the 1980s. According to the National Head Injury Foundation of U.S., the number of programmes for people with head injury went up from 40 in 1980 tom 700 in 1988[2]. Even so, the Foundation estimation that less than 10% of patients with moderate and severe injuries get adequate rehabilitation services.

In India, statistics relating to the prevalence and incidence of TBI are sketchy, and largely derived from police records and reports in the print media. Efforts towards planning and establishing rehabilitation services for such patients are quite inadequate. Even though the exact magnitude of the problem in the country is not known, it is necessary to recognise that disabilities caused by trauma and injury are likely to increase. In a recent newspaper article, it was reported that the number of accidents in just one highway in Karnataka State rose from 346 in 1991 to 420 in 1993. More alarming, between January to March of 1994, the number sputed to 255[4]. All these are pointers

that the number of people with TBI may be large enough to warrant planning for rehabilitation services for this group in an organised fashion.

The importance of rehabilitation for persons with TBI cannot be overly emphasised, especially since a majority of these individuals are in the younger, more productive age group. Rehabilitation measures aimed at reducing disabilities and handicaps, and restoring of functional abilities to the extent possible would beneffit the individual, and also help to reduce the burdens on the family, on the community and at a larger level, on the nation's scarce resources. In order to achieve the goals of better long-term outcome in rehabilitation, there is increasing recognition of the need for a co-ordinated multi-disciplinary approach in relation to TBI. Such an approach can contribute towards better functioning off the individual, shorter hospitalisation and reduction of secondary complications. All these can produce significant cost savings for health. Besides, with the more recent methods such as cognitive rehabilitation programmes for TBI, the effectiveness of the rehabilitation effort may be markedly improved, as compared to earlier[2]. Considering all these issues, the need for, and the value of, rehabilitation for people with TBI in India comes out clearly as a priority to be addressed.

Following the UN Declarations of 1981 as the International Year of Disabled Persons, and 1983-1992 as the International Decade of Disabled People, many developing countries including India have formulated their policies in rehabilitation. As in other developing countries, rehabilitaion services in India had been mainly institution based and urban centred, with hardly 3% of those in need of services able to have access to them. The trend is now changing, with the promotion of various rehabilitation programmes by the Government and non-Governmental organisations (NGO's). There is now a shift in emphasis towards universal coverage of services and decentralisation of planning to involve local communities. Alongside it has been recognised that many institution based services, which have a longer history, have developed into high cost programmes with problems of mounting staff costs, wastage of manpower and low efficiency of services. As a consequence, the community based rehabilitation (CBR) approach is viewed as a possible cost efficient alternative to rehabilitation institutions[5]. The last decade and a half have witnessed the growth of the CBR approach in India. The principles of coverage, integration and self reliance are common to most CBR programmes in India, but the way they are translated into action would differ from community to community, because of the differing social, economic and cultural factors which can influence CBR programmes. Therefore, there is no universal model of CBR which is applicable across the country. The disabilities which have been accorded

priority for support are loco-motor disabilities, visual disabilities, communication disabilities and mental handicap, with emphasis on childhood disability, early identification and intervention. The approach to rehabilitation is an integrated, multi-sectorial one, which includes interventions in education (special, integrated, non-formal), medical rehabilitation, vocational training and income generation,social rehabilitation and awareness building and prevention.

The involvement of the families and communities in the programmes is emphasised, as also the need for good referral systems and back-up support from existing facilities.

The Government of India, under the Ministry of Welfare, launched the District Rehabilitation Centre (DRC) scheme in 1985, in an effort to provide community based services to disabled people in rural areas [6]. This scheme which was started in 11 districts, is based on the principlesof the primary health care system,and has a structure which is similar and parallel to the district health services. Services are provided at various levels, depending on the size of the target population covered, from the village rehabilitation worker upwards through the primary health centre rehabilitation unit, to the District Rehabilitation Centre at the District headquarters. The Regional Rehabilitation Training Centre (RRTC), which are 4 in number, provide the training inputs, with support from the National Institute. The DRC schemes are supervised by a Central Administrative and Co-Ordinating Unit, based in Delhi. Many NGOs have initiated CBR programmes as well, covering smaller populations and fewer villages, but in tandem with the principles mentioned earlier. The services are programme activities are based in the community and carried out by families and other non-professional staff drawn from the same communities, under the guidance of specialists/professionals. The Government has plans to increase the coverage of the DRCs to many more districts in the coming years, in collaboration with NGOs.

The major advantage of the DRC scheme in planning for rehabilitation of TBI is its location which is administratively and geographically close to the district health services schemd. In such a case, it is relatively easy to shift a patient with TBI from the curative system, i.e., the district hospital, to the rehabilitative system, i.e. the DRC, to continue with the post-acute rehabilitative care. This is possible, provided there are well established referral channels between the two systems. With regard to acute care, it is recognised that the need is great and that present facilities are not adequate to deal with the problem effectively. The possibility of having specialists in neurological sciences at the district health services level may need to be considered, so that specialist acute care is made

available to larger numbers of people requiring them. Better quality and better coverage can be achieved, at costs which may not be very high, through such decentralisation. Planning for rehabilitation would also be made easier, since the DRCs are present at the district headquarters level and can be involved from the acute care stage onwards. A multidisciplinary rehabilitation team which is necessary for TBI rehabilitation, and many of whom are already available at the DRC, can be utilised more effectively from the outset. At the DRC level, the existing staff would require additional training inputs in relation to TBI, such as specialised skills like cognitive remediation, dealing with adults more than children, and more emphasis on vocational training than education.

Following the acute care phase, most people with TBI today return to their families, with no formal rehabilitation programmes being planned for them. In most cases, the families carry out some form of spontaneous rehabilitation at home, with the little knowledge at their disposal. What they need are the technical skills to upgrade the quality of home and community based care and rehabilitation. This is where the village rehabilitation workers of existing CBR programmes can play a role. These workers can follow up peoplewith TBI right from the time of discharge from the acute care facility, down to the home level in the communities. They can provide the necessary technical skills to the families and thereby improve the quality of care. What is required are the necessary training-inputs for the village workers, to take on TBI rehabilitation into their ongoing portfolio of responsibilities. However, certain factors need to be kept in mind in promoting CBR for people with TBI, which set them apart from people with other disabilities; people with TBI are usually from the productive, active, younger adult age group who previously led "normal" lives; the disability is sudden in onset; they are likely to have multiple disabilities, including cognitive, behavioural and emotional deficits; the sequelae of TBI require long term ediation wouinterventions; newer specialist techniques such as cognitive remediation would be necessary; family counselling and supports need greater attention, as would the economic, social, psychological, and emotional implications.

The possibility of using family members as therapists may also need to be explored. This may be more feasible in India, with its largely intact extended family networks and social support systems. Issues to be examined are the ability and willingness of the rehabilitation workers and the families of people with TBI to absorb the new skills needed; and the question of what degree of severity of the disability can be managed adequately at the peripheral level and by the families. It may not be possible for all people with TBI to benefit from such programmes, but at least in some cases, the

quality of care can be improved, with little additional costs. Promoting of self help groups of families and of people with TBI at the community, also needs to be encouraged, along with associations of people with TBI, to promote independent living and self empowerment. These groups would also need to be supported in advocacy efforts to bring about changes in their environment and to reduce the handicaps they face in daily living.

Since rehabilitation of people with TBI is relatively new in India, there are certain important issues to be addressed for the rehabilitation effort to be successful.

a) It may be necessary to include TBI rehabilitation into the curriculum in medical schools and training institutes for therapists, psychologists, social workers, CBR workers and other rehabilitation personnel.

b) Additing TBI rehabilitation as a subject in existing information and resources centres at different levels - national, regional, community - would be of help to those involved in rehabilitation care, including families.

c) Existing centres for aids, appliances and adaptive equipment will need to be oriented towards the needs of people with TBI as well.

d) More attention needs to be focused on research on different aspects of TBI, starting with epidemiology and going on to factors influencing long term outcomes in rehabilitation.

e) Therre is the need to develop policies and legislation at the national level, with particular reference to prevention of TBI, improved facilities for acute care and rehabilitation.

The rehabilitation scenario in India is an evolving one. The efforts of the Government and NGOs as of today are to improve coverage and facilitate integration of all disabled persons in the community. Since the incidence of TBI is likely to increase in the future and since it is likely to contribute to the increased incidence of disabilities in the country, it is important for planners and policy makers to recognise the importance of the rehabilitation effort in making people with TBI productive members of society to the extent possible. It is also necessary to consider that such rehabilitation efforts can be integrated into the framework of the existing rehabilitation plans at a slight extra cost,

without the necessity of setting up new systems and infrastructure. It may be worthwhile for some of the existing CBR programmes to take on TBI rehabilitation as part of their existing programmes.

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APPENDIX

Benefits for the disabled in India

There are various schemes for the disabled under ministry of welfare, Government of India. In December 1996 Indian Parliament passed the "Persons with disabilities" (equal opportunities protection of rights and full participation) act. The aim of this legislation is to remove all discriminations against disabled persons and integrate them into the mainstream of society. This act provides legal protection, education, employment and affirmative action for the disabled. This bill also defines disability and eligibility for benefits.

Eligibility : Indian citizens with blindness, low vision, hearing impairment, locomotion disability, mental retardation and mental illness and disfigurement due to leprosy are the beneficiaries of this act. A person with disability is defined as one suffering from not less than 40% of any of the disability as certified by a medical authority.

Prevention and early detection : According to this act the Government and local authorities are obliged to undertake surveys, research and investigations in the field of disability limitation and prevention. The public should be educated through mass media about the causes and consequences of disabilities, methods of prevention and facilities available for the disabled.

Education : The disabled children are entitled for free education, transport, books, uniforms and equipment needed for education till the age of 18 years. The Government should provide and promote special schools and vocational training facilities for the disabled.

Employment : Three percent reservation is provided for persons with disabilities. 1% each for subjects with blindness, hearing impairment and locomotion disability. Inter change of reservation among these three categories in the absence of suitable candidate under any one head is possible. If the vacancies are not filled up they shall be carried forward to next recruitment. There are 23 special employment exchanges, 55 special cells and 17 vocational rehabilitation centres for catering to the employment needs of the disabled.

Affirmative action : This act allows Government to allot land at confessional rate for the house, business, special recreational centres, special schools, research centres and factories for the disabled. There are also schemes to provide aids and appliances to persons with disabilities.

Non-discrimination : The act guarantees non-discrimination of disabled individuals in transport, employment and public buildings. Rail compartments, buses, aircraft, ships and boats must be designed to permit easy access to wheel chairs users. All the public places and waiting rooms should have toilets designed for the use of the disabled. The building rules must be amended to make ramps for wheel chairs users mandatory. Braille symbols and auditory signals must be provided in lifts and elevators. No employee can be dismissed or demoted for the reason of disability. He can be shifted to some other position with same pay and service conditions. No promotions can be denied because of disability.

Research and manpower development : National level institutions are established for research, manpower development, dissemination of information, documentation and model service for disabled. They include:

1. National Institute of Orthopedically handicapped, Calcutta,
2. National Institute for Mentally Handicapped, Secunderabad,
3. National Institute for Visually Handicapped, Dehradun,
4. National Institute for Hearing Handicapped, Bombay,
5. Institute for Physically Handicapped, New Delhi,
6. National Institute for Rehabilitation, Training and Research, Cuttack and
7. Artificial Limbs Manufacturing Corporation of India, Kanpur

Assistance to Non-Government Organisation (NGOs) : There are various schemes under ministry of welfare to provide assistance to NGOs for detection and prevention of disabilities, education, motivation and vocational training of disabled, and physical, psychological, economic and social rehabilitation of the disabled. Up to 90% of the expenditure is provided as grant-in-aid. There are separate schemes for assistance of organisations for rehabilitation of the field of cerebral palsy and mental retardation, voluntary organisations for rehabilitation of leprosy cured persons, special schools for the disabled and for distribution of aids and appliances.

District Rehabilitation Centre Scheme : The aim of this scheme is to provide community based rehabilitation services and manpower development for service delivery to disabled. Eleven district rehabilitation centres and four regional rehabilitation training centres have been established under this scheme. Four regional training centres are located at Lucknow, Cuttack, Madras and Bombay.

National awards scheme : On the occasion of "World disabled day" on third sunday of march president of India gives National awards to

1. Best employer of handicapped,
2. Best handicapped employee and self employed,
3. Best individual working for handicapped welfare,
4. Best institution working for handicapped welfare,
5. Best placement officers and
6. National technology awards for welfare of handicapped.

Travel : Railways provide 75% concession in basic fare for disabled persons and escorts. Indian Airlines allows 50% concession fare to blind persons on single journey.

Postage : Blind literature packets weighing less than 7 Kg is exempted from both inland and foreign postal charges.

Customs and excise duties : Braille paper and audiocassette for the blind imported by specified organisations are exempted from customs duty.

Allowances : All central government employees who are blind or orthopedically handicapped are granted conveyance allowance at 5% of basic pay subject to a maximum of Rs. 100. Tuition fees of physically and mentally retarded children of central government employees up to Rs.50 are reimbursed by the state.

Income tax concession : An amount of Rs. 20,0000 can be deducted from the total income of a person with disability, preventing them from engaging in gainful employment.

Award of dealership by Oil companies : Ministry of petroleum and Natural Gas has reserved 7.5% of all dealerships for orthopedically and visually handicapped persons.

Postings : Physically handicapped candidates should be posted as far as possible, near their native places. Request for transfers by physically handicapped persons are given preference by Central Government.

Economic assistance by public sector bank : All physically handicapped persons and institutions working for the disabled are eligible for loans at 5% interest rate. A subsidy of 50% up to a maximum of Rs 5,000 only is also possible.