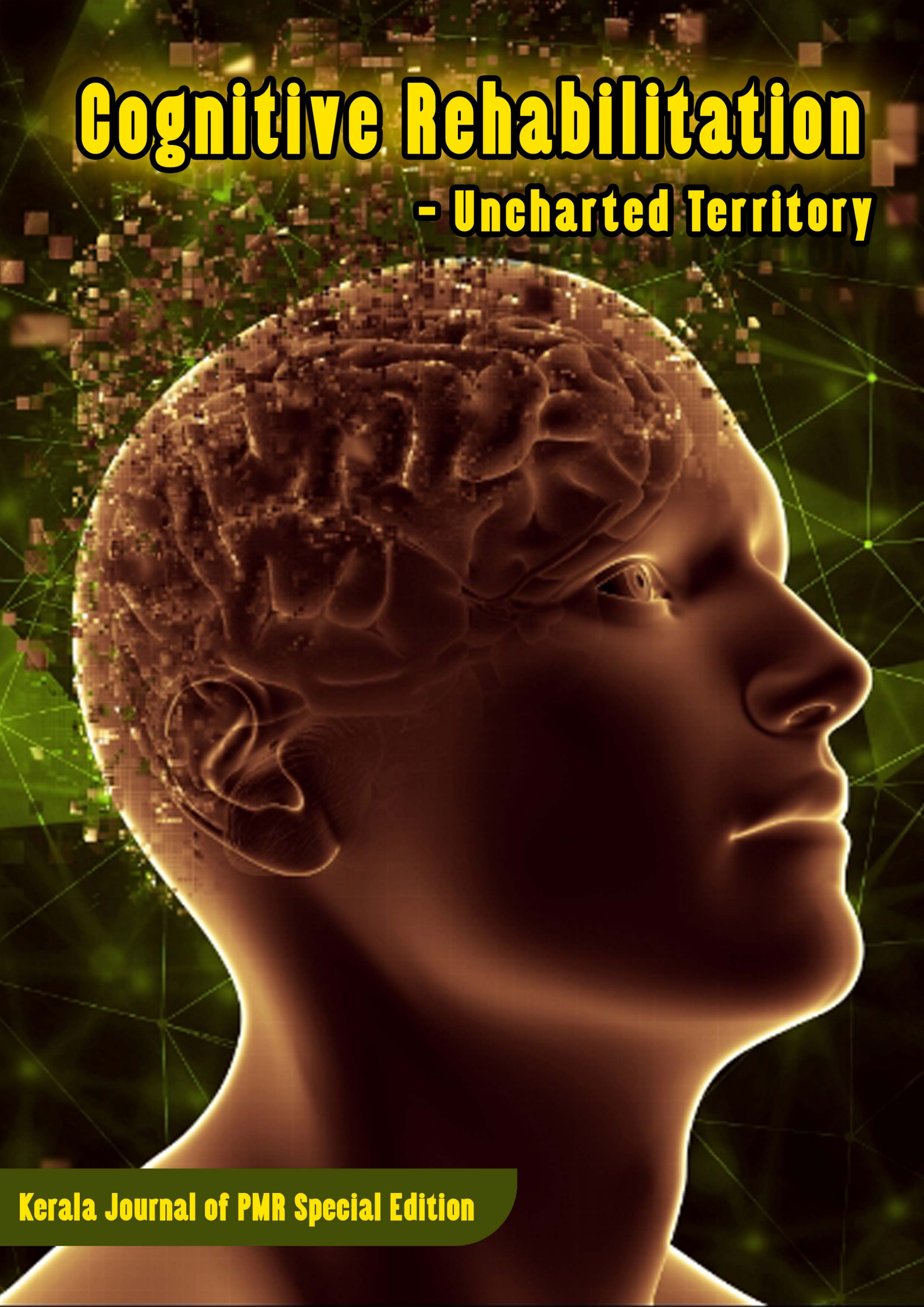
Cognitive Rehabilitation
- Uncharted Territory



Kerala Journal of PMR Special Edition

President's Message

Dr. Muralidharan P.C

President of the Kerala Chapter of IAPMR;
Organising Chairman of Rehabcon 2021

We now live in a world where rehabilitation needs are rising at a rate that it is imperative that we Physiatrists ourselves with as much arm possible, to meet them. In knowledge & skill as is medicine keeping abreast any field of modern information & with the latest daunting task. Keeping this developments is a Chapter of IAPMR decided in mind, the Kerala CME on 'Cognitive to conduct it's annual Cognition has been rehabilitation'. perceiving & interpreting described as a way of events. Though the the self, others, & digest, the neural substrates definition is easy to baffling to say the least. of Cognition are Cognition is an umbrella Add to that the fact that Executive function, term which includes Calculation, Psychomotor Attention, Memory, speed, & more....

Identifying the mainly affected domains of Cognition warrants detailed knowledge of the pathways as well as use of appropriate diagnostic tools & caveats (attention deficit can masquerade as memory deficit, etc). Modern Physiatrists should not hesitate to start new (or newer drugs) wherever/whenever needed.

Through rehabilitation we as Physiatrists aim to improve the functional status of a patient & reintegrate them back into their 'normal life'. Francis Crick, who won the Nobel Prize for the discovery of the structure of DNA, expressed the "astonishing hypothesis" that "you, your joys and your sorrows, your sense of personal identity and free will, are in fact no more than the behavior of a vast assembly of nerve cells and their associated molecules".As is obvious by now rehabilitation needs will vary widely from person to person, depending on the disease, economic status, & expectations of patients with cognitive dysfunction. Thus a holistic approach is essential while dealing with these patients.

The rehabilitation strategies available to the modern Physiatrist include:-

Environmental modifications

Medications

Occupational therapy measures

Psychological measures

Artificial Intelligence

Exergaming

Mobile Apps

To widen the horizons of Physiatrists we had included sessions from a Psychiatrist, Neurologist, Neuropsychologist, & an Occupational therapist. We hope this conference will encourage Physiatrists, young & old, to boldly manage & rehabilitate patients with Cognitive dysfunction. I thank all the faculties who participated in the deliberations and helped to convert the conference to an academic feast in an area less touched by a Physiatrist. Our faculties are experts in their respective domains and the experiences they share will definitely throw light and give encouragement to the daring Physiatrist who will take up the challenge put before him/her. I also take this opportunity to wish the new editorial team the very best. I hope they take all the steps needed to make KJPMR an indexed journal with high impact ……

From the Editor's Desk

Dear Readers,

What you are reading is the first of possibly more special issues. The theme is centered around our conference on Cognition conducted towards the end of August in 2021. Eminent Physiatrists like Dr Abhishek Srivatsava, Dr Raji Thomas, Dr Uma Pandiyan, and Dr Unnikrishnan shared their expertise on the topics they were given regarding adults. The Oration was delivered by Dr. U Nandakumaran Nair, who briefed us on everything from thoughts on thinking, to Cognition, to Artificial Intelligence, & even perception & modern art. A session on assessment of Cognition was presented by an eminent Psychiatrist, Dr.Ganga Kaimal. The Pediatric symposium was carried by Dr Chitra, and Dr Ramashekar Menon dilated on Cognitive decline in the elderly. Occupational therapists and Neuro-psychologists also contributed. To wrap it up was a session on how to set up a unit. Dr George Joseph N chaired this. The virtual conference was well attended, and was carefully orchestrated by a dedicated team. The seniors expressed great appreciation for the topics covered and many mentioned how they felt enriched by this.

We've taken the highlights of the conference and brought the best to you in this issue. The last four articles are based on the free paper & poster session & are not theme based. I express my heartfelt gratitude to Dr. Ravi Sankaran sir for giving me a chance to edit this special edition. This special issue is loaded with information, so all interested can dig right in. ☺

Dr. Bineesh Balakrishnan
Editor

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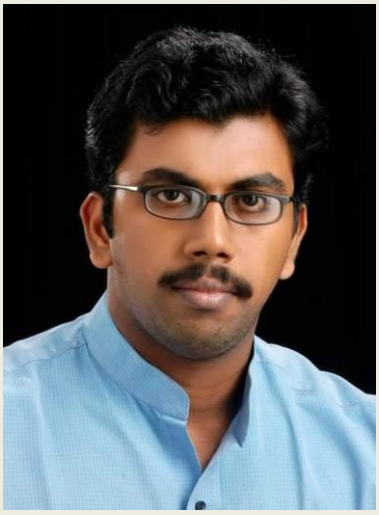
"Articles are the responsibility of the Authors"

Einstein's Brain's Afterlife…



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Albert Einstein was indisputably one of the most intelligent scientists to ever walk the earth. Most people are aware of Einstein's groundbreaking scientific achievements. He was also a world renowned peace activist & philosopher. But did you know that Einstein's brain was stolen for further research? We'll briefly follow the odyssey of his brain after his death…

It was on 18 April 1955 that Einstein the world renowned scientist, mumbled something in German & breathed his last, at Princeton Hospital. Since the nurse on duty didn't know German, we'll never get to know what his last words were. His cremation took place later that day in New Jersey, but the following day his son, Hans Albert learned that the body in the coffin had not been intact. The pathologist who conducted the autopsy, Dr. Thomas Harvey, had gone further than simply identifying the cause of death, he removed Einstein's brain for further study!! "Whether he took it for himself, or took it for science- it was hard for people to know which, & that's what put him in the crosshairs for a lot of people," says the journalist Michael Paterniti, who met Harvey near the end of his life[1] .Though Harvey was not a Neurologist, he hoped to rope in the country's biggest specialists to study the brain & publish their findings soon. But this didn't happen & with time Einstein's brain was forgotten. More than two decades later, in 1978 a young reporter, Steven Levy was assigned the task to report on the illustrious organ. Unfortunately the brain was nowhere to be seen at Princeton Medical

Center, & neither was the Pathologist, Thomas Harvey. After a laborious search, Levy eventually tracked him down to Wichita, Kansas. But then Harvey was not eager to talk. Levy kept pressing the Pathologist for an interview, & he finally yielded. Harvey was a bit reserved, & it soon became obvious that he still wished to publish that article. On repeated requests, to see photographs of Einstein's brain,

Levy was shown big mason jars with the brain. Harvey had cut the brain into 240 pieces back in 1955. He then created 12 sets of 200 slides containing tissue samples indexed to the blocks. These were then sent to the leading Neuropathologists of the time. However Harvey didn't hear back from these eminent doctors. Many studies on the slides & photographs were published, & only recently, in 2013, was a detailed description of the surface anatomy of the entire cerebral cortex of Einstein's brain reported by Dr. Falk & colleagues[2]. The unusual features of Einstein's cerebral cortex from various studies is listed below:-

The Frontal lobes:-

i. A study determined that Einstein's prefrontal cortex had a greater neuronal density than those of normal controls because it packed approximately the same number of neurons into a thinner cortex[3].
ii. As in all normal people, the central sulcus separates the primary somatosensory cortex at the front (rostral end) of the parietal lobes from the primary motor cortex at the back (caudal end) of the frontal lobes. There is a large 'knob'-shaped fold (the 'knob', known to surgeons as the sign of omega) in the right hemisphere that represents enlarged motor representation for the left hand. This is an unusual feature that is seen in some long-time right-handed violinists, such as Einstein[2].
iii. The pre-central superior sulcus is continuous with the pre-central inferior sulcus in both hemispheres, forming a long, continuous sulcus. The pre-central inferior sulcus terminates extraordinarily high above the Sylvian fissure in the left, but not the right, hemisphere. Diagonal sulcus exists in both hemispheres.
iv. The presence of unusually long and continuous mid-frontal sulcus on the right hemisphere which divides the middle frontal region into two distinct gyri, giving rise to four frontal gyri rather than the typical three gyri.
v. A long, branched inferior frontal sulcus and an additional triangular sulcus in the left hemisphere, which are located in the pars triangularis.
vi. The caudal segment of the inferior frontal sulcus is connected with both the diagonal sulcus and the pre-central inferior sulcus in the left hemisphere[2].

The Parietal Lobes:-

i. Morphological features suggest expanded primary sensory and motor representations of face and tongue in the left hemisphere.
ii. The left Sylvian fissure is separated from the postcentral inferior sulcus by a submerged gyrus.
iii. A significantly larger volume of grey matter in the angular gyrus in the left hemisphere than that in the right hemisphere.
iv. Einstein's left superior parietal lobule is separated from the inferior parietal lobule by a continuous intraparietal sulcus that stems from the postcentral inferior sulcus. In the right hemisphere, the intraparietal sulcus consists of two partly parallel segments that are joined by a shallow connection.
v. The united segments of the intraparietal sulcus on the right side course upward into the superior parietal lobule, rather than along its inferior border[2].

The Temporal Lobes:-

i. Expanded superior surface of the posterior part on the left side than on the right.

ii. Collateral sulcus is divided into two separate segments in the left hemisphere[2].

The Occipital Lobes:-

i. The locations of the anterior occipital sulcus in each hemisphere appear to be relatively caudal which may be due to caudal expansion of the inferior parietal lobules, especially on the left.
ii. In addition to the normal variation of a forked transverse occipital sulcus at the caudal end of the paroccipital sulcus in both hemispheres (which is more forked on Einstein's left hemisphere), Einstein's brain has a separate medial segment of the transverse occipital sulcus on the left that crosses the superior margin of the hemisphere.
iii. A relatively large additional transverse occipital sulcus in the right hemisphere.
iv. Each hemisphere has a lateral occipital sulcus that forks at its caudal end (a normal variation), which divides the latero-posterior region of the occipital lobes into superior and inferior parts. In Einstein's case, the superior parts are quite convoluted. Each contains a substantial superior occipital sulcus . On the left, the superior occipital sulcus forks at its rostral end into two of the aforementioned segments of the transverse occipital sulcus; on the right, the superior occipital sulcus appears as a continuation of the superior sagittal sulcus from the medial surface of the brain.
v. Surprisingly large retrocalcarine sulci course from the medial to the lateral surface of both occipital lobes, and a small lateral calcarine sulcus appears on the right.
vi. Relatively large additional transverse occipital sulcus in the right hemisphere, indicating relatively wide occipital lobes near their dorsal rostral borders[2].

The Medial & Internal Surfaces of the Brain

i. The marginal sulcus ends at the superior medial border instead of extending on the dorsolateral surface of the brain on the medial surface of the left hemisphere.
ii. The left hemisphere also has a second cingulate sulcus (sometimes called the paracingulate sulcus or the superior cingulate sulcus) that is separate from and rostral to the first. This is referred to as a 'double parallel' pattern.
iii. The middle portion of Einstein's left and right cingulate gyri each contain a long unnamed sulcus. Near the caudal end of the unnamed sulcus on the left, the cingulate sulcus gives off four inferiorly directed branches (two tiny and two long, which cross the cingulate gyrus. These features suggest that Einstein's cingulate gyri may have been relatively convoluted, especially in the left hemisphere, which manifests the double parallel cingulate sulcus .
iv. No limb of the H-shaped subparietal sulcus crosses the superior margin of the right hemisphere.
v. The cuneus of the left hemisphere are relatively ramified, containing inferior and superior sagittal sulci.
vi. The superior sagittal sulcus and the parieto-occipital sulcus are connected in the left hemisphere
vii. In both hemispheres, parieto-occipital incisures are distinct from each other and just caudal to the limiting sulcus of the precuneus. The cunei are found unusually convoluted[2].

The Petalia Pattern

The left occipital lobe is lower, wider towards the posterior end than the right occipital lobe. The right frontal lobe is wider in the anterior end than the left frontal lobe. These features contribute greatly to the asymmetry of Einstein's brain[2].

What all this adds up to?

Ideally, Einstein's brain should be evaluated in terms of its size as well as signs of neurological reorganization indicated by sulcal patterns, histological features and anatomical asymmetries. Some of this is possible now; some must await (hopefully productive) analyses of the histological slides from his brain. Einstein was 76 years old when he died in 1955, and his brain manifested certain normal signs of ageing such as a thinned cerebral cortex and widened sulci that probably contributed to a decrease in brain size, which is to be expected .

Classical Broca's speech area (and its homologue in the right hemisphere), however, is a circumscribed region of the posterior left inferior third frontal gyrus, which includes cytoarchitectonic areas, Brodmann's Area BA 44 (pars opercularis) and BA 45 (pars triangularis), the latter of which is usually bordered partly by the ascending and horizontal branches of the anterior Sylvian fissure. Today, many also include BA 47 (pars orbitalis) as part of Broca's speech area. BA 44 and BA 45 are part of the inferior frontal gyrus, which appears quite extensive in both of Einstein's hemispheres. The pattern of gyri in Einstein's right frontal lobe is highly unusual because a relatively long single midfrontal sulcus separates the middle frontal region into two distinct gyri, which gives the impression that Einstein's inferior frontal gyrus is a fourth rather than the typical inferior third frontal gyrus. This pattern and the complicated branching of the midfrontal sulcus in the rostral part of the left frontal lobe suggest that Einstein had relatively expanded prefrontal association cortices, which is interesting because in humans, the greatest part of the midfrontal sulcus is an evolutionarily new sulcus associated with expansion in this region. In Einstein's case, this cortex appears to be highly convoluted around the midfrontal sulcus in both hemispheres, including the most anterior (fronto-polar) part of the frontal lobes, which contains BA 10. The convolutions of Einstein's prefrontal lobe suggest that, volumetrically, his prefrontal association areas may have been on the high end of the range of variation for humans. This is consistent with the evolutionary hypothesis that an increase in a cortical region's number of sulci may be associated with differential expansion of its volume accompanied by increased complexity of its internal wiring[2].

BA 40 is one continuous region that is involved with short-term maintenance of phonemes and syllables during language tasks. the unusual superficial cleaving of BA 40 and seamless melding of its rostral portion with the postcentral gyrus in Einstein's left hemisphere may have been associated with his well-known delay at acquiring language and the fact that he repeated sentences to himself softly until the age of about seven[4].

The highly evolved human prefrontal cortex, including BA 10, is just the sort of mental machinery that one would use for thought experiments like those Einstein famously (and productively) engaged in, such as imagining himself riding alongside a light beam, and being enclosed in an elevator accelerating up through space. Such imaginings might additionally recruit parietal and occipital association cortices.

The newly recovered photographs also underscore the extraordinary expansion of the lateral part of Einstein's left primary

somatosensory and left primary motor cortices. It is difficult to interpret the expanded lower parts of the primary sensory and motor cortices, other than to note that these parts of the brain normally represent face and tongue. In this context, it is interesting that Einstein famously wrote that thinking entailed an association of images and 'feelings', and that, for him, the elements of thought were, not only visual, but also 'muscular' .

Another interesting feature of Einstein's primary motor cortex is the enlarged 'knob' that represents the left hand, which is probably associated with his early and extensive training on the violin.

The surface areas and shapes of the inferior and superior parietal lobules of Einstein's brain appear highly asymmetrical. The surface area of the angular gyrus (BA 39) and, indeed, the entire inferior parietal lobule (usually consisting of BA 39 + BA 40), appears to be substantially expanded in Einstein's left compared with the right hemisphere. This is intriguing in light of an earlier histological study that found a significantly greater ratio of glial cells to neurons in Einstein's left BA 39 compared with the average for a sample of 11 older control males, whereas comparable ratios on the right side did not differ significantly between Einstein and the others. This study is interesting in light of recent indications that glial cells may play a critical role in regulating synaptic function and plasticity.

The inferior parietal lobule on the left is concerned with language, body image and mathematics, whereas that on the right focuses more on non-verbal visuospatial processing. More specifically, the angular gyrus is involved with the recognition of visual symbols and (especially on the left) sensory aspects of language such as those entailed in reading. The supramarginal gyrus on the left is a part of Wernicke's area that is involved with speech comprehension and short-term maintenance of phonemes and syllables during linguistic tasks .

Einstein's right superior parietal lobule appears wider than the left and extends further rostrally and inferiorly, so that it is superior to the supramarginal gyrus on the right but not on the left. This suggests that Einstein's right superior parietal lobule may have been substantially larger than the left. Einstein's right intraparietal sulcus courses into the superior parietal lobule rather than remaining as its inferior border, which is highly unusual. The superior parietal lobules are involved in spatial attention, spatial orientation, localizing stimuli in space, and sensing the location of one's body parts and guiding their movements. The right superior parietal lobule may be differentially involved in certain aspects of visuospatial imagery. Interestingly, the superior parietal lobules, especially their posterior parts, function during mental arithmetic, presumably because this activity co-opts parietal circuitry involved in spatial coding . Einstein's visuospatial and mathematical thinking may have been facilitated by extraordinary parietal substrates .

The medial surfaces of the occipital lobes contain primary visual area BA 17 as well as association visual areas BA 18 and BA 19. As described above, these surfaces of Einstein's brain are very convoluted, which may be significant because visual cortex is activated when subjects close their eyes and visualize objects .

We have shown that Einstein's inferior temporal gyrus is relatively expanded on the basal surface of each hemisphere. This gyrus normally contains BA 20 and, caudal to that, BA 37, which on the left is involved in reading and has been called the 'brain's letterbox'. In general, the inferior temporal gyrus is activated during high-level processing associated with the remembering, recognizing and naming of visual

objects and forms (i.e. it facilitates the ventral 'what' visual stream).

It is also worth noting that in addition to external brain morphology, other factors, such as cortical thickness, neuronal density, the neuron to glia ratio, and brain chemicals, those also play important parts in a person's mental activities. This information is obviously not available to us, which makes it very difficult to fully understand Einstein's brain. Furthermore, we must not over interpret Einstein's brain. Because interpretation bias occurs when people are constantly aware that they are looking at the famous Einstein's brain rather than a brain of unknown identity[3]…

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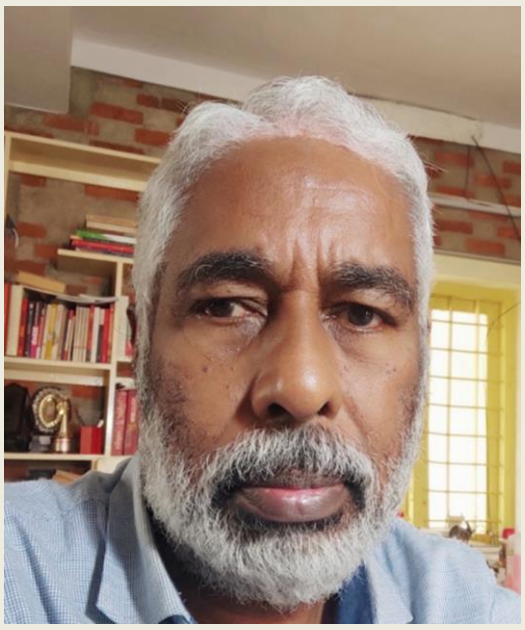
Cognition, Rehabilitation, and Other Stuff



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Thinking is a complex process and humans have been at it ever since the human brain egan to evolve. Our thoughts shape our view of the world and colour our life experiences. Perhaps, cognition can be defined as a mental process of grasping the world. This includes thoughts and experiences, understanding the world through our senses, and occupying ourselves with thought. This also needs a systematic method of communication and language.

If we look at how our thinking has changed in the last 400 years, we would see how science and technology have shaped our very concept of thought:-

- I think, therefore I exist (the clichéd words of Rene Descartes)
- I think and I exist/ I exist, therefore I think
- I exist, therefore I can pass my thinking to others
- I exist, therefore I can make objects think (when computers started helping out with complex tasks)
- I exist to make better thinking objects (with the emergence of Artificial Intelligence)

To assess the complexity of the sub-processes in thinking let's look at this problem: 2+3=5. Although it looks simple today, according to Peter Schumer, Professor of mathematics and philosophy, Middlebury, "...it took thousands of years to progress from counting "one, two, many" to our modern mathematical world." (https://theconversation.com/when-did-humans-first-learn-to-count-97511)

- First you focus your attention on the numbers (after selectively diverting attention from whatever it is you are doing), recognize them & the symbols with their meanings.
- You do the math, which for an educated man will take a few seconds.
- You verify the answer.
- You then shift your attention elsewhere, wherever it's needed.

One of the most famous myths about the brain is that most humans use only 10% of it. This is an appealing idea because it suggests that we

could achieve much more, intellectually and creatively, by using as much as is possible of the remaining 90%. This might sound inspiring but nothing could be farther from the truth. Using fMRI it is shown that a simple action like clenching & unclenching your hand or saying a few words requires activity in far more than a tenth of the brain. Even when you think you're doing nothing the brain is doing rather a lot by controlling body functions like breathing, and heart rate, or recalling the items on your to-do list. In 1980, a British Pediatrician, John Lorber mentioned that he had patients with hydrocephalus who had hardly any brain tissue, & yet could function[1].

What's in Cognition?

Cognition is a higher neural function as we all know & it has a physical basis. There are about 100 billion nerve cells in the brain. Each of these can have up to 10,000 connections with other nerve cells called neurons.

Cognition also acts as a control mechanism. This is regulated by discrete brain circuits via a number of neurotransmitter systems. A number of brain chemicals play major roles in cognitive processes; including dopamine, noradrenaline (norepinephrine), serotonin, acetylcholine, glutamate and GABA.

In cognition, there are intricate and complex considerations, calculations and formulations that are tempered by input from multiple elements such as memory, learning, and emotion. It is difficult to clearly explain the normal functioning of these higher human functions for two reasons. One, we simply do not yet know much of it, and two, current human language skills may fall short when trying to communicate such complexity. The prefrontal lobes and their connections play a major part in neurocognitive and neurobehavioral functions. Three main circuits have been identified which appear to play a major role. These circuits are structurally and functionally distinct; they traverse the frontal lobe, striatum, globus pallidus, and thalamus. The three circuits are:-

1. The dorsolateral prefrontal circuit
2. The lateral orbitofrontal circuit
3. The anterior cingulate circuit[2]

Cognition is diverse; it operates at multiple levels, individually and collectively. We have Social cognition, where we respond to emotion laden stimuli, where the following sub-processes are involved:-

- Emotion recognition- for identifying emotions in facial expressions.
- Emotional bias- where we have information processing biases for positive/negative stimuli.

Then there's Executive function, which involves a high level of thinking & decision making, which includes:-

- Mental flexibility- the ability to adapt thinking & behaviour to the task at hand.
- Working memory- which helps to devise a strategy for accomplishing the task.
- Planning- which helps in using strategy to solve the problem in a stepwise manner.
- Response inhibition- to suppress the inappropriate responses while attending to a task.

Memory, is short-term or long-term storage of information, is a part of Cognition, which includes:-

- Episodic memory- associating an event with a place or time.
- Working memory- holding & manipulating information in mind.
- Recognition memory- which involves recognition of visual, object & spatial information.

Attention, which involves attending to specific information & ignoring the others. This includes:-

- Sustained attention- which includes continuous performance (of a task) & sustained visual attention.

Psychomotor speed is also a part of cognition. This enables us with competence in:

- Choice reaction time, movement time & vigilance.

These are then phased into an elaborate network of information sharing and processing. Some of them that are part of the cognitive process are:

The Imagination Network..

Randy Buckner et al, defined the Default Network as a network one that is "constructing dynamic mental simulations based on personal past experiences such as used during remembering, thinking about the future, and generally when imagining alternative perspectives and scenarios to the present." The Imagination Network is also involved in social cognition. When we imagine what someone else is thinking, this brain network is active. The network involves areas deep inside the prefrontal cortex and temporal lobe (medial regions), along with communication with various outer and inner regions of the parietal cortex.

The Executive Attention Network…

This network is active when you're concentrating on a challenging lecture, or in complex problem solving and reasoning that puts heavy demands on working memory. This neural architecture involves efficient and reliable communication between lateral (outer) regions of the prefrontal cortex and areas toward the back (posterior) of the parietal lobe.

The Salience Network..

The Salience Network constantly monitors both external events and the internal stream of consciousness and flexibly passes the baton to whatever information is most salient to solving the task at hand. This network consists of the dorsal anterior cingulate cortices [dACC] and anterior insular [AI] and is important for dynamic switching between networks.

Mapping Thoughts on Rehabilitation…

Many eminent philosophers & scientists have propounded theories on cognition. Now we'll delve into the most important theories on Cognition & seminal developments in this field...

Lev Kuleshov's Work

Lev Kuleshov was a famous Russian film maker. Besides being a ground breaking film maker, he is most remembered for the 'Kuleshov effect'. This is a film editing (montage) effect first demonstrated by Kuleshov in the 1910s & 1920s. This is a mental phenomenon in which viewers derive more meaning from the interaction of two sequential shots than from a single shot in isolation.

Kuleshov edited a short film in which a shot of the expressionless face of a Tsarist matinee idol, Ivan Mosjoukine, was alternated with various other shots (a bowl of soup, a girl in a coffin, a woman on a divan). The film was shown to an audience who believed that the expression on the idol's face was different each time he appeared, depending on whether he was "looking at" the bowl of soup, the girl in the coffin, or the woman on the divan, showing an expression of hunger, grief, or desire, respectively. The footage of Mosjoukine was actually the same shot each time[5]. Kuleshov appears to say that while humans have the ability to read faces cognitively, they do so within contextual frames.

In Dreams…

Humans have been trying to decipher the meaning of dreams for ages. Psychologists have filled tomes with assumptions & sometimes pure drivel, about the meanings of dream sequences. Recently, a Japanese scientist broke new ground when he & his team used functional neuroimaging to scan the brains of people as they slept & enabled them to decode the visual content of their dreams. Kamitani and his colleagues built a "dream-trained decoder" based on fMRI imagery of the V1, V2, and V3 areas of the visual cortex. "We find some rule, or mapping, or pattern between what the person is seeing and what activity is happening in the brain," noted Yukiyasu Kamitani. Kamitani et al, recorded dream appearances of 20 key objects, such as "male" or "room" & used a machine-learning algorithm to correlate those concepts with the fMRI images to find patterns that could predict what people were dreaming about without having to wake them[6].

Oscar Reutersvärd (1915–2002)

The work of this Swedish artist shows the complexity that can be envisaged by the human brain. He is known as the "father of the impossible figure'. Though he reportedly suffered from dyslexia, & had difficulty in estimating the distance & size of objects, his family was artistic & encouraged his efforts at painting & sculpting. In 1934, at the age of just 18, he pioneered the art of 3D drawings that may initially appear feasible, yet cannot be physically constructed. His first work the "impossible triangle" is composed of a series of cubes in perspective.

According to the book, World of Mathematics, 2006, "the triangle at first seems like the simple geometrical shape with which all schoolchildren are familiar. However, as the eye tries to follow its outlines, the triangle abruptly becomes a dizzying experience as its bottom link plays havoc with the brain's intuitive knowledge of physical laws." He was honored by a series of Swedish postage stamps in 1982, which went out of use in 2 years, because of the change in postage rate(s), & the remaining unused stamps were destroyed by the government. These stamps are now prized collectibles[7].

Are our thoughts purely our own, or are they processed through fixed filters??

The Inheritance of Thoughts..

The human subconscious is easily conditioned by external influences. From stuff taught in school, to posters on the roadside, to political & religious ideologies, all form grooves in the way our thoughts are processed. The same image or symbol can be interpreted completely differently by two people with different upbringings. Colors, chants, or abbreviations can all be interpreted differently by different people.

The Age of Artificial Intelligence..

Much of the earth shattering work in this field was done by a scientist, Kevin Warwick. He is known for his work on direct interfaces between computer systems & the human nervous system. He & his team believe that the human brain is nothing unique, & that neuroscience & technology exist to make better thinking objects. He believes that many human limitations can be outperformed by machines & has even gone on to say-"There is no way I want to stay a mere human." As is expected most of his work is controversial to say the least. Besides AI, he is also working on deep brain stimulation for treating Parkinson's disease, developing robots, tracking devices, to name just a few[8].

Left or Right Brained…?

Another myth that needs debunking is that depending on the personality, you can state

which half of the brain is dominant in a person. The left brain is thought to be really realistic, analytical, practical, organized, and logical, and the right brain so darn creative, passionate, sensual, tasteful, colourful, vivid, and poetic!! Again fMRIs have helped to debunk this myth as well. Whether it is maths or art, both halves of the brain have a role & no specific dominance is noted while being engaged in doing them.

Our 'mental surface' is made of the momentary thoughts, explanations & sensory experiences that make up our stream of consciousness. This is all there is to mental life. It will be safe to say that our mind is flat.

We need to know a lot more on cognition; surely we are learning fast.

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Cognitive Function Assessment

Cognitive functions are the most complex functions of the brain. Cognition is a dynamic process that "involves all mechanisms by which sensory input is transformed, reduced, elaborated, stored, recovered, and used" (Urlic Neisser). This happens as a result of the organization and interactions between several systems and subsystems.

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For assessment, this complex system is being reduced into its constitutive elements and assessed systematically in a hierarchical manner.

Consciousness is the individual's ability to relate both to self and to the environment. The level of consciousnessis a continuum from full alertness to deep coma. This is assessed by the 1) intensity of the stimulation needed to arouse the patient, 2) degree and quality of movements by the patient and 3) what the patient does on cessation of stimulation.

Attention and concentration

Attention is the individual's ability to attend to a specific stimulus without being distracted by other internal or environmental stimuli and concentration is the ability to maintain attention to a specific stimulus over an extended period.

Assessment is done using Digit Span Test, "A" random letter test, Serial subtraction test.

Orientation

Test the patient's orientation with respect to time, place, and person.

Memory is the process that allows the individual to store information for later recall. The 3 stages of the memory process are 1) Registration- information is received through various sensory modalities and registered.2) Retention- It is the active process in which the registered information is stored in a more permanent form.3) Recall or retrieval which is an active process of mobilising this stored information on request or as needed. Each stage of this memory process relies on the integrity of the previous stages.

Clinically memory is divided into 1) Immediate memory which represents recall of a memory trace after an interval of a few seconds; 2) Recent memory which is the individual's

capacity to remember current, day to day events. It is the ability to learn new materials and retrieve that material after an interval of minutes, hours or days; and 3) Remote memory which is the capacity to recall facts or events that occurred years previously.

Immediate Memory is assessed by Digit span test; Recent memory is assessed by 4 word recall test and the recall of events that happened in the past 24 hrs; and remote memory is assessed by asking the patient regarding his personal information and important events that happened in the past. Visual memory is assessed by Hidden objects test.

Frontal lobe functions can be divided into motor functions and prefrontal functions. Prefrontal functions include cognitive and social-emotional processes and executive functions. This can be divided into 1) Energization which is the process of initiating or sustaining any nonreflex response; 2) Task setting which is developing and implementing a plan for carrying out activities; 3) Monitoring which is the process of checking that one remains on task over time, with adjustments in behavior as required for successful completion; 4) Behavioral/emotional regulation; and 5) Metacognition which is the "awareness,understanding, control and manipulation of the individual cognitive processes," / The ability to be aware of one's own state of mind and that of others.(Theory of mind).

Executive Functions are a subset of prefrontal functions and is the ability of the individual to cope with the continuous and ever changing challenges of the environment. Task setting and monitoring are the two processes that fit the definition of EF.

Tests used to assess the prefrontal functions are Phonemic word fluency, Hand sequencing, Drawing a sequence, go/no-go task, Stroop test, Trail Making Test, Tests of abstract thinking such as proverb interpretation and similarities. Careful history and observation of the patient are the most important steps in assessing any cognitive function, especially prefrontal functions.

Language:-

Spontaneous speech is assessed by asking the patient open ended questions.

Verbal fluency is assessed by asking the patient to generate as many words that begin with "Pa or Ma" in 1 minute.

Naming and Word finding are assessed by pointing to a variety of objects in different categories and asking the patient to name them.

Comprehension is assessed by asking the patient to point to different objects in the room and asking simple and complex questions that require only "yes" or "no" answers.

Repetition is assessed by asking the patient to repeat the word/ sentence verbatim.

Reading and writing are directly related to education. They're assessed by asking the patient to read a paragraph from a newspaper, reading and obeying and writing short sentences.

Parietal lobe function

Superior parietal lobule contains a representation of the body and limbs as well as a map of the space immediately surrounding the body. It is concerned with selecting and attending to a specific target located on the skin or in the nearby extra personal space. i.e.;"where" the target is located and the direction and velocity of the movement of the target.

Inferior parietal lobule integrates sensation of touch and proprioception with vision and

integrates information representing these senses to determine the identity of the target.ie; "what" a target is.

Parietal lobe dysfunctions

Agnosia- unable to recognize and identify objects, persons, or sounds using one or more of the senses despite otherwise normally functioning senses (Tactile agnosia, Astereoagnosia, Movement agnosia, Finger agnosia)

Apraxia- loss of the ability to execute or carry out skilled movements and gestures, despite having the desire and the physical ability to perform them. (Ideomotor apraxia, Ideational apraxia, Constructional apraxia)

Assessed by asking the patient to "Protrude your tongue", "Kick a ball", "Stand like a boxer" or asking the patient to fold a letter, place it in an envelope, seal it, address it, and place a stamp on the envelope; copying 2 and 3 dimensional drawings.

Right- left disorientation, which is assessed by identification on self, crossed commands on self, identification on examiner, and crossed commands on the examiner.

All cognitive processes are in a dynamic relationship with each other and with the other information processing systems, such as language, perception, and emotions. The function of any brain region or its dysfunction after damage cannot be understood in isolation but only in conjunction with that of other connected regions, and by considering not only how a region responds to stimuli or tasks, but its spontaneous activity and its interactions with other parts of the network."(Maurizio Corbetta).

Clinical Approach to Executive Function and Rehabilitation

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Executive function is an area of cognition which is inadequately addressed in rehabilitation. The other areas of cognition include attention, language, memory, and visuospatial skills. Executive function is particularly difficult to assess, especially at the bedside.

Frontal lobe is the largest lobe of the human brain; which continues to develop into young adulthood. The area corresponding to the executive function in the brain is the Dorsolateral prefrontal cortex (DLPFC). The sub cortical circuits connect it to the Striatum, Globus pallidus, Thalamus and back to the Dorsolateral prefrontal cortex.[i] A wide variety of aetiologies such as strokes, trauma, tumours,

multiple sclerosis and degenerative diseases can affect these circuits.

Executive function is an integrative function which includes the ability to plan, the will to act, the ability to make complicated choices of how to do it and when to stop, and to adjust the outcome. Any of these processes can fail and cause executive dysfunction. The Mini Mental State Examination (MMSE) is used to assess the instrumental function of cognition, attention, language, and memory prior to the evaluation of executive function. Some of the neuropsychological assessments for executive function are:

Volition fluency is tested by the Animal Naming Test[ii] (ANT) - The patient is assessed on their ability to name all the animals they know in one minute, wherein the individual searches all one's lexical memory to respond. (A score of 18 in one minute is normal. Less than 12 in one minute is abnormal.)

Mental control is assessed by serial subtraction as in MMSE, reverse spelling (world), naming months of the year in reverse. (If time taken to list January-December is five seconds, the reverse December to January is approximately 10 seconds.)

Assessment of memory has two components. Storage and recall – There is impairment of recall in executive function. It is a retrieval deficit, which can be assessed with cues (in amnesia - the individual cannot store and recall).

Assessment of program is done byclock-drawing (10 minutes after 11) or copying patterns of alternating programmes (alternating letters m/n).When the pattern is abnormal, there is executive dysfunction.

Assessment of (a) implementation, (b) adjusting and stopping- Trail making tests, A and B[iii] respectively. In the latter, alternate sets of alphabets and numbers are used. The individual has to disengage from one set and shift to another set. The speed of processing is also observed.

At this stage, the neurologist/neurosurgeon refers the individual to a physiatrist to improve functional outcome. The patient and the family also want functional outcomes similar to pre-illness. A neuropsychological assessment provides the limitations the patient has[iv]. It is for the multi-disciplinary team to provide restorative or compensatory strategies. The presence of family member or caregiver is beneficial to identify the short-term and long-term issues (ADL, IADL, driving, return to work).

Sometimes family members may misconstrue that the patient is disinterested or depressed or lazy. They need to be educated on executive dysfunction. They must know that the process to perform a task requires the ability to plan, to recall, program, implement, and adjust when to stop. These processes are repeated for the entire day and may be interrupted at many levels.

Initially, the general rehabilitation plan is to address short-term goals in one or two areas. Multi-disciplinary approach works best. Mood and behaviour need to be addressed and medication to be used appropriately. The tasks are broken down into simpler steps with short and direct commands. The pace of training is as important as the type and duration.

Ethical challenges faced by physiatrists in dealing with persons with executive dysfunction usually pertain to relationships, financial issues, career, acceptance of disability and parenting. In the USA, if a family/spouse does not have the best interests of the patient (e.g., risk for abuse), then an ‘ad litem’ guardian is identified and advocated. This person controls the patient's financial, medical and other conditions until the patient is determined to be capable (medical personnel determine capacity and lawyers determine competence). In our practice, we work with a psychologist and a social worker. After a point, the patient goes back to the same challenging environment which the Rehabilitation team cannot control. Our medical education needs to prepare doctors for such dilemmas.

The most important thing is that we as physiatrists appreciate and understand the challenges of executive function impairment and anticipate them during the phases of rehabilitation. It is preferable to have a team which promotes a professional approach to support patients during the process of acceptance of disability and enable them to develop skills required to lead a life of dignity.

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Cognitive Rehabilitation in TBI

Cognitive impairments are the most persistent and permanent sequelae of TBI. Rancho Los Amigos (RLA) levels of cognitive functioning helps to plan rehabilitation strategies for patients with TBI. Broadly, they are Vegetative state and Minimally conscious state, Confusional state, Post-confusional state and stage of Social competence and community re-entry. Various assessment scales are used in these stages to assess the cognitive deficits and their impact on ADL.

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Several factors influence cognitive impairment- the severity of the brain damage, medical problems, coexistent conditions, and premorbid personality. Hearing, vision, ADL, communication, speech, behaviour, and emotional stability should be assessed**.** Infections, sleeplessness, hydrocephalus, and side effects of medications should be considered whenever there is a cognitive decline.

In RLA 1, 2 and 3, where arousal is the main problem, multimodal sensory stimulation is given. Amantadine and other Dopaminergic agents are used as neurostimulants. Electrical stimulation of the right median nerve(1),Noninvasive brain stimulation (2) and Deep brain stimulation have been tried.(3)

RLA Stage 4 is characterised by post traumatic amnesia, severe attentional deficit and agitation. The Galveston Orientation and Amnesia test serially measures orientation.(4)Learning through repetition is possible in this stage. Orientation to time, person, place is improved using a structured environment, minimizing unfamiliar and unexpected situations and giving one activity at a time. Establishing a good sleep hygiene is crucial. Agitation is measured using Agitated Behaviour Scale.(5)Post traumatic delirium due to infections, electrolyte or metabolic disturbances have to be ruled out.One-to-one

monitoring, behavioural modification, physical restraints, environmental modification and medications areused to reduce agitation. Antecedent-Behavior and Consequence charting will help to identify and avoid antecedents. Medications such as Beta blockers , Carbamazepine, Antipsychotics like Quetiapine, Risperidone or Olanzapine may have to be initiated starting slowly, increasing slowly with the aim of stopping as soon as possible(6).Short-acting benzodiazepines or atypical antipsychotics may be used during severe agitation.(7)

In RLA Stage 5 or the confused-inappropriate stage, patients continue to be disoriented. They may show confabulation and perseveration. In Stage 6 or the confused-appropriate stage, they can follow a schedule but are confused with changes of routine. In RLA 7 and 8 or the post confusional states, patients haveproblems withplanning, starting, and following through with activities. They can be rigid and need supervision. In the next stage, they realize that they have problems and begin to compensate. They needhelp in new situations and to make decisions.Addenbrooke's Cognitive Examination, Montreal Cognitive Assessment (MoCA), Cognitive Linguistics Quick Test and Loewenstein Occupational Therapy Cognitive Assessment (LOTCA) may be used for assessment. Detailed neuropsychological testing is done when ready to return to school or work. In Multiple Errands Test (MET**)** real-world tasks are given to check executive functions.

Cognitive deficits following TBI include deficits in attention, memory and visuospatial processing, slow information processing, intellectual decline, executive function deficits as well as problems with social cognition, spatial cognition and cognitive communication.(8)

Amantadine(9) and Methyl Phenidate (10)may improve arousal and attention , the latter drug without strong evidence.Hyperbaric oxygen therapy is also useful. (11)Rivastigmine (3-6 mg/day)(12) and Donepezil (5-10 mg / day) (13)are used to improve attention and memory . Bromocriptine (2.5 mg/day)(14)and Amantadine(maximum 400 mg/ day)(15)may improve executive function.

Cognitive retraining includes Process Specific Training, Training of Compensatory Strategies and Functional Skills BasedTraining. In process training, multiple repetitions of the cognitive skill is done through paper pencil tasks, table top tasks or computer based training. In training of compensatory strategies, internal and external strategies are utilised to improve the cognitive domain. In Skills Based Training, the functional activity is practised repeatedly, breaking the task into simpler steps and in an errorless manner. Assistive technologies such as portable voice organizers and pagers have been devised as compensatory strategies.(16)Support groups provide emotional support and networking.(17) Coordination with employers help to develop work schedules and supported work environment. Comprehensive-holistic programs integrate activities for cognitive remediation, functional skills and interpersonal functions. Transcranial magnetic stimulation and tDCS, (2)Virtual Reality, (18)Rehabilitation Game System(19)*and* Prism Adaptation for Visual Neglect(20)have been studied.

Cognitive rehabilitation is effective for patients with TBI(21)and should be directed at improving patients' everyday functioning and quality of life. Treatment should be individualized according to the needsof the patient at the particular phase of recovery.

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Pharmacotherapy for Cognitive Rehabilitation Post Traumatic Brain Injury

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Introduction

Cognitive impairments are common in patients recovering from brain injuries. They can be a result of direct cortical, subcortical, or brainstem injury or disturbances in neurochemical pathways that modulate function within these networks. More commonly seen cognitive disturbances are hypo arousal, slowing of information processing, attention deficits, memory impairments, communication disturbances, and executive dysfunction. In patients who show significant improvements in motor function following brain injuries, cognitive deficits may become apparent and interfere with functional rehabilitation.

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Cognitive rehabilitation is an integral part of neurological rehabilitation, which involves functionally oriented therapeutic activities, which is prescribed on basis on systematic cognitive assessment and understanding of the patient's medical condition. Therapeutic interventions are aimed to reinforce previously learned patterns of behavior or create new patterns of cognitive activity using compensatory strategies to overcome the impaired cognitive domains. Multiple domains of cognition such as attention, comprehension, memory, communication, reasoning, planning, problem solving, judgement and self-monitoring needs can be targeted with cognitive rehabilitation. Sufficient evidence exists supporting the efficacy and effectiveness of cognitive rehabilitation, which has become the treatment of choice for

cognitive impairments and leads to improvements in cognitive and psychosocial functioning. Various rehabilitation techniques in for cognitive training are Attention training, Memory training, Mental imagery techniques, Dual tasking strategies like cognitive-motor training, low intensity exercise programs and multimodal programs. Other interventions used for improving cognition are music therapy, board games, puzzles, computer based training and virtual reality. While the literature is mixed regarding outcomes with cognitive retraining, some studies have been shown it to be effective for mild to severe brain injuries and found it to be beneficial at any time post-injury[1].

Pharmacotherapy is another intervention in cognitive rehabilitation, which is regarded as an adjunct to non-pharmacologic therapies. Our present understanding of the neurochemistry of cognition and the neurochemical basis of posttraumatic cognitive impairments suggest that augmentation of cerebral catecholaminergic and cholinergic function are likely to be the most useful neurochemical targets for pharmacologic intervention in this population[2].

Neurotransmitters in Cognition

The effect of brain injury on the CNS neurotransmitter system will provide some insights on the rationale of pharmacotherapy for cognitive impairments. While the relationship between neurotransmitter dysfunction and cognitive impairments are not definitely established, the present evidence suggests that excess of glutamate, acetylcholine and catecholamines have neurotoxic effects. Such destructive effects have been found to be greatest in basal forebrain, hippocampus, striatum, and frontal cortices as the neurotransmitters are co-localized in these areas. As the acute neurotransmitter storm resolves in the subacute period, long-term deficits develop in cerebral cholinergic systems and possibly also in catecholaminergic systems. Deficits in these systems would be expected to contribute to impairments in the cognitive functions they support: arousal, speed of processing, attention, memory, and executive function.

Glutamate-mediated neuronal damage is found to be particularly severe in hippocampal and frontal cortices where glutamate is both the dominant and key neurotransmitter and can lead to impairments in the cognitive functions served by these areas, like memory and executive functions[3]. Acetylcholine appears to play a key role in the development of normal arousal, attention and memory. Brain injury produces acute elevations and long-term deficits in cortical cholinergic function which can cause memory impairments[4].
The major cerebral catecholamines, dopamine and norepinephrine, serve as key modulators of information processing circuits in the brain and strategies to augment them are reasonable targets for pharmacotherapies in cognitive dysfunction[5]. Persistent posttraumatic serotonergic deficits are likely to be relevant to the development of posttraumatic mood and anxiety disorders, which may themselves contribute to posttraumatic cognitive impairments. Treatment of posttraumatic depression with serotonin reuptake inhibitors may facilitate improvement in depression related cognitive impairments[6].

Pharmacotherapy

Pharmacotherapy is used in Cognitive Rehabilitation for Arousal, Facilitating speech & language recovery, Recovery of cognitive functions and Modulation of mood & behavior.

Pharmacotherapy for Improving Arousal

The commonly used drugs for improving arousal include:

Amantidine

Amantidine is a partial Dopamine Agonist & NMDA antagonist. Besides being used as drug in Parkinsonism, it is also a sheet anchor in managing disorders of consciousness in

traumatic brain injury. A Placebo Controlled Trial with 184 patients in vegetative or minimally conscious state following TBI were randomly assigned to receive placebo or Amantidine for 4 weeks & were then followed up for 2 weeks after the treatment was discontinued. The rate of functional recovery on the Disability Rating Scale was compared. During the 4 week treatment period, the recovery was significantly faster in the Amantidine group than in the placebo group indicating a benefit with primary outcome measure. In a pre-specified subgroup analysis, the treatment effect was similar for patients in a vegetative state & those in a minimally conscious state. The rate of improvement in the Amantidine group slowed during the 2 weeks after treatment (weeks 5 & 6) and was significantly slower than the rate in the placebo group. There were no significant differences in the incidence of serious adverse events[7].

Z Drugs

These drugs appear to exert their CNS effects via interaction with benzodiazepine receptors, classified as BZ1 or ω1 subtypes. In contrast to benzodiazepines, these drugs bind more selectively, interacting only with $GABA_A$ receptor isoforms that contain α1 subunits. Z-drugs" such as zopiclone and zolpidem, show apparently paradoxical arousal effects. However such effects have only been observed in individual cases of coma treatment. Due to the minimal potential for side effects, trial use of zolpidem for a few days may be useful for patients with unresponsive wakefulness as well as minimal conscious state[8].

Methylphenidate

Methylphenidate is a catecholamine agonist used widely for managing Attention Deficit & Hyperactivity Disorder. It can be used for increasing arousal in patients with disorders of consciousness. A Meta-analysis of Methylphenidate on command following and communication in brain injured patients with severe disorders of consciousness (DoC) did not identify a clinically meaningful effect in the overall study group or in any subgroup. Methylphenidate has been shown to have some positive effects in improving attention deficits in higher-level patients with TBI[9].

Pharmacotherapy for Improving Speech &Language

Pharmacotherapy of Post Stroke Aphasia is an area of much interest, with researchers trying to prove if the drug is superior to standard Speech Language Therapy in hastening recovery. The clinical examination of language should include the assessment of naming, spontaneous speech, comprehension, repetition, reading, and writing. A deficit of naming (anomia) is the single most common finding in aphasic patients. Nonfluent aphasias are more common and are characterized by impaired articulation, melodic production, phase length and decreased grammatical complexity. The production of fewer than 15 words per minute is used to define nonfluent aphasias.

Bromocriptine

An ergot alkaloid, bromocriptine acts as a partial agonist at dopamine D2 receptors in the brain. The drug increases the functional activity of dopamine neurotransmitter pathways, including those involved in extrapyramidal functions. A double-blind study with high dosage of Bromocriptine(30 to 60 mg in divided doses) prescribed according to a dose-escalating protocol and Speech Therapy in non-fluent chronic aphasia after stroke found a late recovery in stable chronic non-fluent aphasia[10]. In another study, a randomized, double-blind trial of bromocriptine in nonfluent aphasia with 38 patients assessed for 16 weeks failed to show any benefit.

Amphetamine

Amphetamines alter transporters of CNS amines including dopamine, norepinephrine, and serotonin, and increase their release.A Double-Blind, Placebo-Controlled Prospective

Study of the Use of Amphetamine in the Treatment of Aphasia using 10mg of dextroamphetaminefound recovery from aphasia in a small group of patients in the subacute period after stroke. Neuromodulation with dextroamphetamine, & perhaps other drugs that increase central nervous system noradrenaline levels, may facilitate recovery when paired with focused behavioral treatment. There is level 1a evidence that Dextroamphetamine appears to improve aphasia recovery when combined with language therapy[11].

Levodopa

Levodopa is a dopamine precursor used in the management of Parkinson's disease, often in combination with carbidopa, a drug that prolongs the half-life of levodopa. A prospective, randomized, placebo-controlled, double-blind study to evaluate the Effect of Levodopa Combined with SLT on Functional Recovery from Aphasia found greater language improvement in verbal fluency and repetition, compared to patients receiving placebo[12].

Piracetam

A randomized, placebo-controlled trial evaluated the role of piracetam, a γ-aminobutyric acid derivative with action on acetylcholine and glutamate combined with intensive SLT. Significant improvement was found in spontaneous speech, comprehension, naming, written language and verbal communication[13].

Donepezil

This is an Acetyl Cholinesterase Inhibitor used in Alzheimer's disease. In a study probing role of Donepezil & Constraint Induced Aphasia Therapy (CIAT) in patients with chronic anomic aphasia, fMRI was done during a naming task & it was compared to the activity in the brain at rest. At baseline the patient had moderately impaired performance on picture naming (33/42) and required widespread, bilateral activation in order to perform the task. After 8 weeks of treatment with donepezil (10 mg/day) naming improved (40/42, or .95) and a much more focal network was engaged. After combined donepezil and language rehabilitation with constraint-induced aphasia therapy (CIAT) naming performance was flawless (42/42, or 1.0) and a more left-hemisphere perisylvian areas were activated.Donepezil may be beneficial for improving naming, but not social communication, repetition, general and auditory comprehension, and global speech and language. The level of evidence is 1b for the above statement, based on 1 RCT[14].

Memantine

Memantine is an NMDA antagonist used in Alzheimer's disease. A study of memantine & Constraint-Induced Aphasia Therapy in Chronic Post Stroke Aphasia showed memantine (20mg/day) group had significantly better improvement on Western Aphasia Battery-Aphasia Quotient compared with the placebo. The beneficial effects of memantine & CIAT persisted on long-term follow up. Memantine may be beneficial for improving discourse, naming, social communication auditory comprehension and global speech and language, but not repetition[15]. The level of evidence is 1b & is based on 1 RCT.

Pharmacotherapy For the Recovery of Cognitive Function

Methylphenidate

Methylphenidate has been shown to have some positive effects in improving attention deficits in higher-level patients with TBI [9].Analysis of the ten double-blind RCTs demonstrated significant benefit in using methylphenidate for enhancing vigilance-associated attention (i.e., selective, sustained, and divided attention) in patients with, especially in sustained attention. However, no significant positive impact was noted on the facilitation of memory or processing speed. More studies on the efficacy

and safety of methylphenidate for the cognitive improvement of patients with TBIs are warranted[16].

Memantine

Excitotoxicity and apoptosis occur after TBI and are mediated through the N-methyl-D-aspartate (NMDA)-type glutamate receptor. Memantine is effective in blocking excessive activity of NMDA-type glutamate receptors and reduces the progression of dementia and may have benefits after TBI. The review shows that despite laboratory and clinical evidence reporting reduced serum NSE and improved GCS, supporting the existence of the neuroprotective properties, there is a lack of reported evidence from RCTs to suggest that memantine directly leads to cognitive improvements in TBI patients[17].

Central acetylcholinesterase Inhibitors

Central acetylcholinesterase inhibitors (CAIs) could reduce cognitive symptoms of chronic traumatic brain injury. There were no significant differences between the three drugs either in effect or tolerability. The age, sex, type of injury, severity of TBI or elapsed time after injury did not affect the response. The mean dose in maintenance therapy was 7.2 mg od for donepezil, 5.0 mg bid for galantamine and 2.3 mg bid for rivastigmine. Side effects or inadequate therapeutic response were the main causes for discontinuation with nearly equal frequency. Paradoxical responses were seen in some patients. CAIs show a very promising therapeutic potential in the treatment of chronic TBI. There were no significant differences between the three drugs. Large-scale randomised double-blinded placebo-controlled studies are clearly needed[18].

Rivastigmine

Rivastigmine is a Carbamate Acetyl Cholinesterase Inhibitor used in Alzheimer's dementia. It is the most commonly used drug for enhancing the recovery of Cognitive function. In a study of Long-term effects of Rivastigmine in patients with traumatic brain injury, patients with traumatic brain injury (TBI) and persistent cognitive impairment received rivastigmine (3–6 mg/day) or placebo. Statistically significant changes were seen in cognitive tests including the Hopkins Verbal Learning Test (HVLT) and the Cambridge Neuropsychological Test Automated Battery Rapid Visual Information Processing (CANTAB RVIP). Treatment with rivastigmine for up to 38 weeks was safe in patients with TBI and cognitive impairment. In a Cochrane Review, Birks et al. (2013) found three RCTs examining the use of rivastigmine for patients with VCI and vascular dementia[19]. The authors concluded that there was insufficient evidence to support or refute the use of rivastigmine in the treatment of dementia, given that only one study detected a benefit on cognition.

Pharmacotherapy for Managing Mood &Behavior

Many different types of antidepressants are being used for managing mood and behavioural symptoms in traumatic brain injury. In general, antidepressants enhance serotonergic or noradrenergic transmission. Selective serotonin reuptake inhibitors, commonly called SSRIs, have been found to be the most effective antidepressants for people with TBI. Specifically, sertraline and citalopram may have the fewest side effects and may even improve cognitive function.

Depression is a common complication of traumatic brain injury (TBI). Selective serotonin reuptake inhibitors are recommended as first-line treatment for depression incontemporary expert consensus clinical practice guidelines for management of TBI. This recommendation is basedon multiple prior meta-analyses of clinical trials in depression after TBI as well as depression in the generalpopulation. Patients

with mild TBI are especially appropriate for antidepressant therapy.

Agitation

It is a neurobehavioral syndrome characterized by cognitive confusion, extreme emotional lability, motor over-activity and physical or verbal aggression. The common causes of agitation include- electrolyte imbalance, malnutrition, seizure, sleep disturbance, hydrocephalus, pain, musculoskeletal injury and drug side effects. Environmental management should be tried with Floor / Craig bed, keeping cool calm environment, reduce external stimulation, keep one to one caregiver n nursing and minimize use of restraints only for self-harm or physical aggression. Pharmacotherapy include

- psychostimulants and antidepressants like Methylphenidate or TCAs in predominantly aggressive patient
- mood stabilizers like valproic acid in alert patient with episodic agitation,
- neuroleptics like Quetiapine, Aripiprazole in Crisis situations.

Amantadine, β-blockers, antiepileptics, and methylphenidate can be considered for scheduled treatment of agitation and/or aggression in patients with TBI[21].

Detrimental Drugs

Certain medications are shown to impair recovery of brain injury and are known as 'Detrimental Drugs' which are better avoided in patients with DOC[22]. Some of the medications with detrimental effects are Haloperidol (D2 Antagonist), Prazosin (Alpha-1 antagonist), Clonidine (Alpha-2 agonist), Benzodiazepines (GABA agonist), Phenytoin and Phenobarbital (GABA mechanism). Antispastic medications in higher dosages have sedative properties which can confound minimal changes in consciousness, hence their dose should be titrated as per patient tolerance. Antiepileptic medications that interfere with cognition should be avoided & optimised as per patient risk profile[23].

Conclusion

Cognitive impairments like hypo arousal, slowing of information processing, attention deficits, memory impairments, communication disturbances, and executive dysfunction are common in patients recovering from brain injuries. They can be a major barrier for functional recovery in some patients needs to be addressed effectively. Cognitive rehabilitation is an integral part of neurological rehabilitation aimed to reinforce previously learned patterns of behavior or create new patterns of cognitive activity using compensatory strategies to overcome the impaired cognitive domains. Pharmacotherapy is an intervention in cognitive rehabilitation, which is regarded as an adjunct to non-pharmacologic therapies. Multiple studies are now available, though with conflicting outcomes, suggesting increasing role of pharmacotherapy in cognitive rehabilitation. The use of amantadine can be recommended for the improvement of the level of consciousness.Dopaminergic drugs have been shown in smaller studies to positively influence functional recovery. Donepezil and Memantine can be used for patients with global and motor aphasia.Rivastigmine is well tolerated and improve cognitive function.Paroxetine is safest and Duoloxetine is fastest acting antidepressant.

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Effect of Cognitive Retraining on Neuropsychological Test Performance of Patients with Mild Cognitive Impairment due to Alzheimer's Disease- A Single Centre Experience

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ABSTRACT

Cognitive impairments, and particularly memory deficits, are a defining feature of the early stages of Alzheimer's disease (AD) and vascular dementia. Interventions that target these cognitive deficits and the associated difficulties with activities of daily living are the subject of ever-growing interest. Cognitive retraining (CR) seeks to directly improve and/or restore cognitive functions utilizing a variety of pen and paper or computerized tests or games requiring cognitive skills such as attention, planning, problem-solving, and/or memory. The present study is to examine the effect of CR on neuropsychological test performance of patients with mild cognitive impairment due to Alzheimer's disease. Twenty participants (age 50-85) who were patients with mild cognitive impairment (MCI) due to AD diagnosed as per Petersen's criteria and NINCDS–ADRDA criteria with clinical dementia rating score of < 2. The participants are recruited through the Memory and Neurobehavioral Disorders Clinic, Dept. of Neurology SCTIMST, Trivandrum. Neuropsychological tests were performed before and after 9 weeks (one hour per week) of cognitive retraining interventions. Patients underwent baseline and serial neuropsychological testing at 3 months and 1 year of followup as per our validated protocol The result indicates that participants showed significant improvement in MMSE (p=0.001), ACE Total (p= 0.028), ACE recall (p=0.000),

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ACE orientation (p=0.019), ACE language(p=0.027), ACE categorical fluency (p= 0.021), ACE visuospatial (p=0.009), clock drawing test (p= 0.035), CDR (p=0.001) at 3 months with no further decline at 1 year. CR is thus a valid approach towards stabilising cognitive functions.

INTRODUCTION

Cognitive retraining (CR) is a therapeutic strategy that seeks to improve or restore a person's skills in the areas of paying attention, remembering, organizing, reasoning and understanding, problem-solving, decision making, and higher-level cognitive abilities. These skills are all interrelated. CR is one aspect of cognitive rehabilitation, a comprehensive approach to restoring such skills after brain injury or other disability. Professionals from a variety of fields, such as psychology, psychiatry, occupational therapy, and speech-language pathology , may be involved in cognitive retraining. The techniques of cognitive retraining are best known for their use with persons who have suffered a brain injury. Cognitive retraining has also been used to treat dementia , schizophrenia , attention-deficit disorder, learning disabilities, and cognitive changes associated with aging. Types of cognitive retraining are given below:-

Attention and concentration retraining- This type of CR aims to improve several abilities, including focusing attention; dividing attention; maintaining attention while reducing the effects of boredom, fatigue and resisting distraction. Attention has been considered the foundation of other more complicated cognitive skills, and therefore an important skill for cognitive retraining. This area of CR has been widely researched, and has been shown to improve patients' abilities in various tasks related to attention.

Memory retraining- Memory retraining involves teaching the patient several strategies that can be used to recall certain types of information. For example, rhymes may be used as a memory aid. A series of numbers, such as a phone number with an area code, may be broken down into smaller groups. A person may be taught to go through each letter of the alphabet until he or she remembers someone's name. Both memory and organization problems are common and disabling after head injury.

Organizational skills retraining- This approach is used when the person has difficulty keeping track of or finding items, doing tasks in a set order, and/or doing something in a timely manner. Strategies may include having one identified place for an item ("a place for everything and everything in its place"). In addition, the person can be taught to keep the items that are used most frequently closer to him or her (the front or the lower shelves of a cabinet, drawer, closet, or desk, for example). Items that are often used together (such as comb and brush, toothbrush and toothpaste) are placed beside each other. Items may be put into categories (Christmas decorations, Easter decorations, for example). These strategies help individuals function better in their home or work environment.

Reasoning-. Reasoning refers to the ability to connect and organize information in a logical, rational way. Reasoning retraining techniques include: listing the facts or reality of a situation; excluding irrelevant facts or details; putting the steps to solve a problem in a logical order; and avoiding irrational thinking, such as jumping to conclusions based on incomplete information, or focusing on the negative aspects of the situation and ignoring the positive. When the person can connect relevant information in a logical way, they are better able to understand or comprehend it.

Problem solving- Problem-solving retraining aims to help people define a problem; come up with possible solutions to it; discuss the solution(s) with others and listen to their advice; review the various possible solutions from many perspectives; and evaluate whether the problem was solved after going through these

steps. This sequence may be repeated several times until the problem is solved. This process is referred to as "SOLVE," from the first letter of the name of each step: Specify; Options; Listen; Vary; and Evaluate. The "SOLVE" technique is more appropriate for use with individuals at a higher level of functioning.

Decision making- Decision-making retraining is used when a person must choose among a number of options. The goal of this retraining is to help him or her consider the decision thoroughly before taking any action. The considerations may range from such practical matters as money, people, rules and policies, to personality issues.

Executive skills- Executive skills retraining or metacognitive strategies refer to teaching individuals how to monitor themselves, control their thinking and actions, think in advance, set goals, manage time, act in socially acceptable ways, and transfer skills to new situations. These are higher-level cognitive skills. Charts and videotapes may be used to monitor behaviour, and a variety of questions, tasks, and games may be used in retraining these skills.

Dementia due to Alzheimer's and other diseases is a leading cause of disability and an enormous health and societal problem. More than 40 million people in the world currently live with dementia, and this number is expected to increase to more than 115 million by the year 2050. Effective treatments to reduce the burden of dementia are urgently needed. Cognitive training (CT) is a non-pharmacological form of treatment that focuses on guided practice on tasks that target specific cognitive functions, such as memory, attention, or problem-solving. Whether CT can help people with mild to moderate dementia maintain or improve their thinking, well-being, and general functioning remains unclear. Alzheimer's disease (AD) is the most common age-related neurodegenerative disorder. Cardinal AD symptoms are memory impairment and cognitive function decline. Mild cognitive impairment (MCI) refers to the transition state between the asymptomatic stage and dementia. According to the affected cognitive domain, MCI is generally divided into amnestic MCI (aMCI) and non-amnestic MCI. aMCI is primarily manifested by memory deficits with or without other cognitive impairments, while non-amnestic MCI is mainly characterized by cognitive impairments other than memory deficits, such as decline in executive function, linguistic function, visuospatial function, and attention. Approximately 10–15% of aMCI patients annually progress to AD and variably up to 30-80% progress to AD dementia within 10 years, although a proportion of this group never progress to dementia and can remain clinically stable or revert back to normal cognition. Up until now there are no proven disease modifying therapies therapies available to reverse or mitigate this process. An efficacious non-medical approach is thus required for MCI interventions.Cognitive training is a promising non-drug alternative to slow MCI-to-AD progression. Studies suggest that game-style cognitive training can effectively improve the cognitive function of elderly individuals and even reverse the age-dependent decline in cognitive capacity. Positive effect of cognitive retraining in healthy adults has been reported, but literature is sparse and Level A evidence regarding its effect in patients with MCI due to AD is lacking. More studies are required to clarify the effect of retraining in dementia patient. The objective of the study is to determine the effect of cognitive retraining on neuro psychological tests of patient with MCI due to AD.

MATERIALS AND METHODS

This prospective pilot study customized weekly one hour nine -week individual -based cognitive retraining program for older adults with MCI due to AD diagnosed as per Petersen's criteria and NINCDS–ADRDA criteria with clinical dementia rating score of < 2 and a sample

comprised of 20 patients (males -, females-) after exclusion of significant anxiety and depression on Hospital anxiety depression scale (HADS). The participants are recruited through the Memory and Neurobehavioral Disorders Clinic, Dept. of neurology SCTIMST, Trivandrum.

Cognitive retraining

The cognitive retraining is carried out weekly for 1 hour. Feedback on the performance of the retraining tasks is given at the end of each session. The patient is given home-based tasks for practicing during the rest of the days. Cognitive retraining is given for the following cognitive domains.

Slno	Cognitive domains	Weeks
1	Orientation	Week 1-8
2	Attention	Week 1-9
3	Mentalspeed/ information processing Letter cancellation Grain sorting	 Week 1-2 Week 1-4
4	Working memory Mental arithmetic problems Rearrangement of jumbled words Rearrangement of jumbled sentences	 Week 1-2 Week 3-4 Week 4-6
5	Verbal fluency	Week 4
6	Response inhibition Design coloring Stop start task	 Week 1-6 Week 5-6
7	Planning Maze completion Planning events Short essay writing	 Week 6-8 Week 6-7 Week 7-8
8	Se shifting ability Categorization of beads Categorization of cards Motor set shifting	 Week 3 Week 4-5 Week 4-5
9	Problem solving	Week 6
10	Visuo-constructive ability	Week 5
11	Visuospatial reasoning	Week 6
12	Abstraction Similarity and differences Proverb illustration	 Week 6 Week 7
13	Verbal memory	Week 2-6
14	Visual memory Memory for location Memory for designs	 Week 2-6 Week 6-7

Orientation &Working memory

Level 1:Help the patient to develop a highly structured daily schedule. Schedule everything that the patient should or will do during the day. Create a simple form with times of the day organized into 30-minute slots of time. Start with the basics of getting up in the morning, washing face, brushing teeth, etc. Schedule meals, chores and "rehab" activities. Also remember to schedule in break periods several times a day, which should last 30 minutes to an hour. Every day should be similar in schedule. Create and review each day's schedule with the patient and keep the schedule posted in several places around the home. Refer to the schedule often with the patient throughout the day, especially when changing activities. Ask questions such as, "What are you supposed to do next?" When appropriate, have the patient fill out the schedule with your assistance.

Level 2: Either use a calendar already in the patient's home or make a realistic one up on paper. Ask the patient to tell you what day, date, month, and year it is. Show the patient on the Calendar what the date is. Cross off days that have passed. Review this date with the patient often throughout the session and the day. Write important events, such as appointments and birthdays, on the calendar. Talk about the events coming up in the next several days. Encourage caregivers to keep calendars around the house and to review the date several times a day.

Level 3: Ask the patient to provide the month, date, year, and day of the week before checking the calendar. Then proceed as in Level 2. Ask the patient to provide the date of a day removed in time. For instance, ask what the date will be tomorrow, next week, in 10 days, 5 days ago, etc. Ask the number of weeks or months until, or since, important dates, such as Christmas,

birthdays, etc. Refer to the calendar as necessary.

Level 4:Once the patient is able to follow and be responsible for his own daily schedule, encourage him or her to buy a schedule book or calendar that can easily be carried. Acquire an inexpensive electronic personal organizer or scheduler, preferably one with an alarm function on it (this will also assist with memory and problem-solving skills). These can be bought at local discount department stores or at office supply retail stores. Help the patient learn how to use the device, program the device and encourage them to use the alarm functions to increase success in recall of daily routines and schedules or even for medication times.

Level 5:The patient is asked to remember where the items of common use are kept in house and to locate them spatially

Attention

Number Connection:

Level 1: Serially connect numbers 1-20 randomly placed on an A4 size paper. Record the time taken to complete the task and number of errors. Once the patient is able to complete the task within 1 minute without errors, move on to the next difficult level.

Level 2: Serially connect the numbers 1-30 randomly placed on an A4 size paper. Reduce the font size of the number. Record the time taken to complete the task and number of errors. Once the patient is able to complete the task within 3 minutes without errors, move on to the next difficult level.

Level 3: serially connect numbers 1-40 randomly placed on an A4 size paper. Further, reduce the font size of the number. Record the time taken to complete the task and number of errors. Once the patient is able to complete the task within 5 minutes without errors discontinue the exercise.

Letter Cancellation:

Level 4: The patient is required to cancel any two target letters from an array presented in an A4 size sheet. They are also told that they can follow a horizontal, vertical, or a random path, according to their choice. Time taken to complete the task and errors of commission and omission were recorded. Once the patient is able to complete the task without errors, move on to the next difficult level.

Level 5: The patient is required to cancel three letters from an array presented in an A4 size sheet. Reduce the font size of the letter/symbol. Record the time taken to complete the task and errors.

Level 6: Repeat the level 4, and 5 tasks in the presence of a distracter. A music played during the task performance is a distracter

Symbol cancellation:

level 7: ask the patient to cancel two symbols from an array presented in an A4 size sheet. Reduce the font size of the symbol further and record the time taken to complete the task and the errors.

Level 8: The patient is required to cancel three symbols from an array presented in an A4 size sheet. Reduce the font size of the symbol. Record the time taken to complete the task and errors.

Level 9: Repeat the tasks 7 and 8 in the presence of a distracter.

Mental Speed/Information Processing

Letter Number Substitution: The task involves substituting designated numbers corresponding

to the letters of the English alphabet. Ask the subject to do the task as fast as he can without omitting any boxes vacant. The task is given at three difficulty levels. Increase in task difficulty is achieved by increasing the number of targeted symbols and numbers of letters to which corresponding symbols had to be inserted. Change the distracters in different phases in terms of different sense of modalities.

Level 1: The subject is required to substitute 6 numbers corresponding to 6 letters within three minutes time. Record the number of the symbols correctly substituted. Once, the subject is able to do that, move on to the next difficulty level.

Level 2: The subject is required to substitute 8 symbols corresponding to 8 letters within three minutes time. Record the number of the symbols correctly substituted.

Level 3: Repeat levels 1 and 2 in the presence of a distracter, say music. Record the number of the symbols correctly substituted.

Grain Sorting Task: The task involves sorting of mixed grains into piles of similar grains with the type of grains ranging from two to four. The quantity of grains sorted in 15 minutes is measured. The task is given at five difficulty levels:-

Level 1: The subject is required to sort two types of big grains. The quantity of grains sorted in 15 minutes is measured. Give the task for 1 week.

Level 2: The subject is required to sort two types of small grains in week 2. The quantity of grains sorted in 15 minutes is measured

Level 3: The subject is required to sort three types of small grains in week 3. The quantity of grains sorted in 15 minutes is measured.

Level 4: In the fourth week, the subject is made to listen to the music while sorting three types of small grains to increase task difficulty. The quantity of grains sorted in 30 minutes is measured

Level 5: The subject is required to sort four types of small grains in the weeks 5 and 6. During the weeks 5 and 6, the subject is made to listen to the music while sorting four types of small grains to increase task difficulty. The quantity of grains sorted in 30 minutes is measured.

Verbal Fluency

Word Generation: The task consists of generating as many as words as possible starting with a selected letter and words belonging to a category within a stipulated time. The task is given in one difficulty level. The task is given during the fourth week of cognitive retraining program. Letters and the category should be the ones other than used in the verbal fluency tests.

Response Inhibition: Stop- start is introduced in the week 5 and 6 of the cognitive retraining programme. In this task while the patient is performing a motor sequence for e.g. clapping hands, he is instructed to stop suddenly without a prior signal and then asked to restart the same sequence when the start signal is given. The start-stop signal is given randomly. The task is performed for 5 minutes in each therapy session,

Stroop Test: The Stroop test is given during week 5 and week 6. The task is divided into two sessions. The patient is asked to read out of the words instead of the colours. In the second phase, the patient is asked to name the colour instead of the words. The errors are noted.

Planning & Memory

Level 1: Help the patient to make a Memory Notebook. Things that could be included can be

1)an autobiographical background section to assist the patient with long-term, recent and short-term memory, 2) a section for daily schedules, 3) a section for medication schedules, 4) important telephone numbers and family/friends' names, etc. Make it part of the patient's routine that the notebook be referenced for information several times daily in response to specific questions, (for example, "What time are you supposed to do your exercise?" or "Can you find that information in your notebook?").

Level 2: A "daily activity" could be to check this Memory Notebook and mark off things that have been completed for that day or week. At this point the patient should be taking on more of the responsibility for keeping his notebook and daily schedule up to date and following it as appropriate. Give the patient more opportunity to use the notebook more independently by suggesting that he or she check the notebook to see if there is anything to be done or if they might have certain information handy.

Level 3: Maze Completion Task: Beginning with the starting point, patient had to trace the way out in the maze without tracing into a blind alley. The task is given at three difficulty levels. Increased task difficulty is achieved by the complexity of maze with increase in blind alleys and the route to reach the endpoint. The task can be given during weeks 6, 7 and 8.

Level 4: Planning Events: The patient is given real life situations and asked to narrate the steps taken to achieve the desired goal. For example, a situation such as planning for a picnic within a given budget is given. The therapist is required to suggest improvements to the plan. The task can be given during weeks 6 and 7.

Level 5: Short Essay Writing: The task involves writing a short essay on a given topic. The topics are related to activities that pertained to patient's interests and daily activities. The task is given at one level of difficulty. The task can be give at week 8.

Set Shifting

Categorization of beads: in week 3, 50 beads of various shapes, colours and sizes are combined together, the patient's task is to categorize the beads employing a different rule every day. The task is given during week 3.

Categorization of playing cards: the patient is required to sort two decks of playing cards. This task is given during weeks 4 and 5. In week 4, the patient has to identify the sorting rule of the therapist while in week 5, the patient himself has to generate a sorting rule.

Motor set-shifting: motor shifting task can be included during the weeks 4 and 5 for 5 minutes. The patient suddenly shifts from one motor sequence such as clapping hands to another sequence such as tapping feet.

Visuo-Constructive Ability

Dot based design construction: the task involves the construction of three target designs, geometrical designs, using the dots placed in each of the boxes. The task is given at a single difficulty level & is given during week 5.

Visuo-spatial reasoning

Tracing embedded figures: the task involves tracing a embedded simple figure hidden within a complex figure. Six simple figures are placed in two rows with three figures per row. beneath each figure, a complex figure is placed. This complex figure contains the simple figure. The task is given in at one level of difficulty. The task is given during week 6.

Abstraction

Proverb illustration: the task involves illustration of a given proverb. The patient is asked to write the meaning of the proverb with an example. The task is given in one level of difficulty. The task is given during week 7.

Similarity: The patient is presented with a pair of objects and asked to tell the similarities between the two objects. The patient is presented with simple to complex objects in the order of difficulty.

Problem Solving

Mental arithmetic problems: 10 mental arithmetic problems are given to the patient during weeks 1 and 2. The task is discontinued as the patient reaches the ceiling level.

Rearrangement of jumbled words: The task is to rearrange the letters to make a meaningful word. Words chosen for this task are simple nouns and words, having an average of four to six letters. The task is given at one difficulty level. The task can be given during weeks 3 and 4 of the cognitive remediation programme.

Rearrangement of jumbled sentences: 10 jumbled sentences are administered to the patient during weeks 5 and 6. Discontinue the task when the patient reaches the ceiling level.

Predicting the outcome of a desired situation: a problem situation is presented to the subject and asks the subject "what you will do if ------- happens?

Eg.: What will you do if electricity goes off suddenly?

Comprehension

Carrying out commands: two objects, a pen and a book were placed in front of the subject. commands of various levels of complexity, when required manipulation of these objects, is given

Sentence Repetition:

a phrase or sentence should be read out to the participant and an immediate repetition was expected. The target sentence should not be very long.

Expressive speech

Naming and describing things (cueing and semantic analysis): the subject is asked to name given objects (initially cues are provided). then he is asked to describe a thing or a picture.

Reading ability

Sight Word Approach: the subject is made to read aloud selected words from a paragraph written on the flash cards and after mastering made to read the paragraph. Record the number of errors committed and the time taken to complete the task.

Phonetics approach: reading aloud a list of words belonging to a particular phonetic family.

Verbal Memory

Temporal encoding: a list of 9 words is presented in a chunk of 3 words at a time and the patient is asked to recall. Teach the patient the strategy of semantic elaboration in the form of organizing the words and constructing a meaningful sentence for each set of words and visualizing the meaning of the sentence.The patient is asked to recall the words again in the same temporal order (immediate and delayed recall). Ensure the encoding in both verbal and visual modality. Present three separate lists of words in each session. After the patient is able to recall all the words in the list after 30minutes, the number of words can be increased to 12.

Visual Memory

Memory for location: tasks for improvement of visuospatial encoding and visual memory for locations require the patient to reproduce the location and sequence of an arrangement of objects on a table, from memory. An increasing number of objects are exposed for 10 seconds. Following maintenance in memory for another 10 seconds, the patient is asked to reproduce the arrangement. The task is given from weeks 2-6, with accuracy of reproduction as the score.

Memory for designs: The task involves drawing a given design from memory. Each abstract design contains five to eight components, which is exposed for 10 seconds. Patient is asked to observe the design carefully. After 10 seconds, the patient is asked to try to memorize the various components. In each session, there are 10 designs. The task is given at one level of difficulty. The correct number of designs forms the score. The task is given during weeks 7 and 8.

Psychotherapy sessions for the patients with psychosocial problems

Initial interview

- Assessment of current difficulties
- Goal definition
- Presentation of treatment rationale
- Beginning treatment

Subsequent therapy sessions:

- Setting the agenda
- Weekly items
- The day's major topic
- Homework assignments
- Feedback

INSRTUMENTS

The neuropsychology tests used to obtain relevant data were: MMSE, Addenbrooke's CognitiveExamination (ACE), subscales of ACE (registration, recall, orientation, language, phonemic fluency, categorical fluency, visuospatial, clock drawing test), Instrumental Activities of Daily living (IADL) and CDR.

STATISTICAL ANALYSIS

Pre and post intervention scores were statistically evaluated by using paired t-test.

RESULT

The value corresponding to neuropsychological test of pre and post cognitive retraining results at 3 months were compared. The mean age of cohort of MCI due to AD was 70 years (range 50-85), education range 10- 17 years. The results indicate that participants showed significant improvement in MMSE (p=0.001), ACE TOTAL (p= 0.028), ACE recall (p=0.000), ACE orientation (p=0.019), ACE language(p=0.027), ACE categorical fluency (p= 0.021), ACE visuospatial (p=0.009), clock drawing test (p= 0.035), CDR (p=0.001) and there is no significant difference in ACE registration, ACE phonemic fluency and IADL on followup analysis.

DISCUSSION AND CONCLUSION

The present study provided evidence that CR has the potential for short term objective stabilisation in cognitive functions in various domains and larger studies with longer periods of followup are warranted for proof of efficacy in improving quality of life in MCI due to AD . Specifically, present study's results shows that cognitive retraining had an improvement on MMSE, Clinical dementia rating scale, Addenbrooke's cognitive examination (ACE total score, orientation, recall, language, categorical fluency, clock drawing test and

visuospatial abilities). In this study patients showed significant improvement in MMSE , which is widely used to test cognitive functions. This includes the tests of orientation, attention/ concentration, memory, language and visuospatial abilities. ACE- Malayalam version, which is a more extensive screening instrument, also demonstrated significant differences post intervention. The ACE encompasses of five cognitive domains such as orientation, attention/concentration, memory, verbal fluency, language and visuospatial abilities. Patients showed significant improvement in ACE total score (p=0.028), orientation (p=0.019), recall (p=0.000), language (p=0.027), categorical fluency(p=0.021), clock drawing test(p=0.035) and overall visuospatial abilities(p=0.009). Our results also suggest significant improvement in Clinical dementia rating scale(p=0.001) which is used to find out six domains of cognitive and functional performance such as memory, orientation, judgment and problem solving, community affairs, home and hobbies and personal care.

CR is a pen and paper or computerized test/game that seeks to directly improve and/or restore cognitive functions such as attention, concentration, memory, organization, problem solving, decision making and executive skills. A study in CR and neuroplasticity proposed that stimulation of neuroplasticity and thereby enhancement of cognitive reserve is thought to represent the underlying neurobiological mechanism. (Park & Bischof, 2013; Raichlen& Alexander, 2017). Also another study in rodents indicates that training induced neuroplasticity entails neural changes such as hippocampal synaptic connectivity in animal models of neurodegenerative diseases. (Stuart et al., 2017) A study shows evidence that the aging brain also has the potential for neuronal reserve and that cognitive functions can be facilitated through cognitive training or engagement in demanding tasks that provide a sustained cognitive challenge. This study also suggested that older adults can greatly improve their functional ability through brain training. (Wollesen, B., Wildbredt, A., van Schooten, K. S., Lim, M. L., &Delbaere, K. (2020). As our CR intervention includes many repeated cognitive challenging tasks so which may improve their cognitive functions the effect of practice effects and learning could pave way for modulation via phenomena such as long term potentiation and long term depression which are proven neurobiological mechanisms of synaptic plasticity..

Burgers, Christian & Eden, Allison &Engelenburg, Mélisande&Buningh, Sander. (2015) in their study proposed that positive feedback boost intrinsic motivation through competence and autonomy needs. In our study it is clearly observed that immediate positive feedback plays a vital role in stimulating patient's motivation to complete and practice their cognitive tasks and which helps them to improve their cognitive functions. An efficient care giver is important in cognitive impaired patients' health care system. Proper training of caregivers resulted in a general improvement of the physical and psychological health of AD patients. (Reinhard SC, Given B, Petlick NH, et al.2008). Patients with supportive caregivers show significant observable improvements in cognitive functions.

More studies are required to ascertain the benefits of cognitive retraining on cognitive functions among elderly. One of the other important factors which we can consider in further studies is training for caregivers of cognitive impaired patients.

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Cognitive Rehabilitation of High Risk New Born Babies

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A newborn, regardless of gestational age or birthweight, who has a greater than average chance of morbidity or mortality because of conditions or circumstances superimposed on the normal course of events associated with birth and the adjustment to extra uterine existence.

Factors to Define A High Risk Infant

A) Demographic social factors:

Maternal age <16 or >40, unmarried, physical stress, low socioeconomic status

B) Past medical history:

Diabetes Mellitus, genetic disorders, hypertension

C) Previous pregnancy:

Intrauterine death, neonatal death, IUGR, IEMs, congenitalmal formations

D) Present Pregnancy:

Vaginal bleeding, PROM, multiple gestation, preeclampsia,abnormal USG findings

E)Labor: and delivery:

Obstructed labor, fetal distress, forceps delivery, meconium stained liquor

F)Neonate:

Birth weight <2000 or >4000,gestation <37 or >42SGA, Respiratory distress, congenital malformation

Low Birth Weight Infant:

Live born baby weighing 2500 gram or less at birth.(VLBW: <1500 gm, ELBW:<1000 gm). -

Preterm:

When the infant is born before term. i.e.: before 38 weeksof gestation.

Premature:

When the infant is born before 37 weeks of gestation.

Full term:

When the infant is born between 38 – 42 weeks ofgestation.

Post term:

When the infant is born after 42 weeks of gestation.

Following are high risk new born babies requiring cognitive rehab

1.Birth Asphyxia

Clinical correlates of adverse neuromotor outcome following birth asphyxia are:-

- Low birth wt and prematurity [periventricular leucomalacia]

- Apgar score of less than 4 at 10 mts or later
- Assissted ventilation for more than 24hrs
- Severe met acidosis
- Hypoglycemia
- Polycythemia
- Intractable seizures
- Severe HIE

2. Preterm Babies

Less than 37 completed week

3. Congenital Anomalies

4. Any Baby Requiring Ventilatory Support

5. Mothers Having Intrauterine Infection

6. Infants of Diabetic Mothers

Disorders encountered in this population are:-

• Hypoglycemia.

• Hypocalcemia.

• Hypomagnesemia.

• Cardio-respiratory disorders

• Hyperbilirubinemia (Unconjugated)

• Birth injuries

• Congenital malformations

7. Neonatal Sepsis

The newborn infant is uniquely susceptible to acquire infection, whether bacterial, viral or fungal.

• Bacterial sepsis and meningitis continue to be major causes of morbidity and mortality in the newborn.

• The mortality rate due to sepsis ranges from 20% to as high as 80% among neonate

Risk Factors for developing Neonatal Sepsis are:-

• Maternal risk factors:

- e.g.: Premature rupture of membrane.

• Neonatal risk factors:

- e.g.: Prematurity

8. Hyperbilirubinemia

Hyperbilirubinemia is an elevation in the neonatal serumbilirubin ≥ 12.9 mg/dl in Full-term, Formula feed infant OR ≥15 mg/dl in Preterm, Breast feed infant

• Characterized by Jaundice, which is defined as "yellowish discoloration of skin and mucous membranes".

9. Meconium Aspiration Syndrome (MAS)

This respiratory disorder is caused by meconium aspiration by the fetus in utero or by the newborn during labor and delivery.

10. Apnea

Definition:- Apnea is the cessation of respiration accompanied by bradycardia and/or cyanosis for more than 20 second.

Types of Apnea:-

- Pathological apnea:

Apnea within 24 hours of delivery is usually pathological inorigin.

- Physiological apnea:

Apnea developing after the first three days of life and not associated with other pathologies, may be classified as apnea of prematurity

Assessment of Motor Development

Alberta Infant Motor Scale [AIMS]

The Alberta Infant Motor Scale is a well know, norm referenced scale that evaluates the gross motor development of children from birth to 18 months. The scale was developed by Piper and Darrah in Alberta, Canada. The clinical application of AIMS is simple, the spontaneous movements of child are evaluated by observation, and the test can be completed in a short period of 20-30 mts.

Ehardt Developmental Prehension Assessment

Measures qualitative change in development of hand function and evaluates children from birth to 15 months.

Examples: Arms on approach (sitting) grasp of a cube, grasp of the pellet, release of a cube, release of a pellet, free writing pattern, crayon or pencil grasp.

Infant Neurological International Battery [INFANIB]

Infant neurological international battery is the developmental screening tool used for the assessment of neuromotor development in neonates aged 1-18months. It was established by Ellison et al. in 1985. It assesses neurological integrity from neonatal ICU through follow up till 18 months.

As first year of infant's life is critical period for brain development due to brain plasticity, thus helps in early identification and intervention of neuromotor abnormalities. Normal INFANIB can be used to reassure parents of risk infants. It can be used to compare the neuromotor assessment of children both in infancy and later variables (cerebral palsy, cognitive function, school performances).

Peabody Developmental Motor Scales

The Peabody developmental motor scale was first developed and published in 1983 by Rhonda Folio and Rebecca Fewell. It assesses child's gross& fine motor skills in relation to adaptive capacities, identify the areas of strength & weakness that facilitate programming. It evaluates 0-83 months old children. It takes 45-60 minutes for administration. The test was designed to identify developmental delay in children, assessing a child's motor competence relative to his/her peers, identifying delay or disorders in development, a useful tool to evaluate a child's progress.

Neonatal Oral Motor Assessment Scale

Sucking problems in infants can be identified by means of visual observation. During the first 2 minutes of regular feeding the infant's sucking skill is assessed, either immediately or on video, whether the infant is able to coordinate sucking and swallowing, whether the infant can maintain sucking, swallowing and breathing during the continuous phase and whether the infant is able to suck rhythmically with equally long bursts. We can differentiate tongue & jaw movements during both non-nutritive &nutritive sucking, can also identify normal &deviant oralmotor patterns. It takes about 10-15 minutes.

Assessment of Sensory Function

Test of Sensory Function in Infants [TSFI]

This will evaluate 14-18 month old babies. There are 24 item tests. It can be done in 20 minutes. 5 subtests are there:

1. Reacting to tactile deep pressure
2. Adaptive motor function
3. Visual tactile integration
4. Ocular motor control
5. Reactivity to vestibular stimulation

Intervention in NICU

Sensory Stimulation-Caregiving modifications like

Tactile stimulus-kangaroo mother care light stroke massage

Vestibular -swadle holding position elevation of head 30degree

Auditory-soft voices for communication avoid sudden loud noises

Visual-avoid abrupt bright lights,minimise visual stimulation, decrease NICU light system

Positioning in NICU

- Prone or side lying

- Swaddle or cover to keep in flexed position
- Attempt to nest the infant
- Promote midline alignment
- Head support

What to Avoid?

- Hyperextension of neck
- Frequent head turning to side
- Lower extremity frogging
- Bigger diaper

Sucking Training for Infant

Non-nutritive sucking or sucking on a pacifier may encourage the development of sucking behaviour and improve digestion of feeding and later you can encourage nutritive sucking, here there is high milk flow during this period of frequent swallowing.

Wearable Sensor Systems

Helps in monitoring body movements of neonates. It is possible to collect, store and process multimodal signal data of infant movements in a more efficient, comfortable and non intrusive way.

Passive Range of Motion Exercises of All Four Limbs

Gentle PROM exercises can be taught to the mother & can be started as early as feasible.

Rehabilitation Measures During Follow up Visits

The following can be done when the baby is brought for follow up in Physical Medicine department OP:

Proprioceptive neuromuscular facilitations

Herman Kabat,Margaret Knott&Dorothy Voss developed a system of movement facilitation techniques for the decrease of hypertonus, for strengthening, coordination and improving joint range. Movement patterns are spiral and diagonal with a synergy or chain of muscle groups.

Neurodevelopmental treatment

Was developed by Karl Bobath &Berta Bobath. The keystone of approach was to inhibit primitive reflexes, to facilitate automatic reactions & normal movement pattern. Bobath trained physiotherapists spent time on reducing the abnormal tone so that the child was prepared for movement. Preparation involves stretching, handling, positioning to improve quality of tone and therefore movement.Even though Bobath system has been challenged, the concept had not changed through the years but the techniques had been developed and refined.

Sensory stimulation for activation &inhibition

Margaret Rood a physiotherapist and occupational therapist, based her approach on many neurophysiological theories, techniques of stimulationsuch as stroking, brushing, icing, heating, pressure, slow &quick muscle stretch, joint retraction & approximation& muscle contractions are used to activate, facilitate or inhibit motor response.

Conductive education

Andras Peto in Budapest Hungary originated CE.The conductor is trained in habilitation of motor disabled children.The task are carefully analysed for group of children 15-20, anall-day program.Task are ADL, motor skills including hand function, balance & locomotion, rhythmic movement & activities in a group setting.

Sensory integration approach

This was devised by Jean Ayres{1979}.Stimulation of all the senses{ tactile, proprioceptive,vestibular visual& auditory} is recognised & often used for motor control & motor learning with functional goals.

NMES

This is used for muscle re-education, strengthening, biofeedback training such as gait pattern or wrist with hand function. The desirable action of a muscle within function is to provide sensory feedback.

Tread mill training

This is carried out with a body harness for body weight support so achild is partially unweighted stepping is promoted with out need for balance. It is useful for keeping children fit & motivated to exercise.

Horse riding

There are two types of therapy- therapeutic horseback riding and hippotherapy. Therapeutic riding is in a small group carried out by specially trained instructor. Hippotherapy is one to one carried out by a physiotherapist giving child various exercises on the horse. Hippotherapy promote postural control & symmetrical abduction of hip.

Swimming & Hydrotherapy

Exercises in water is hydrotherapy, which differs from swimming therapy .Both are carried out in groups for social stimulation &peer encouragement. Hydrotherapy focuses on strengthening, passive range of motion & stretching.Movements in water eliminate problems of posture control and balance and reduce risk of joint loading.

Kinesio taping

This is a developing technique used by Simsek et al & Kaya Kara et al at 2015that activates cutaneous input proprioception & sensory feed back. Tapes are polymer elastic, which can be stretched, and zinc oxide tape which are non elastic. Taping of upper limb and lower limb muscles can be done.

Rebound therapy

Rebound therapy involve the use of Trapoline to develop gross motor abilities and participate in a playful experience with friends. It is usually a 20 mts session once a week for 6 weeks.

MOVE (Mobility Opportunities via Education)

This is a mobility program using learning methods. The motor task are broken down into components. It uses prompts for guiding movement, which are decreased as the child achieves each component after repetition.

QUIZ

1. The main circuits that play a major role in neurocognitive &neurobehavioural syndromes are:-
   - (i)The dorsolateral prefrontal circuit
   - (ii)The lateral orbitofrontal circuit
   - (iii)The anterior cingulate circuit
   - (iv)The posterior cingulate circuit

   (a)i, ii
   (b) iv, i
   (c) ii, iii
   (d) i,ii,iii
   (e) i, ii, iii, iv

2. _____ memory requires the hippocampus for memory consolidation and utilizes the frontal cortex for memory retrieval.
   (a) Procedural
   (b) Implicit
   (c) Instinctual
   (d) Declarative

3. Working Memory & Executive Function are closely related in that both processes involve the _____?
   (a) Cerebellum
   (b) Thalamus
   (c)PreFrontal Cortex
   (d) Hippocampus

4. The most common Cognitive deficit seen in Multiple Sclerosis is….?
   (a) Slowed processing speed
   (b) Disinhibition
   (c) Verbal processing deficit
   (d) Spatial reasoning deficit

5. _____ deficits are one of the most common clinical manifestations of cognitive impairment in epilepsy, likely due to the high incidence of seizures originating in the medial temporal lobes.
   (a)Attention
   (b) Memory
   (c) Verbal processing
   (d) Spatial reasoning

6. Left Temporal Lobe Epilepsy results in _____ impairment.
   (a) Attention
   (b) Spatial reasoning
   (c) Verbal memory
   (d) Visual memory

7. The cognitive deficits caused by Anti Epileptic Drugs include...?
   (a) Memory
   (b) Executive functioning
   (c) Processing

(d) a& b
(e) a, b, c
(f) b& c

8. The most common autosomal dominantly inherited mutations causing FTD involve the MAPT or GRNgenes,both-on-chromosome____.
(a) 15
(b) 12
(c) 17
(d) 13

9. As with several other central nervous system (CNS) structures, the limbic lobe morphologically is a ____shaped structure.
(a) D
(b) C
(c) B
(d) E

10. The parahippocampalgyrus and hippocampal formation are considered part of the _____ lobe.
(a) Parietal
(b) Temporal
(c) Medial Frontal
(d) Occipital

11. In the presence of disease, orientation to ____ is impaired first and then orientation to _____
(a) Time, place
(b) Place, person
(c) Place, time
(d) Person, time

12. The first stage of memory—immediate or working memory—involves the circuits used to register, recall, and mentally manipulate transitory information. The _____ mediates much of this function.
(a) Medial temporal lobe
(b) Lateral temporal lobe
(c) Parietal lobe
(d) Occipital lobe
(e) Prefrontal cortex

13. _____memory includes generalized knowledge and factual information not related to memory of a specific thing or event, such as what a movie is.
(a) Working
(b) Episodic
(c) Declarative
(d) Semantic

14. _____ is the prototype cortical dementia where aphasia, apraxia, and agnosia are commonly present.
(a) FTD
(b) Vascular dementia
(c) Alzheimer's Dementia
(d) Pick's disease

15. *"This type of dementia typically causes recurrent visual hallucinations, fluctuations in attention and alertness, episodes of confusion, parkinsonian features, visuospatial impairments, and a particular sensitivity to neuroleptic medications. The pathology resembles that of PD dementia, and it is classified as an alpha-synucleinopathy. Symptoms characteristically fluctuate ("good days and bad days"). In one study, daytime drowsiness and lethargy, naps of 2 or more hours, staring into space for long periods, and episodes of disorganized speech occurred in 63% of patients with this dementia, compared with 12% of AD patients and 0.5% of normal elderly persons. Findings on Mental Status Examination are similar to AD, but patients with this dementia have relatively greater deficits in attention, executive function, visuospatial and constructional skills, psychomotor speed, and verbal fluency. Impaired visual perception may be involved in the visual hallucinations as well as the impaired visuospatial capability."* Which dementia are we talking about here?

(a)CJD
(b) Dementia with LewyBodies
(c) Vascular Dementia
(d) FTD

16. The single strongest risk factor for dementia is _____

(a) Pre-existing memory loss
(b) Stroke
(c) Increasing age
(d) Mild head trauma

17. Subtle cumulative decline in _____ memory is a natural part of aging.

(a) Episodic
(b) Semantic
(c) Procedural
(d) All of the above

18. Once the memory loss becomes noticeable to the patient and spouse and falls ____standard deviations below normal on standardized memory tests, the term Mild Cognitive Impairment is applied.

(a) 1
(b) 0.5
(c)1.5
(d) 0.75

19. Several genes play important pathogenic roles in at least some patients with AD. One is the APP gene on chromosome _____

(a) 21
(b) 13
(c) 12
(d) 10

20. A discovery of great importance has been that the Apo ε gene on chromosome ___ is involved in the pathogenesis of late-onset familial and sporadic forms of AD. Apo ε participates in cholesterol transport and has three alleles: ε2, ε3, and ε4.

(a) 12
(b) 20
(c) 13
(d) 19

21. ____bodies are argyrophilic, staining positively with the Bielschowsky silver method and also with immunostaining for tau.

(a) Lewy
(b) Classical Pick
(c) Ebstein
(d) Presenilin

22. A very important aspect of real-world function is ____ memory, which is the ability to remember to do something in the future.

(a) Prospective
(b) Explicit
(c) Retrospective
(d) Recent

23. The proposed meachanism of action of Amantadine is by..

(a) Altering Dopamine release in the Striatum
(b) Anti Cholinergic action
(c) Blocking NMDA receptors
(d) a & c
(e) a, b, &c

24. Memantine is a noncompetitive antagonist of the _____type Glutamate receptor.

(a) NMDA
(b) AMPA
(c) Kainate
(d) Kappa

25. Armodafinil, the R-enantiomer of modafinil (a mixture of R- and S-enantiomers) is also indicated for….?

(a) Narcolepsy
(b) To improve wakefulness in shift workers
(c) a& b
(d) To combat excessive sleepiness in Obstructive Sleep Apnoea patients
(e) a, b, & d

26. Explicit memory is heuristically divided into the sub processesof..?

(a) Acquisition of information
(b) Storage of information
(c) Retrieval of information
(d) All of the above
(e) a & b
(f) c& b

27. The Temporal pole is Brodmann's area ___?

(a) 34
(b) 38
(c) 36
(d) 37

28. The cytoarchitecture of the Temporal pole resembles that of the ____ cortex.
    (a) Calcarine
    (b) Entorhinal
    (c) Perirhinal
    (d) Striate

29. The superior dorsolateral prefrontal cortex is the center of an executive cognitive network that links the posterior parietal lobe and anterior cingulate gyrus. It is denoted by Brodmann'sareas ..?
    (a) 9
    (b) 10
    (c) 11
    (d) a& b
    (e) a& c

30. _____ syndrome is an involuntary and unconscious simulation of altered mental status (confusion or dementia) in a patient who is not malingering and believes in the validity of his or her symptoms.
    (a) Leriche
    (b) Kaplan
    (c) Leroy
    (c) Wernicke's
    (d) Todd's
    (e) Ganser's

31. In what cell types do Tau aggregates accumulate in Progressive Supranuclear Palsy?
    (a) Neurons
    (b) Astrocytes
    (c) Oligodendrocytes
    (d) a& b
    (e) a, b, & c

32. What are the manifestations of cerebral amyloid angiopathy (CAA) as it relates to dementia?
    (a) Lobar macrohemorrhages
    (b) Cortical microhemorrhages
    (c) Cortical superficial siderosis/focal convexity subarachnoid hemorrhages
    (d) a& b
    (e) b& c
    (f) a, b, & c

33.The first line drugs in mild or moderate Alzheimer's disease include all except?
    (a) Donepezil
    (b) Memantine
    (c) Rivastigmine
    (d) Galantamine

34. First line of treatment in Parkinson's disease Dementia is?
    (a) Rivastigmine
    (b) Amantadine
    (c) Pemoline
    (d) Donepezil

35. A patient has Severe Alzheimer's disease, his MMSE score is…?
    (a) 21-26
    (b) <10
    (c) 10-20
    (d) 20-24
36. All of the following are major parts of the motivational sub systems except
    (a) Hippocampus
    (b) Hypothalmus
    (c) Amygdala
    (d) Cerebellum
37. One of the following impairments affect cognition the most-
    (a) Spasticity
    (b) Motor deficit
    (c) Mood disturbance
    (d) Loss of vibration
38. One of the following areas are important in executive attention network
    (a) Globus pallidus
    (b) Nucleus ventralisposterolateralis
    (c) Anterior Cingulate gyrus
    (d) anterior part of internal capsule.

39. One of the following is not a step in cognitive rehabilitation
    (a) Process training
    (b) Strategy training
    (c) Functionalactivity training
    (d) Management training
40. Compensatory treatment in cognitive rehabilitation aims at
    (a) Improving cognition in a wide range of activities
    (b) Training specific problem areas
    (c) Improving communication
    (d) Is a group training technique
41. A complex mental function that refers to how an individual receives & begins to process internal and external stimuli is
    (a) Language
    (b) Concentration
    (c) Attention
    (d) Interaction
42. One of these is not cognitive impairment in Traumatic brain injury
    (a) Impaired attention
    (b) Easy distractibility
    (c) Slow verbal & visual information processing
    (d) Altered sleep wake cycles
43. Purdue peg board and finger tapping test is used to assess
    (a) Learning ability
    (b) Memory

(c) Executive function
(d) Psychomotor function.

44. One of the following is an internalised strategy in compensatory strategy training in memory
    (a) Notebooks
    (b) Mobile phones
    (c) Visual imagery
    (d) Memory logs
45. Donepezil is used to
    (a) Reduce spasticity
    (b) Improve language
    (c) Improve attention
    (d) Reduce hyperactivity
46. Spatial scanning(visuospatial) is mainly used to
    (a) Improve reading
    (b) Improve acuity of vision
    (c) Reducehemineglect
    (d) Reduce distraction
47. Ability to successfully engage in independent purposeful self serving behaviour is a ------ function
    (a) Attention
    (b) Communication
    (c) Executive
    (d) Memory
48. CHRP in Traumatic Brain injury rehabilitation is
    (a) comprehensive holistic rehab program
    (b) comprehensive higher rehab program
    (c) compensatory home based rehab program
    (d) compensatory holistic rehab program
49. CHRPs in traumatic Brain injury rehab emphasises
    (a) Motor improvement
    (b) Attention
    (c) Community reintegration
    (d) Improvement of consciousness
50. Metacognitive strategy training includes
    (a) Self monitoring and self regulation
    (b) Improvement of memory
    (c) Writing skills
    (d) Activities of specific recall

Cognitive Rehabilitation- An Occupational Therapy Perspective

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Cognition is the mental action or process of acquiring knowledge and understanding through thought, experience and the senses. It encompasses various aspects of high level intellectual functions and processes such as attention, memory, knowledge , decision making, planning reasoning, judgment, perception, comprehension, language and visuo-spatial function among others[(1)].

Occupational Therapy is one of the primary professions with knowledge and skills to address the consequences of cognitive decline [(1, 2, and 3)].

Cognitive decline can occur secondary to traumatic brain injury, non-traumatic neurological diseases, aging, chronic psychiatric diseases [(3, 4)].

Theoretically, visual perceptions and cognition are difficult to separate. Perceiving visual information involves cognitive processes such as attention and memory. However, clinically one can differentiate between visual perceptional dysfunction and cognitive dysfunction. For example, in visual perceptional dysfunction, the patient may have difficulty in task requiring attention, memory, organization or problem solving involving visual information. The patient may not demonstrate problems in auditory reception, attention or memory. In contrast, cognitive dysfunction is exemplified by difficulty in processing information regardless of the sensory modality. [(1, 4, 5, and 6)]

Cognition and Perception

The perceptual motor process is a chain of events through which the individual selects, integrates and interprets stimuli form the body and the surrounding environment. Cognition can be conceived as the method used by the central nervous system to process information [(3)].

Cognitive and Perceptual deficits

Attention disorders - Patient who is inattentive or distractable will have difficulty in processing and assimilating new information or techniques.

Memory disorders- Inability of the individual to store experiences and perception to recall at a later time.

Executive function disorders- Disturbances in volition, planning, purposive action and effective performance.

Body scheme or Body image disorders- Unilateral neglect, anosognosia, somatoagnosia, right-left discrimination and finger agnosia.

Spatial relation disorders- Figure-ground discrimination, form discrimination, spatial relations, position in space, topographic disorientation, depth and distance perception, vertical disorientation.

Agnosias - Visual objectagnosia, auditory agnosia, tactile agnosia.

Apraxia- Ideomotor apraxia, ideational apraxia, constructional apraxia.

Assessment

1. Mini mental status examination.
2. Montreal cognitive assessment.
3. Addenbrookes cognitive examination.
4. Bristol Activities of Daily Living.
5. Loewenstein Occupational Therapy Cognitive Assessment.

Approaches

1. Remedial or Restorative Approach - It consists of direct retraining or restoration of impaired core areas for cognitive skills. This method targets the underlying mechanism of memory, attention and executive function [3, 5, and 6].

2. Adaptive or Compensatory Approach- It targets the task and functions a person needs or wants to perform. It directly teaches and trains a person to accomplish the task despite the cognitive disability with the strategy being an adaptation or compensation. Compensation refers to the changes that need to be made in the patient's approach to a task.

   A few general suggestions when teaching compensatory techniques are

   a) Use simple directions
   b) Establish and carry out a routine
   c) Do each activity in a consistent manner
   d) Employ repetition as much as necessary

   Adaptation refers to the alterations that need to be made in the human and physical environment in order to accomplish the task. For example, if the patient cannot differentiate between right and left or tends to neglect left side of the body, a piece of red tape on the left shoe during gait training will allow the patient to attend more easily to the left side and thus to follow the therapist's instruction more accurately [3,6].

3. Specific Task Training Approach- In this approach, the therapist attempts to circumvent the cognitive deficit that hampers performance by teaching an actual functional task. The intervention is designed to help the individual achieve the occupational performance goal by learning a routine so that the cognitive deficits no longer interfere with the occupational performance. By addressing the basic skill training, clients may be able to improve self-awareness, mental efficiency and organization resulting in continued cognitive improvement[3, 7].

4. Cognitive Retraining embedded in Functional activity – In Cognitive Retraining, cognitive processes are

addressed within the context of the activity (Example: Attention retraining during driving reeducation); the retraining is "context specific". Studies suggests that performance on a particular task after training will improve to the extent that processing operations required to carry out that task overlap with the processes engaged during the training. (Example: Problem solving strategies developed in the context of a simple front closing shut donning activity will carry over to the front closing jacket donning activity when that process is engaged)[7]

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Cognitive Rehabilitation: Recent Developments in Neuropsychology

Jwala Narayanan is a Consultant Neuropsychologist at Manipal Hospital and the Annasawmy Mudaliar General Hospital. Her clinical work involves working with people across the life span suffering with a neurological condition (e.g. stroke, brain injury, epilepsy, infections, dementia, etc.). Her research interests are in memory, bilingualism and neuropsychological rehabilitation. Most of her research has been in the area of dementia. She has publications in peer reviewed journals. She runs an enrichment program for healthy ageing and people with dementia. She recently received a grant from the Burning Man US to spread awareness on managing dementia and engaging with community to get involved in the cause.

Neuropsychological Rehabilitation (NR) is an interdisciplinary approach to improve cognition, emotion and psychosocial functioning for a person with brain injury. NR is a goal oriented method of rehabilitation, where the goals are set by patients, their family and the therapists. The rehabilitation program is usually tailored to the needs of the individual in their given context. The aim of rehabilitation is to help improve independence in daily living and quality of life. Cognitive Rehabilitation (CR) is one of the core components of neuropsychological

rehabilitation. CR is an intervention to help ameliorate difficulty in everyday life

subsequent to cognitive impairment. It is important to integrate evidence-based recommendations into clinical practice as the field is fast progressing.

As in other fields of rehabilitation, goal setting is an important aspect of the rehabilitation process. The goals are set in collaboration with patients, families and therapists based on recognised difficulties. An assessment of premorbid levels of occupational, leisure and community based activities would help set goals and plan activities. The process of goal setting involves short and long term goals making the process of rehabilitation more tangible for the patient. An important aspect of the goal setting process is to gently bring awareness of the deficits in a safe space to reduce embarrassment and poor self-worth. Awareness levels can be varied in patients and appropriate measures need to be taken to address the deficits.

CR involves a process based approach that focuses on various cognitive domains including attention, memory, executive function, language, visuospatial abilities and social cognition. While attempts are made to restore

cognitive abilities, this may not always be possible. When cognitive deficits are not suitable for retraining, patients can be taught strategies to cope with cognitive deficits. These strategies may be practiced to promote automaticity for easy access when required in daily living. In patients where retraining or strategies may not be successful, compensatory tools may be substituted. When a patient is no longer able to engage in activities they used to, due to the cognitive impairment, new ways must be identified to substitute the need (e.g. audiobooks, for someone who is unable to read due to visuospatial difficulty).The ulterior goal of CR is the generalizability of the skills learnt in rehabilitation and its impact in everyday life. Cicerone KD., et al. (2019) reported evidence based cognitive rehabilitation guidelines for clinical practice based on systematic reviews.

In a nutshell, following are some practice standards outlined by Cicerone KD. et al. Severity of cognitive impairment will also determine use of appropriate treatment options.

- Attention & Working memory: there is little evidence for direct retraining of cognitive abilities except for attention and working memory. These have been advised to be coupled with metacognitive strategies to promote generalisation to daily function.
- Visuospatial ability: Visual scanning for unilateral neglect and specific gestural training is recommended for apraxia.
- Memory: Memory strategy training using internalised strategies (e.g. visual imagery) and external compensations (e.g. using diaries and reminders) are recommended.
- Communication & social cognition: Cognitive-linguistic therapies are recommended for language deficits and specific interventions for functional communication deficits including pragmatic conversational skills and recognition of emotions from facial expressions are recommended.
- Executive functions: metacognitive strategies that include self-monitoring and self-regulation have been recommended including self-regulation of emotions, problem solving and goal management.

Assistive technology have been explored widely in aiding rehabilitation. Voice assistants including Google Assist or Alexa have been found to be helpful as external compensatory aids. They tend to encourage patients to be more independent by helping with schedules, retrieving information, communication, shopping, leisure activities (playing games/puzzles) and also controlling the environment (turning on television or lights). Exergames like the Microsoft Wii and internet based applications for neurorehab have also been helpful in engaging patients in rehabilitation in more enjoyable ways. Research using virtual reality (VR) for cognitive rehabilitation has also seen benefits in training attention, some skills in executive function, everyday skills (driving, cooking) and coping with PTSD.

In summary, it is important to not lose sight of the patients' goals and generalizability of the rehabilitation program. As therapists it may be helpful to approach rehabilitation creatively, encouraging more meaningful activities rather than just exercise based activities. Cullen et al. (2016) also propose focusing on well-being, encouraging gratitude and kindness which may have an overall positive outcome. Interacting with other rehabilitation therapists and working in an interdisciplinary team has many benefits, this may be optimised where possible.

KEY TO THE QUIZ

1. (d)

   Prefrontal lobes & their connections play *a major part* in *neurocognitive & neurobehavioural syndromes*. *Three main circuits have been identified* which appear to play a major role in these higher functions. *These circuits are structurally & functionally distinct* & *all traverse the frontal lobe, striatum, globus pallidus & thalamus.*

   1. *The dorsolateral prefrontal circuit* links Brodmann's areas 9 & 10 with the dorsolateral head of the caudate. This may be active in executive functions including motor planning.
   2. *The lateral orbitofrontal circuit* originates in the inferolateral prefrontal cortex & projects to the ventromedial caudate. Lesions have been associated with personality changes like disinhibition, or irritability, or obsessive compulsive disorder (OCD).
   3. *Lastly, the anterior cingulate (limbic) circuit* arises in the anterior cingulate gyrus & projects to the ventral striatum, which receives input from the amygdala, hippocampus, & perirhinal & entorhinal cortex. Damage has been associated with apathy, mutism, & OCD.

   All the symptoms of prefrontal lesions can also occur with lesions of *these networks in any location*. *For example, similar symptoms may occur with thalamic or caudate lesions*. It may be noted that in general, medial structures like the medial prefrontal areas are more concerned with the 'self' & internal conditions while lateral structures like the lateral prefrontal areas & dorsolateral cortex are more concerned with external conditions.

2. (d)

   Declarative memory requires the *hippocampus for memory consolidation & utilizes the frontal cortex for memory retrieval.*

   Generally defined, *memory refers* to the *ability of the brain to store and retrieve information*, the necessary *prerequisite for all learning*. Clinical neurologists have historically divided memory into *three temporal stages*. These stages can be helpful when conceptualizing diagnosis and difficulties in independent living and have a general correspondence to the stages and concepts of memory proposed by cognitive neuroscientists.

   The first stage, referred to as immediate memory by clinicians, corresponds to Baddeley's concept of working memory. *Immediate or working memory* refers to *the system that actively holds pieces of transitory information in conscious awareness, where it can be subsequently manipulated or used to perform a task.* There has been recent debate over the true capacity of working memory, but *the general consensus* is that *the normal adult human being can retain 5 to 9 meaningful items in working memory. This information can generally reside in conscious awareness indefinitely with attention and rehearsal*. However, *without rehearsal*, this information is *lost in approximately 18 to 20 seconds. As an example, most people can hear or see a telephone number, walk across the room, and dial the number without difficulty. Once the number is dialed and conversation is started, the number fades from working memory.*

The second stage of memory, referred to by clinicians as short-term or recent memory, involves the *ability to encode and retrieve specific items, such as words or events, after a delay of minutes or hours.* In clinical parlance, *short-term memory is synonymous* with *recent episodic memory*. *Short-term or recent episodic memory requires the function of the hippocampus and parahippocampal areas of the medial temporal lobe for both encoding and storage. The amygdala, a structure adjacent to the medial temporal cortex, is not essential for episodic memory but seems crucial for the encoding of emotional or social contexts of specific events. In contrast, the retrieval of recent episodic memories tends to rely on a delicate interaction between prefrontal regions and medial temporal regions. A simple analogy for remembering the anatomical organization of recent episodic memory is useful. The frontal lobes are considered the "filing clerk" of the memory system, deciding what memories to retrieve and from where to retrieve them. The medial temporal lobes are the "recent memory filing cabinet," where recent memories are stored. Patients with medial temporal lobe damage (e.g., Alzheimer disease) have a damaged file cabinet, in which memories are unable to be stored. In contrast, patients with frontal lobe damage (e.g., stroke, tumor) have difficulty in properly organizing the files in the cabinet or difficulty locating them during retrieval.*

The third stage, referred to as remote or long-term episodic memory, refers to *the ability* to *retrieve specific items, such as words or events, after a delay of weeks, months, or years*. An *example* of this would be asking the patient about *the last movie they have seen* or *what they did on their last birthday*. Retrieval of remote episodic memories tends to require less hippocampal and medial temporal lobe involvement. Consolidation of long-term memories can occur at the synaptic and systems levels. Synaptically, consolidation occurs through long-term potentiation and protein synthesis in the hippocampus during the first few hours of learning.

Semantic memory is referred *to as factual knowledge that includes memory of meanings, understandings, and other concept-based knowledge as well as general knowledge about the world*. Recall of famous figures or events, such as presidents or wars, and knowledge of semantic information, such as the definitions of words and the differences between words, are examples of semantic memory. Semantic memory differs from personal long-term memory in that the subject can continuously replenish such knowledge by reading and conversation. *Semantic memory is thought to reside in multiple cortical regions such as the visual association cortex for visual memories and the temporal cortex for auditory . memories*. This concept of *multiple localizations of semantic memory* is *supported by functional brain imaging research*.

3. (c)
4. (a)

Slowed processing speed is the *most common Cognitive deficit seen in Multiple Sclerosis*. In MS, delays in processing speed are connected to learning impairments. Cognitive dysfunction can include memory loss, impaired attention, difficulties in executive functioning, memory, problem solving, slowed information processing, and problems shifting between cognitive tasks. Euphoria (elevated mood) was once thought to be

characteristic of MS but is actually uncommon, occurring in < 20% of the patients. Cognitive dysfunction sufficient to impair the activities of daily living is rare in MS.

5. (b)
   Memory deficits are one of the most common clinical manifestations of cognitive impairment in epilepsy, likely due to *the high incidence of seizures originating in the medial temporal lobes*. There is general support for *left temporal lobe epilepsy* (TLE) resulting in *verbal memory impairment*, but there are mixed findings regarding right TLE specifically resulting in visual memory impairments. In addition to memory, general intellectual functioning can be impaired. More specifically, executive functions and visuoconstructional abilities can be mildly impaired in both left and right TLE compared to healthy individuals, with earlier age of onset being associated with worse deficits, but right TLE was specifically impaired for psychomotor speed and higher level cognitive estimation abilities as well if the focus of seizure activity is in the language dominant hemisphere. Postictal language recovery is also affected by seizure origin and generalization in frontal lobe epilepsy patients.
6. (c)
   Refer to the explanation of Question 5
7. (e)
   A complicating issue in identifying cognitive and behavioral deficits associated with epilepsy is the independent effect antiepileptic drugs (AEDs) commonly have on neuropsychological functioning. *Memory, executive functioning, and processing* are the *most frequently affected by both classic and new AEDs*, with *older drugs such as T opiramate having worse cognitive side effects than newer medications*. The varying effects these medications have on cognition must be weighed against the benefits of minimizing the frequency and severity of seizures. *An emerging area* where neuropsychology is significantly contributing is *the diagnosis and treatment* of *psychogenic nonepileptic seizure disorder (PNES). PNES is a conversion disorder* in which the patient has the *behavioral manifestations of a seizure without any accompanying epileptiform activity*. The *typical PNES case is a young adult female* with *lower intellectual functioning, a history of trauma, and possibly another psychogenic disorder that tends to experience psychogenic seizures when under stress*. At least 10% of these patients also have EEG diagnosed seizure disorder. While the *gold standard for diagnosing PNES is video electroencephalography*, this is not always available and neuropsychological testing is recommended as part of a staged comprehensive diagnostic process for PNES.
8. (c)
   Frontotemporal dementia (FTD) often begins in the fifth to seventh decades, and *in this age group* it is *nearly as prevalent as Alzheimer's dementia(AD)*. Early studies suggested that FTD may be more common in men than women, although more recent reports cast some doubt on this finding. Unlike in AD, behavioral symptoms predominate in the early stages of FTD. *Although a family history of dementia is common, autosomal dominant inheritance is seen in about 10% of all FTD cases. The clinical heterogeneity in familial and sporadic forms of FTD is remarkable, with patients demonstrating variable mixtures of behavioral, language, movement, and motor neuron symptoms. The most common autosomal dominantly inherited mutations causing FTD involve the MAPT or GRN genes, both on chromosome 17*. MAPT mutations lead to a change in the alternate splicing of tau or cause

loss of function in the tau molecule. With GRN, mutations in the coding sequence of the gene encoding progranulin protein result in mRNA degradation due to nonsense-mediated decay.

9. (b)

The limbic lobe is sometimes considered a separate lobe of the brain, more because of its function than its anatomy. *Components of the limbic lobe* include the following: the *hippocampus*, which lies deep in the medial temporal lobe and becomes continuous with the fornix; *the mammillary bodies* (part of the hypothalamus); *the anterior nucleus of the thalamus*; *the cingulate gyrus*; and *the parahippocampal gyrus. As with several other central nervous system (CNS) structures, the limbic lobe morphologically is a C-shaped structure*. It begins anteriorly and superiorly in the paraterminal gyrus and subcallosal area beneath the rostrum of the corpus callosum. The body of the C is formed by the cingulate gyrus, which merges at the isthmus of the cingulate into the parahippocampal (hippocampal) gyrus. The end of the C is the hippocampal formation. The cingulate gyrus lies just above the corpus callosum. The parahippocampal gyrus begins at the isthmus of the cingulate and runs to the temporal tip, lying between the collateral sulcus and the hippocampus; it curls around the hippocampal fissure to form the uncus. *The hippocampal formation is composed of the hippocampus proper (Ammon's horn), the dentate gyrus, and the subiculum*. When not regarded as part of the limbic lobe, the anterior and posterior parts of the cingulate gyrus are considered parts of the frontal and parietal lobes, respectively. The parahippocampal gyrus and hippocampal formation are considered part of the temporal lobe. *The structures of the limbic lobe are connected in Papez circuit (cingulate gyrus → parahippocampal gyrus → hippocampus → fornix → mammillary body → anterior nucleus of the thalamus → cingulate gyrus).*

The other C- shaped structures in the CNS….

- *The Caudate nucleus* is a long, C-shaped structure with bulbous ends.
- *The language areas* form a C-shaped mass of tissue around the lips of the Sylvian fissure extending from Broca's area to Wernicke's area.

10. (b)

Refer to key for Question 9

11. (a)

The *formal mental status examination usually begins* with an *assessment of orientation. Normally, patients are said to be "oriented times three" if they know who they are, their location, and the date. Some examiners assess insight or the awareness of the situation as a fourth dimension of orientation*. The details of orientation are sometimes telling. The patient may know the day of the week but not the year. Orientation can be explored further when necessary by increasing or decreasing the difficulty level of the questions. Patients may know the season of the year if not the exact month; conversely, they may be oriented well enough to know their exact location down to the street address. *Most patients can estimate the time within 1 hour. Orientation questions can be used as a memory test for patients who are disoriented. If the patient is disoriented, she may be told*
the day, the month, the year, the city, etc., and implored to try to remember the information. Failure to remember this information by a patient who is attentive and has registered it suggests a severe memory deficit. Occasionally, patients cannot remember very basic

information, such as the year or the city, despite being repeatedly told, for more than a few seconds. *In the presence of disease, orientation to time is impaired first and then orientation to place; only rarely is there disorientation to person.*

12. (e)
    The prefrontal cortex mediates much of this function. This is a short-lived operation in which material is not actually committed to memory.
13. (d)
14. (c)
    Typical Alzheimer's disease (AD) dementia initially presents with an episodic memory impairment reflecting the selective vulnerability of the medial temporal lobe to AD pathology. Episodic memory relates to our ability to remember information specific to a time and place when that memory was formed (e.g., What did you eat for dinner? What did you do on a trip?). Recent episodic memory is particularly impaired in early AD.

    While episodic memory is the hallmark of early AD, subsequent cognitive decline is heterogeneous as the pathology spreads to the association cortices. While individual presentations vary significantly, *Feldman and Woodward have described the typical symptom progression in AD dementia*: *Mild AD (recent memory impairment, repetitive questions, loss of interest in hobbies, anomia, impaired instrumental ADLs), moderate AD (aphasia, executive dysfunction, impaired basic ADLs), severe AD (agitation, complete loss of independence, sleep disturbance).*
15. (b)
    The parkinsonian dementia syndromes are under increasing study, with many cases unified by Lewy body and Lewy neurite pathology that ascends from the low brainstem up through the substantia nigra, limbic system, and cortex. *The DLB clinical syndrome is characterized by visual hallucinations, parkinsonism, fluctuating alertness, falls, and often RBD [**Rapid eye movement (REM) sleep behavior disorder]** . *Dementia can precede or follow the appearance of parkinsonism.* Hence, one pathway occurs in patients with long-standing PD without cognitive impairment, who slowly develop a dementia that is associated with visual hallucinations and fluctuating alertness. *When this occurs after an established diagnosis of PD, many use the term Parkinson's disease dementia (PDD). In others, the dementia and neuropsychiatric syndrome precede the parkinsonism, and this constellation is referred to as DLB. Both PDD and DLB may be accompanied or preceded by symptoms referable to brainstem pathology below the substantia nigra, and many researchers conceptualize these disorders as points on a spectrum of α-synuclein pathology.* Patients with PDD and DLB are highly sensitive to metabolic perturbations, and in some patients the first manifestation of illness is a delirium, often precipitated by an infection, new medicine, or other systemic disturbance. A hallucinatory delirium induced by L-dopa, prescribed for parkinsonian symptoms attributed to PD may likewise provide the initial clue to a PDD diagnosis. Conversely, patients with mild cognitive deficits and hallucinations may receive typical or atypical antipsychotic medications, which induce profound parkinsonism at low doses due to a subclinical DLB-related nigral dopaminergic neuron loss. *Even without an underlying precipitant, fluctuations can be marked in DLB, with episodic confusion or even stupor admixed with lucid intervals.* However, despite the fluctuating pattern, the clinical features persist over a long period, unlike delirium, which resolves following correction of the

inciting factor. Cognitively, DLB features relative preservation of memory but more severe visuospatial and executive deficits than patients with early AD.

16. (c)
    Dementia can result from numerous causes including brain injury (cerebrovascular disease or trauma) or infectious and metabolic diseases, but *the most common causes are neurodegenerative diseases.*
17. (a)
    Subtle cumulative decline in episodic memory is a natural part of aging. This frustrating experience, often the source of jokes and humor, is *referred to as benign forgetfulness of the elderly.* Benign means that it is not so progressive or serious that it impairs reasonably successful and productive daily functioning, although the distinction between benign and more significant memory loss can be difficult to make. *At age 85, the average person is able to learn and recall approximately one-half the number of items (e.g., words on a list) that he or she could at age 18.*
18. (c)
    A measurable cognitive problem that does not disrupt daily activities is often referred to as mild cognitive impairment (MCI). In the early stages of the disease, the memory loss may go unrecognized or be ascribed to benign forgetfulness. Once the memory loss becomes noticeable to the patient and spouse and falls 1.5 standard deviations below normal on standardized memory tests, the term MCI is applied. *This construct provides useful prognostic information, because approximately 50% of patients with MCI (roughly 12% per year) will progress to AD over 4 years.*
19. (a)
    Adults with trisomy 21 (Down syndrome) consistently develop the typical neuropathologic hallmarks of AD if they survive beyond age 40. Many develop a progressive dementia superimposed on their baseline mental retardation. APP is a membrane-spanning protein that is subsequently processed into smaller units, including Aβ, which is deposited in neuritic plaques. Aβ peptide results from cleavage of APP by β and γ secretases.
    Investigation of large families with multigenerational FAD led to the discovery of two additional AD genes, the presenilins. Presenilin-1 (PS-1) is on chromosome 14 and encodes a protein called S182. Mutations in this gene cause an early-onset AD (onset before age 60 and often before age 50) transmitted in an autosomal dominant, highly penetrant fashion. More than 100 different mutations have been found in the PS1 gene in families from a wide range of ethnic backgrounds. Presenilin-2 (PS-2) is on chromosome 1 and encodes a protein called STM2.
20. (d)
    The Apo ε4 allele confers increased risk of AD in the general population, including sporadic and late age-of-onset familial forms. Approximately 24–30% of the nondemented white population has at least one ε4 allele (12–15% allele frequency), and about 2% are ε4/4 homozygotes. Among patients with AD, 40–65% have at least one ε4 allele, a highly significant difference compared with controls. Conversely, many AD patients have no ε4 allele, and ε4 carriers may never develop AD. Therefore, ε4 is neither necessary nor sufficient to cause AD.
21. (b)

Frontotemporal lobar degeneration (FTLD) is a category of dementing illness with different clinical subtypes: behavioral-type FTLD and primary progressive aphasia (PPA). *Within the PPA type, three subtypes are distinguished: nonfluent/agrammatic, semantic, and logopenic. Formerly, Pick disease was a term used for the clinical syndromes now encompassed by FTLD, but it is currently used to refer to only one of the several types of pathology that cause these conditions. FTLD subtypes, just like AD, cannot be diagnosed with structural neuroimaging, but CT and MRI show changes that can assist in differentiating them from each other and other dementias, as well as excluding other etiologies. A characteristic finding in these dementias is atrophy of the frontal and temporal lobes, which may be asymmetrical or symmetrical. When the disease is advanced, gyral atrophy may be so prominent that it has been termed knife-edge atrophy.*

22. (a)
    Prospective memory (capacity for remembering to remember) & future episodic memory (creation of scenarios requiring drawing upon past experiences to guide what might happen in the future) are subtypes of declarative memory.
23. (e)
24. (a)

The amino acid glutamate is the main excitatory neurotransmitter in the brain and spinal cord and may be responsible for 75% of the excitatory transmission in the CNS. Glutamate acts on both ionotropic and metabotropic receptors in the CNS. There are three subtypes of ionotropic glutamate receptors, each named for its relatively specific agonist. These are the AMPA (α-amino-3-hydroxy-5- methylisoxazole-4-propionate), kainate, and NMDA receptors.

Activation of the metabotropic glutamate receptors (mGluR) leads to either an increase in intracellular inositol 1,4,5-triphosphate (IP3) and diacylglycerol (DAG) levels or a decrease in intracellular cAMP levels. There are eight known subtypes of mGluR located at either presynaptic (mGluR2-4,6-8) or postsynaptic sites (mGluR1,5) and are widely distributed in the brain.

Memantine is an N-methyl-D-aspartate (NMDA) receptor antagonist. It also blocks the 5-hydroxytryptamine-3 receptor. Memantine was approved by the FDA for the treatment of *moderate to severe AD and can be used in conjunction with AChEIs or on its own.* Side effects are rare although confusion and dizziness have been reported.

Dosage:- Orally administered, *initially at a dose of 5 mg once daily*, then increased in steps of 5mg every week; usual maintenance dose is 20mg daily.

25. (e)
    Armodafinil is the longer-acting isomer of modafinil (R-(-)-modafinil). Start with 75mg/day and increase if needed to150 mg/day (maximum 250 mg/day). Half-life is approximately 15 hours, reduces efficacy of steroidal oral contraceptives, can cause serious rashes and allergic reactions.
26. (d)
27. (b)
    The cytoarchitecture of the temporal pole (Brodmann's area 38) resembles that of the perirhinal cortex (areas 35 and 36).

The portions of the frontal lobe anterior to area 6, area 8, and the motor speech centers are areas referred to as the prefrontal cortex. It includes areas 9 to 12, 32, 45, 47, and others. These areas are connected with the somesthetic, visual, auditory, and other cortical areas by long association bundles and with the thalamus and the hypothalamus by projection fibers. *The prefrontal cortex is the main projection site for the dorsomedial nucleus of the thalamus. The prefrontal cortex projects to the basal ganglia and substantia nigra; it receives dopaminergic fibers that are part of the mesocortical projection from the midbrain. The dopaminergic neurons are associated with reward, attention, short-term memory tasks, planning, and drive. Clinically, the prefrontal region can be divided into the dorsolateral prefrontal cortex (DLPFC), the medial prefrontal cortex (MPC), and the orbitofrontal cortex (OFC). The cellular structure of the prefrontal region is strikingly different from areas 4 and 6 (the motor and premotor areas). The cortex is thin and granular; the pyramidal cells in layer 5 are reduced in both size and number.*
The motor areas of the frontal lobe include the primary motor cortex (area 4) as well as the premotor and SMAs. The SMA consists of areas of cortex lying on the medial aspect of the hemisphere just anterior to the primary motor cortex at the posterior medial aspect of the frontal lobe (area 6).

The primary sensory (somesthetic) cortex (S1; areas 3, 1, and 2) occupies all but the lowest part of the postcentral gyrus, continuing onto the medial surface into the adjoining part of the paracentral lobule. Recent work suggests that the designation primary sensory cortex should be restricted to area 3. The secondary somatosensory cortex (S2) lies in the parietal operculum adjacent to the lower portion of S1 near the sylvian fissure. In the depth of the central sulcus, area 3 abuts area 4. The postcentral cortex is homotypical (granular) cortex with six well-developed layers. The interparietal sulcus extends posteriorly from the midpoint of the postcentral gyrus and divides the remainder of the parietal lobe into the superior parietal lobule above and the inferior parietal lobule below. *Area 5a—the preparietal area, in the upper part of the parietal lobe just posterior to area 2— contains large, deep pyramidal cells, some as large as the smaller Betz cells in area 4. Area 5b, the superior parietal area, occupies a large part of the superior parietal lobule, extending over the medial surface of the hemisphere to include the precuneus. Area 7, the inferior parietal area, constitutes the major portion of the parietal lobule; it includes the supramarginal and angular gyri and receives many afferents from the occipital lobe. S1 receives enormous projections from the ventral posterolateral and ventral posteromedial nuclei of the thalamus.*
Occipital lobe is more nearly a structural and functional entity than the other cerebral lobes; all of its functions are concerned either directly or indirectly with vision. It is composed of Brodmann's areas 17, 18, and 19. The primary visual cortex (area 17) is located on the lips of the calcarine fissure and adjacent portions of the cuneus above and the lingual gyrus below. The cortex is granular in type and extremely thin. Layer 4 is relatively thick with a prominent outer band of Baillarger (line or band of Gennari), which is visible grossly and gives the area its designation of striate cortex.

The temporal lobe includes the superior, middle and inferior temporal, lateral occipitotemporal, fusiform, lingual, parahippocampal, and hippocampal gyri. Heschl's gyri

and the planum temporale lie on the superior surface. The superior temporal gyrus subserves auditory and language functions. The middle and inferior gyri receive abundant projections from the occipital lobe and serve to integrate vision with temporal lobe auditory and language functions. The hippocampal formation is a center for learning and memory. There are abundant connections between the temporal lobe and the limbic system.
The auditory radiations run from the medial geniculate body to the auditory cortex (areas 41 and 42) in the superior temporal gyrus. Area 41 is composed of granular cortex similar to that in the parietal and occipital regions; area 42 has large pyramidal cells in layer 3. Hearing is bilaterally represented in the temporal lobes, although there is a contralateral predominance. Nearby areas in the superior temporal gyrus allow for the differentiation and the interpretation of sounds. The superior temporal gyrus may also receive vestibular impulses.

Layers of the cortex…
Most of the cortical mantle has six identifiable layers; some areas of the brain have less. ix-layered cortex is referred to as neocortex, isocortex, or heterogenetic cortex. *The six layers, from superficial to deep, are as follows (can be numbered as 1,2,3,4,5,6): (I) molecular (plexiform), (II) external granular, (III) external pyramidal, (IV) internal granular, (V) internal pyramidal (ganglion), and (VI) multiform.*

28. (c) Refer to key for Question 27
29. (d)
    The dorsolateral prefrontal cortex (DLPFC)-subcortical circuit is principally involved in attentional and higher order cognitive executive functions. Executive functions include the ability to shift sets, organize, and problem solve, as well as the abilities of cognitive control and working memory. Shifting sets is related to mental flexibility and consists of the ability to move between different concepts or motor plans, or the ability to shift between different aspects of the same or related concept. Working memory is the online maintenance and manipulation of information. *The DLPFC–subcortical circuit includes the dorsolateral head of the caudate and the lateral mediodorsal globus pallidus interna and the parvocellular aspects of the mediodorsal and ventral anterior thalamic nuclei.* Dysfunction in this circuit has been linked to environmental dependency syndromes (including utilization and imitation behavior), poor organization and planning, mental inflexibility, and working memory deficits. *Executive dysfunction is also a principal component of subcortical dementias. Deficits identified in subcortical dementias include slowed information processing, memory retrieval deficits, mood and behavioral changes, gait disturbance, dysarthria, and other motor impairments. Vascular dementias, Parkinson's Disease, and Huntington's Disease are a few examples of conditions that affect this circuit.*
30. (e)
    In the assessment of patients with functional cognitive symptoms, approximate answers to questions (e.g., "How many legs has a horse got?" Answer: "3") are classically described. This has been called Ganser syndrome after the nineteenth-century German psychiatrist who first reported it. In our experience, this is rare and is a marker of factitious symptoms. More common is the early loss of relatively "protected" knowledge such as the names of spouses or children, discrepancies between real-life function and test results, or discrepancies between results on cognitive tests that localize to the same anatomical region

(e.g., memory). The individual may do better in more complex tasks in the same domain than simple ones. In such situations, cognitive "effort tests" may be useful. These are very simple tests that even patients with severe dementia or head injury should be able to perform well. For example, the coin-in-the-hand test involves 10 trials of showing a patient which hand a coin is held in, asking them to close their eyes for 10 seconds and then choose the hand with the coin. A score at chance indicates poor effort. A score below chance is sometimes used as evidence of factitious disorder/malingering, although in reality it cannot distinguish between conscious or unconscious deception.

31. (e)

The nomenclature of progressive supranuclear palsy (PSP) is confusing. PSP-syndrome (PSP-S) refers to the classical clinical presentation, while PSP pathology refers to pathologic substrate. The classical clinical presentation of PSP-S is characterized by unexplained falls, symmetric predominantly axial rigidity, bradykinesia, poor levodopa responsiveness, and impaired vertical gaze, especially downgaze. This typical presentation is also called Richardson syndrome. Other clinical features include eyelid apraxia, dysphagia, abnormal neck posturing, pseudobulbar affect, long tract signs, and impaired saccadic pursuits. Five clinical presentations associated with PSP pathology have been described:

1) *PSP-S*
2) *PSP-parkinsonism (characterized by limb and axial rigidity, tremor, levodopa responsiveness)*
3) *PSP-pure akinesia with gait freezing (early gait disorder with subsequent freezing, early micrographia, phonation difficulties, lack of levodopa response or early eye movement abnormalities)*
4) *PSP-corticobasal syndrome (CBS as previously described)*
5) *PSP-progressive nonfluent aphasia/apraxia of speech (language and/or speech disorder characterized by agrammatism and/or speech apraxia)*

The disease duration is approximately 7 years. Most patients die from complications related to dysphagia. Neuropsychologic profile includes prominent executive dysfunction. Language and speech difficulties can be present or even dominate the clinical presentation. Structural MRI demonstrates midbrain atrophy and has been called the "hummingbird sign".

Midbrain atrophy ("Mickey Mouse ears") due to enlargement of 3rd ventricle + interpeduncular fossa + decreased AP diameter of midbrain.

Enlargement of the third ventricle is also described in PSP. The FDG-PET scan reveals frontal-subcortical hypometabolism. Midbrain hypometabolism is also described and has been called the "pimple sign".

Gross pathologic findings with PSP pathology include midbrain, superior cerebellar peduncle, and subthalamic nucleus atrophy. PSP is a 4-repeat tauopathy characterized by globose tangles in the globus pallidus, substantia nigra, and subthalamic nucleus. The pathology also affects motor cortex, striatum, pontine nuclei, inferior olive, and dentate nucleus. Tufted astrocytes are also present. The neuronal loss in PSP correlates with NFT's rather than the astrocytic pathology. No disease-modifying treatment is available for PSP. Subgroups of PSP (PSP-parkinsonism) patients respond to levodopa therapy. SSRIs can be used for depression, anxiety, and pseudobulbar affect.

32. (f)

CAA is characterized by selective deposition of β-amyloid in the walls of cerebral vessels, primarily small and mediumsized arteries of the cortex and leptomeninges. Because the frequency of CAA increases steadily with age, reaching 60% in unselected autopsies of individuals older than 90 years, it characteristically causes ICH in the elderly and is rarely documented before the age of 55 years. In addition, the superficial location of the affected vessels in the cortex and leptomeninges is responsible for a predominantly lobar location of ICH. *The widespread character of the angiopathy is responsible for the observation of both recurrent and multiple simultaneous, predominantly lobar, hemorrhages in elderly patients. An additional characteristic of CAA is its association with histopathological features of Alzheimer disease. There is clinical and progressive dementia in 10% to 30% of patients with CAA and neuritic plaques in approximately 50% of cases. CAA may present with features other than ICH, such as episodes of transient focal neurological deficit clinically suggestive of either transient ischemic attacks or partial seizures.*

33. (b)

Memantine is an N-methyl-D-aspartate (NMDA) receptor antagonist. It also blocks the 5-hydroxytryptamine-3 receptor. Memantine was approved by the FDA for the treatment of moderate to severe AD and can be used in conjunction with AChEIs or on its own. Side effects are rare although confusion and dizziness have been reported.

Dosage:- Orally administered, initially at a dose of 5 mg once daily, then increased in steps of 5mg every week; usual maintenance dose is 20mg daily.

34. (a)

Rivastigmine is another AChEI with FDA approval. Rivastigmine is initiated at 1.5 mg twice daily, which can be increased by 1.5 mg twice daily every 2 weeks to a maximum of 6 mg twice daily. The side effects of rivastigmine mirror those of donepezil (vivid dreaming, diarrhea, and nausea) although gastrointestinal side effects may be more common. Rivastigmine has been formulated in a patch form and has demonstrated similar efficacy with decreased side effects.

In addition to treatment of motor symptoms, a recent multicenter placebo-controlled study of the AChEI rivastigmine in Parkinson's disease Dementia showed an improvement in cognitive, neuropsychiatric, and functional features, leading to FDA approval.

35. (b)

The normal range of score depends on education. Some experts suggest a normal cut-off of 19 for uneducated people, 23 for graduate of elementary or junior high school, 27 for high school graduates and 29 for college graduates. Usually, a score of < 23 for educated people is taken to indicate cognitive impairment. The test is weighted towards orientation and language (these elements form 19 of the 30 score points). It can be normal in patients with right hemisphere and frontal lobe damage. An abnormal score does not distinguish between a focal and a diffuse disease of the brain.

*Normal**: 27–30*

Mild dementia: 20–26

Moderate dementia: 10–19

Severe dementia: <10

36. (d)

37. (c)

Mood disturbance affects Cognitive function the most among the given options.

38. (c)
39. (d)
40. (b)
41. (c)

Attention is the ability to focus on a particular sensory stimulus or mental activity to the exclusion of others. When there is sustained attention we call it concentration. Arousal, which is a heightened general alertness , may enhance concentration in some cases, but can also impair it at extreme levels as in pain or terror. Attention can be projected globally or focally & it can be focused on external events or directed internally to our own mental processes. *Attention allows for concentration & vigilance. When attention is faulty, distractability, impersistence, or confusion can result.*

The ascending reticular activation system (ARAS) in the brainstem plays a key role in attention through an ascending electrical activation of the cortical areas. Neural networks from the reticular formation pass through the reticular nuclei in the thalamus & on to the different areas of the cerebral cortex. *The cortical areas have a reciprocal influence on the ARAS, thus forming a self-sustaining 'loop'. Networks involving the prefrontal lobes (concerned with executive function) are particularly important & appear to play a role in modulating attention to various stimuli based on the plans & goals of the individual. The medial temporal, medial frontal, & cingulated cortices & amygdalae participate in networks that modulate attention based on emotional & motivational aspects of the concerned stimuli. The posterior parietal cortex is involved in networks that modulate attention based on the importance of the concerned stimuli to the individual's current state (based on the contextual importance of the stimuli).* Networks also involve the parahippocampal gyri, isthmus, & lingual gyri bilaterally.

Attention can be directed diffusely, like a heightened sense of awareness in dangerous situations, or may be focused on a particular brain function (domain) such as a particular sensory modality. Attention to a particular domain is modulated by the cortical areas that are concerned with that function. For example, visual neurons are involved in generating attention to visual stimuli whereas somatosensory cortical neurons are involved in attention to sensory stimuli. Mood & motivation strongly influence attention through projections from limbic structures like the amygdala. Limbic structures can induce widespread attentional modulations that modify the impact of sensory inputs based on the emotional & motivational relevance of the event.

Poor performance on complex tests of higher intellectual function cannot be attributed to cortical dysfunction if the patient is not attentive to the tasks. Defective attention taints all subsequent testing. Patients may appear grossly alert but are actually inattentive, distractible, and unable to concentrate. An early manifestation of toxic or metabolic encephalopathy is often a lack of attention and concentration in an apparently alert patient, which may progress to delirium or to a confused state. *Confusion, inattention, and poor concentration may also be seen with frontal lobe dysfunction, posterior nondominant hemisphere lesions, and increased intracranial pressure. Lesions causing apathy or abulia also impair attention. Patients with dementing illnesses are not typically inattentive until*

the cognitive deficits are severe. Consider the possibility of a central nervous system toxic or metabolic disturbance when the patient is inattentive.

42. (d)

Cognitive deficits are among the most debilitating and complex aspects of TBI to manage and treat. Cognitive problems after TBI can span a number of neuropsychological domains, including arousal, attention, memory, and executive control. The neurobiological basis of both physiological cognitive function and TBI-induced dysfunction are not completely understood, and neuropharmacological approaches for treating TBI mediated deficits have largely drawn upon literature from related disorders that present with cognitive dysfunction. Among these are attention deficit hyperactivity disorder (ADHD), Parkinson disease, narcolepsy, and Alzheimer/dementia. However, *recent research has shown that the patho-mechanisms involved in TBI-related cognitive deficits are unique and are not wholly similar to these other diseases.*

Arousal is one of the most basic cognitive functions required to engage in other higher levels of cognition. Arousal is governed by the reticular-thalamic, thalamocortical, and reticular-cortical networks. Multiple neurotransmitter systems, particularly monoamines, impact arousal. These monoaminergic nuclei originate in the brainstem at the reticular formation, and each has multiple forebrain terminal projections. While other neurotransmitters influence arousal, norepinephrine (NE) is central to this function, which impacts other cognitive domains such as attention and memory.

Fundamental to injury-induced reductions in arousal is impaired consciousness.

43. (d)
44. (c)
45. (c)

Donepezil is a reversible inhibitor of Acetyl Cholinesterase.

It has been reported to improve both cognitive and odor identification scores in patients with Alzheimer disease. The mechanism of action of AChEIs is to improve cholinergic functioning in the brains of patients with AD by increasing the concentration of acetylcholine through inhibition of acetylcholinesterase. Tacrine was the first AChEI approved for use in AD, but was limited by four times a day dosing and strict hepatic function monitoring requirements due to a risk of toxicity. These limitations in combination with the development of newer drugs resulted in its disappearance from the market place. *Donepezil, a reversible AChEI, that can be administered with a single daily dose and does not require laboratory monitoring was the next AChEI approved by the FDA. Donepezil is initiated at 5 mg per day for 28 days and if tolerated can be increased to 10 mg daily. Initial donepezil studies were 12 to 24 weeks in duration and demonstrated improvement on neuropsychological testing and clinician evaluation.*

46. (c)

Hemineglect can arise from injury to any node in the cortical limbic-reticular network, which directs attention and integrates the localization and identification of a stimulus and its importance to the person.

Community-based studies of stroke survivors detect visual neglect in 10% to 30% of patients. The neglect is modestly associated with poorer ADL scores and slower recovery, although severe neglect is rare beyond 6 months. Right-sided inattention, when looked for,

has been detected in 15% to 40% of nonaphasic patients with acute left cerebral infarcts, although it is clinically the most prominent after right brain injury. Patients with anosognosia, visual neglect, tactile extinction, motor impersistence, or auditory neglect have the lowest BI scores at 1 year, even after the data are adjusted for initial ADL scores and for poststroke rehabilitation. During rehabilitation, patients with left hemiplegia and neglect tend to improve on the motor portion of the FIM more than those with neglect and anosognosia.

The therapeutic options available for hemineglect are:-

- *Multisensory visual and sensory cues, then fading cues*
- *Verbal elaboration of visual analysis*
- *Environmental adaptations (bed position, red ribbon at left book margin)*
- *Computer training*
- *Video feedback*
- *Monocular and binocular patches*
- *Prisms*
- *Left limb movement in left hemispace*
- *Head and trunk midline rotation*
- *Vestibular cold caloric stimulation*
- *Contralesional cervical nerve stimulation*
- *Repetitive transcranial magnetic stimulation of unaffected hemisphere*
- *Reduction of hemianopic defects by a few degrees*
- *Pharmacotherapy trials; rotigotine*

47. (c)

Executive functions include the ability to shift sets, organize, and problem solve, as well as the abilities of cognitive control and working memory. Shifting sets is related to mental flexibility and consists of the ability to move between different concepts or motor plans, or the ability to shift between different aspects of the same or related concept. Working memory is the online maintenance and manipulation of information. The DLPFC–subcortical circuit includes the dorsolateral head of the caudate and the lateral mediodorsal globus pallidus interna and the parvocellular aspects of the mediodorsal and ventral anterior thalamic nuclei. Dysfunction in this circuit has been linked to environmental dependency syndromes (including utilization and imitation behavior), poor organization and planning, mental inflexibility, and working memory deficits. Executive dysfunction is also a principal component of subcortical dementias. *Deficits identified in subcortical dementias include slowed information processing, memory retrieval deficits, mood and behavioral changes, gait disturbance, dysarthria, and other motor impairments. Vascular dementias, Parkinson's Disease, and Huntington's Disease are a few examples of conditions that affect this circuit.*

48. (a)
49. (c)
50. (a)

*Information has been presented to help better understand Cognition & Cognitive rehabilitation (some points have been repeated). For more information on Cognition & Cognitive Rehabilitation read the following books:-

1. Bradley's Neurology in Clinical Practice, 7th Edition (Chapter 43 & 95)

2. Harrison's Neurology in Clinical Medicine, 4th Edition (Chaprter 29)

3. DeJong's The Neurologic Examination, 8th Edition

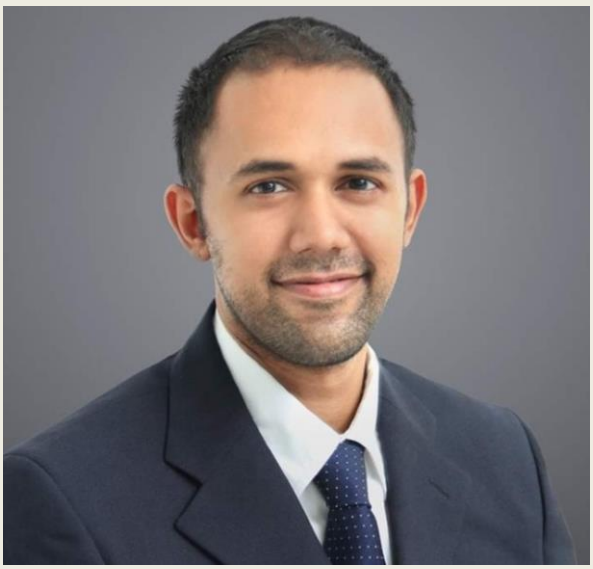
4. Higher Neural Functions: A Clinical Approach, by Dr. M. Madhusudhanan

5. Oxford American Handbook of Neurology

6. Physical Medicine & Rehabilitation Secrets, 3rd Edition

7. Braddom's Physical Medicine and Rehabilitation, 6th Edition

The Road to Starting a Cognitive Rehabilitation Clinic



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Having trained in Fluroscopic injections as a resident I never thought I'd be looking into cognition for my patients. Frankly I had no idea what direction I would grow in after training at MSU. Much of Neuro-rehabilitation for residents in the US is Acute Inpatient Rehabilitation. On the floor Neuro-pyschology was active, and our PMR consultants did the long-term follow-up. My exposure was zero. It took a few encounters in Amrita to make me realize this was a problem PMR could be active in too. The first time was a decade ago with a TBI patient in the ward. Neurosurgery had stabilized and transferred him to us for 'Physio'. Something seemed off about the patient's affect though. He wasn't expressing much and had increased tone in his right elbow. Despite being on a hefty dose of Baclofen, it was still tight. Through the exam it occurred to me that the hypertonia was biphasic, meaning it was more likely rigidity. This matched his imaging and I switched him to Syndopa. The next day his behavior, speech, and tone were normal. He improved so fast the family requested discharge in a few days. He actually didn't need a therapist once the neuro-chemical imbalance was addressed.

The next time I saw this was when neurosurgery called me to botox a patient of theirs with trephination syndrome. The diagnosis was new to me, but again I caught the abulic stare of a man who could do all his activities. The problem was someone had to tell him what to do, and when. Neurosurgery felt if his R equinus deformity was corrected with botox he could walk and be sent home. Instead Syndopa was started and within 24 hours he was as good as presurgery. The deformity also got corrected (it was just a weight distribution issue). He went home the next day and resumed his chappal store manager role in a month. It occurred to me that the presentation of the body depended a lot on the condition of the brain.

A few months later we got a patient from Vellore. After a TBI and decompressive hemicraniectomy surgery they stagnated for a

month. The family brought him home to Kerala for their convenience, and to us as they lacked confidence in care. We accepted a somnolent young man with L hemiplegia. Going for a hat-trick we added a dopaminergic again, and he was up walking in a week. He later resumed his IT job and got married.

There is no value in being a one-trick pony though. I knew I was missing something but was not sure where to start looking for solutions. Neurosurgery as usual had a TBI patient for us. He was comatose, so for them he couldn't go home. Instead they sent him for 'Physio+ ?'. They were learning that we also had a role in care aside from therapy services. This time a dopaminergic took him from non-responsive to dancing in bed for all the time he wasn't sleeping or getting tilt-table therapy. He didn't open his eyes or talk though. The family tied his limbs and he kept wriggling around doing his version of the 'Harlem shake'. Of course we ruled out seizures etc, but this guy would not wake up further despite escalating the dose, untying him, etc. The day he woke up to alertness the family promptly took him to Vellore. I forgot about him until we met a year later, but all that time I wondered what more I could do to help people like him.

Something was missing. We were addressing neuro-chemical imbalances but not the structural issues. At this time my neurosurgeon buddy asked me if I would start a HBOT service here. His interest was controlling vasospasm in post aneurysm clipping patients. Seeing the 1 crore price-tag I was scared. As it is no one knew what we do, and when we innovated they often rejected it. Other departments would either tell patients to only take physio and nothing else, or outright argue us about services they knew little of. In my prayers I mentioned my fears. The answer I got was 'Be bold, don't be afraid to do the right thing'. After all I wasn't doing this for personal gain or fame. All this was to fill a missing gap in patient care.

After getting the chamber I still was unsure what to expect if I put a person in. How many sessions did they need? What should we expect? Thanks to Neurosurgery I found out. They gave me their latest TBI whom they cared for over 2 months, through sepsis etc. When he came to us he was as usual constipated, got a UTI the next day, then went into sepsis. After treating all these, his BP would not come down, he was always tachycardic, and sweating all the time. Soon we learned about Paroxysmal Sympathetic Hyperactivity and began its treatment. He also had a Right Antero-rotacollis. Neurology wanted to botox the SCM for me, but ensured it would not wake him up. His Mom had no money for this. Propranolol could not control this, so as a last ditch effort I put him into the new Hyperbaric chamber. He woke up a week later and asked if he was in the lunatic asylum. He had regained consciousness, spoke to his Mom after 2.5 months, made a relatively logical deduction, and had no more involuntary neck movements. I knew I was onto something with this treatment.

Neurosurgery was impressed and started calling for us before they wanted therapy, just to get patients to wake up sooner. The result was those who did went home without coming to our side. It was an acceptable loss though. One should never get too comfortable and take things for granted. One of the latest Neurosurgery specials was a man with an EDH. Despite drainage he hadn't woken up. HBOT took him there, and he went home walking and doing his ADLs independently. At three months follow-up, his wife was angry. Why? The man she took home from the hospital had her husband's face, but not his personality. He had spent the last three decades dominating her and having his way with her. She had adapted to this. The man who came home from the hospital would cry if she said hello to him. This she could not tolerate.

She knew HBOT helped earlier, but I had neither the evidence nor the experience to address his behavior issues with it at that time, so we used psychiatric meds instead. After a month he was the same, and she insisted on HBOT. He took another 10 sessions after which his behavior got better and their relationship improved. More importantly we got him off pysch meds.

A woman who obsessed with urination came next. An outside referral from a nearby centre came for TBI management. She was Ranchos 4 with us during admission and continued to scream and shout without trigger or warning though OPD based rehab. All she wanted was to go home. Having no corticospinal deficits, we let her go, though the husband wanted more from us. Trazodone at the upper limits was not able to get her to calm down. On one month follow-up she was calmer, but her husband mentioned she always wanted to urinate or defecate, even after having done so just 10 minutes before. He asked me if HBOT could help her. All three of us were naïve to its potential, but only he was convinced it would help. I gave her Tolterodine for a month and asked them to review earlier if the symptoms weren't better. After two weeks he came alone asking if they could try HBOT, the drugs were not working. Unsure and wary I did a MOCA for her, and then we tried 10 sessions. I had to make sure my results were objectively track-able. At the end of that her MOCA had gone from an 11 to 27/30, and the urge for bowel/ bladder became normal. From her I learned we can improve cognition in these patients.

Rehab is always teamwork. I could do MOCA and it would show me domain deficits. From there I could plan specific interventions, but I could not stop my 12 hour work day to do the exercises with patient. Some families were confused by my accent, others just wanted hand-on-hand guidance. I also realized I didn't fully understand the variety of diagnoses that deficits could fit into. Knowing the diagnosis facilitates neuro-pharmacological agent selection. To meet those needs the head of Neurology, a clinical Psychologist, a speech therapist and myself banded together to serve these patients better. This happened two years ago. Then Corona came.

As always it's how we face the obstacles that determine the success of an endeavor. Families want their patient to be as good as possible. Sometimes their expectations don't match reality. It's important to try correlating imaging, testing, and affect when you plan. It lets the patient/ family know you are working with everything at hand. It also allows for transparency, so that when they are ready to accept reality, they have no enmity towards you. The next obstacle is the stigmata of going to Psychology. The implications are that there is something really wrong with the patient, which may not make sense to the family. One of my TBI patients recovered quite well after HBOT and had subtle memory deficits. The wife insisted he was perfectly fine and didn't need a Psychologist to get better. After doing the MOCA things changed. He was an expert at math, but now could not do serial 7's. She immediately agreed to the referral and did all the exercises with him in the room afterwards. He improved.

Who needs to do this? There are a variety of services our allied departments can perform to help our patients. It is up to you to figure the rest out. As long as your focus is translating all the medical and paramedical jargon into a functional outcome, you're likely on the right path. The worst outcome then is nothing changes. At least you tried, and can learn from the experience. Anything else is a success.

Short-Term Functional Outcomes in Five Strand Hamstring Graft for Anterior Cruciate Ligament Reconstruction among Young Adults – A Prospective Pilot Study

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ABSTRACT:

Anterior Cruciate Ligament (ACL) injuries are common sports injuries and ACL doesn't have healing property. So, it is repaired by graft surgery. This pilot study assess the short-term functional outcomes among young adults undergoing five strand Hamstring graft for anterior cruciate ligament injury using IKDC score. A total of 14 patients were included in the study and were followed up for 6 months post ACL reconstruction. The ACL injuries are more common in physically active age group of 20 to 40 years. We found that pre-operative Mean IKDC scores were 43.22+/- 9.91. IKDC scores at 6 months follow up were improved to 92.6+/- 1.85 with significant differences (p- value <0.001). There was improvement in Pain Visual Analog score (VAS) from mean pre-operative VAS score was with standard deviation 1.21 to post-operative VAS score 1.07 with standard deviation 1.49 (p value < 0.0001). The results of this study can be used to plan a bigger study with a large

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sample size and extended follow-up.

Key words: ACL injuries, reconstruction, five-strand hamstring graft

INTRODUCTION

ACL injuries are most commonly associated with sports-related trauma. Injured ACL don't have healing property so it has to be replaced by other grafts. Many grafts are available for ACL reconstruction. Different surgeons proposed different techniques for preparing five strand hamstring graft and different fixation methods were used for five strand hamstring graft fixation in the tibial and femoral tunnel. In this current study, we used Bioscrew /Endobutton for femoral fixation and Bioscrew /Cortical screw+ washer for tibial fixation.

Table-1: Comparison of different fixation methods in different surgical techniques

Study	Femoral fixation	Tibial fixation	Graft
Lee et al.[1]	femoral cortical button	tibial interference screw	3ST+2G
Prodromos et al.[2]	cancellous screw	cortical screw	3ST+2G
Lavery et al.[3]	EndoButton	Screw	3ST+2G
Calvo et al.[4]	Tight Rope (Arthrex)	interference screw	3ST+2G
Tutkus et al.[5]	absorbable interference screws	absorbable interference screws	3ST+2G
Present study	Bioscrew /Endobutton	Bioscrew /Cortical screw+ washer	3ST+2G

Patellar tendon and the hamstring grafts are among the most commonly used grafts for ACL reconstruction. Recently hamstring grafts got attention due to lack of comorbidities. But failure rate varies according to graft diameter[1]. Usually, four-strand hamstring graft is used for ACL reconstruction but the revision surgery rate is high due to less graft diameter. To overcome this problem five strand hamstring graft can be used, the extra strand used increases graft diameter by 1-1.5mm[2].

This study was conducted for assessment of functional outcomes at short- term follow up in ACL reconstruction by five strand hamstring autograft in the young population and to study the associated comorbidities with hamstring graft.

AIMS AND OBJECTIVES

To assess the short-term functional outcomes among young adults undergoing five strand Hamstring graft for anterior cruciate ligament injury using IKDC and Lysholm scores.

MATERIALS AND METHODS

We studied 14 patients, in which ACL reconstruction was done by five strand hamstring graft. All patients were subjected to a standardized surgical technique and post-operative rehabilitation protocol and followed up till 6 months post-operatively. Post-operative follow up included IKDC scores, Lysholm scores, anterior laxity, VAS score and 1 RM max for hamstring muscle was documented and compared with pre- operative scores.

Study Site: The study was carried out in the Orthopaedics department, JIPMER, Pondicherry, India.

Study Design: A Prospective cohort study.

Sample size: 14 patients

Study Duration: 24 months

Treatment follow up period: 6 months

Eligibility for the study: Inclusion criteria:

1. Patients with symptomatic ACL deficient knee undergoing arthroscopically assisted ACL reconstruction due to instability within one year from the time of injury.
2. Age 18-45 year

3. Normal contralateral knee

Exclusion criteria:

1. Patients having associated PCL rupture.
2. Prior knee surgery/infection.
3. Avulsion fracture
4. Grade 3 collateral ligament injury/meniscal tear involving ≥ 2/3rd of meniscus
5. ≥ Grade 2 Kellegren-Lawrence arthritis

Methodology: This was a Prospective cohort study done on ACL deficient knee patients from July 2015 to December 2017 in the Department of Orthopaedics, JIPMER, Puducherry. The Institutes Ethical Committee and Post-graduate research monitoring Committee approved the study. A total of 14 patients were included in the study, all patients were operated with five-strand hamstring autograft. These patients were followed-up for 6 months.

Operative method: ACL reconstruction was performed with the anatomical single- bundle technique. A 4 cm incision anteromedially on the tibia was made directly over the pes anserinus in line with the hamstring tendons. After the sartorius expansion has

been identified, a longitudinal incision was made in Sartorius fascia to identified gracilis and semitendinosus tendons at their distal insertion on the anterior aspect of the tibia and tagged prior to harvest. All soft-tissue adhesions from each tendon were carefully removed before it is harvested. A tendon harvester was placed around the distal end of the tendon and was advanced along its substance into the proximal portion of the thigh. Gentle countertraction was applied to the distal end of the tendon with use of an angled retractor or clamp. When the harvester was advanced past the muscle-tendon junction, the tendon releases proximally and then pulled out of the harvesting device and out of the tibial incision. The tendons on the graft preparation table and cleaned of soft tissues, the length and the feasibility of tripling the semitendinosus were assessed and was converted in the triple layer with each strand in same tension. The gracilis was doubled and was kept over three strand of semitendinosus. We used a no 5 Ethibond suture to stitch all five strands at distal third part of hamstring autograft. In order to minimize bulk, we did Whip stitching in a non- locked pattern.

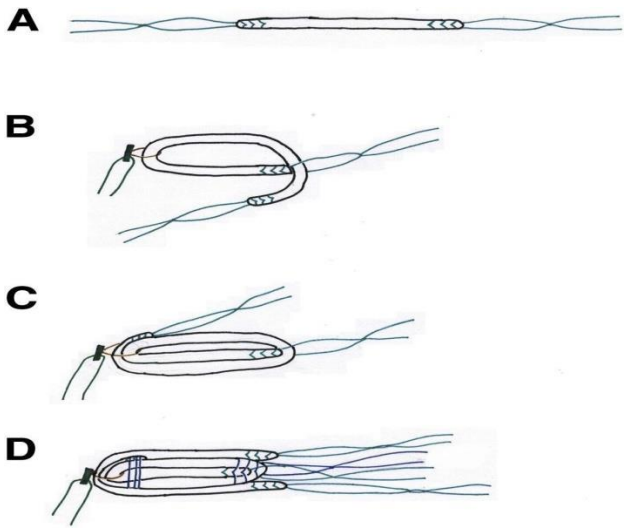

Fig 1 showing the 5-strand hamstring autograft technique. (A) ST graft is prepared at both ends with suture. (B) ST graft is tripled over itself. (C) A total of 5 mm of the thinner end of the ST graft is passed over the tripled graft (D) Final 5-strand graft with doubled gracilis graft on the top.

Post op care: The knee was immobilized in above knee POP slab. Post-operative day 2 the POP was discarded, and a small dressing and Bandaid used to cover the wounds and port sites. Drain removed and antibiotics stopped. Check x-ray was taken on post- operative day 2. The knee was then put on a hinged knee brace or a posterior splint, and knee mobilization as per standard protocol (Appendix 6) was initiated. Simultaneously partial weight bearing was initiated with the help of axillary crutches. The target was 90^{o} knee flexion within 2 weeks and full flexion within 4-6 weeks. On the 14th-day stitch, removal was done. Crutches were

used till ~4 weeks. Weaning off brace was achieved at 4-6 weeks by this time ligamentisation of the autografts starts. At 6 months post-operative IKDC, LYSHOLM score, anterior laxity, 1 RM MAX for hamstring muscle strength, Visual Analog Scale (VAS) score was recorded and donor site morbidity was checked. 1 RM MAX was checked with the patient in proneposition on leg curl machine.

Statistical Analysis**:** The analysis was done by SPSS version 19 (IBM Inc, Chicago, IL, USA) software. We presented data as mean ± SD. Paired student t test was used to analyze the primary outcome measures (Differences in the scores) before and after the operation. We kept the level of significance at 5%. We considered P value lessthan 0.05 as significant.

RESULTS:

In this study, 12 patients (85.8%) were in age group of 20-40 years,1 patient (7.1%) was less than 20 years of age and 1 patient (7.1%) was of more than 40 years age.

Pre-operative Mean IKDC scores were 43.22 +/-9.91. Mean pre-operative Lysholm score was 49.07 +/-8.93. IKDC knee examination score was grade C. IKDC scores at 6 months were 92.6 +/-1.85 with significant differences (p-value <0.001). At 6 months mean Lysholm scores were 96 +/-3.16 with significant differences (p-value <0.001). At 6 months, all patients had IKDC Grade A score.

Table 2: IKDC subjective knee scores and Lysholmscores

	N	Mean	Std. Deviation	Std.Error Mean
IKDC	14	43.22	9.91	2.650
LYSHOLM	14	49.07	8.93	2.386

	N	Mean	Std. Deviation	Std. Error Mean	p- value
IKDC 6m	14	92.6	1.853	.495	<0.001
LYSHOLM 6m	14	96	3.162	.845	<0.001

Pre-operative 1RM max for Hamstring muscle was 5-10kgs and post- operative 6months 1RM max was 10-15kgs.

Table 3: Pre-operative 1RM max and at 6 months follow up

	N	Mean	Std. Deviation	minimum	max
1RM max 0m	14	6.4	2.3	5	10
1RM max 6m	14	11.4	2.3	10	15

Patients had pre-operative VAS score range 2-6. The mean pre-operative VAS score was 4.35 with standard deviation 1.21. Patients had post-operative 6 months VAS score range 0-3. The mean post-operative 6 months VAS score was 1.07 with standard deviation 1.49. p value < 0.0001

Table 4: VAS score pre-operative and 6 months follow up

	N	**Mean**	**Std. Deviation**	**Std. Error Mean**
VAS 0m	14	4.35	1.21	.324
VAS 6m	14	1.07	1.49	.368

Intra-operative hamstring graft diameter range was found in between 8.5- 9.5mm and mean was 9.1 mm. Anterior laxity in the pre-operative period was grade C (5-10mm) and after post-operative 6 months, anterior laxity was grade A (0-2mm). None of the patients had laxity. Around 50% patients complained of anterior knee pain but the intensity was significantly decreased compared to pre-operative period. Out of 14 patients, one patient complained of decreased sensation over scar site. Patients had very good functional outcomes in post-operative 6 months follow up as compared pre-

operative status.

DISCUSSION

ACL injuries are usually related to sports activities, and the vast majority of them require surgical reconstruction with a reasonable graft to Successfully re-establish the normal functions of the knee joint and to eliminating anterior tibial subluxation. The quadrupled hamstring grafts is an ideal graft for ACL reconstruction. Quadrupled hamstring grafts diameter is 7-8mm and due to less graft diameter, there is a more ACL graft failure rate.

The diameter of ACL hamstring graft is directly correlated with graft strength [6]. It has been found that graft of larger size behaves close to native ACL in relation to fiber length changes which control the knee laxity in flexion range [7].

It has been found that revision surgery rates are high in hamstring grafts of smaller size. So, new techniques have been developed to increase the size of hamstring graft. In a new technique, the standard 4-strand hamstring graft can be converted into 5- stranded one with the use of 3 equal strands of semitendinosus and 2 strands of gracilis. In five strand hamstring graft usually, graft diameter is 9-10mm. This extra strand of graft increases the diameter by 1-1.5mm and provide more stability, better functional outcomes and the less failure. The present study was aimed at investigating the functional outcome of the 5 strand hamstring grafts.

The age distribution of patients in our study shows that ACL injuries are common in the physically active population group of 20-40 years. The mean age of patients in our study was 28.78 years. In another Indian study done by Patel et al [8] in 25 patients with quadrupled hamstring graft with biodegradable interference screw found mean age 28.2 years which was similar to the current study. In a retrospective analysis done by Calvo et al [4] to compare the outcomes of Five-Strand Hamstring Autograft and Quadruple Hamstring Autograft. In this study, they found that mean age in five strand hamstring group was 30.6 years which is similar to our study.

Table-4: Comparison of four-strand and five strand hamstring graft- demographic data and intra-operative graft diameter data

Study	**Type of study**	**Patients** 5-strand	**Mean age(**5-strand**)**	Average graft diameter **(**5-strand**)**
Calvo et al.[75]	retro	37	30.6	9.2 mm
Current study	pro	14	28.7	9.1 mm

More than half of the patients (57.14 %) were injured during a sporting activity. 28.57 % were injured in a road traffic accident. Also, more often than not females are more prone to ACL injuries [9]. However, our study doesn't have any female patient. This reflects the lower participation of females in sports and outdoor activities owing to their social obligations.

The present study included patients who presented within one year of injury. It was conducted in a population of predominantly rural background where still indigenous treatment techniques such as native

bandaging hold a prominent place in injury management. Indian rural population usually present to modern tertiary healthcare facilities after some time of intentional neglect or after a period of native treatment. We had 2 patients who had at least 1 month of indigenous treatment before approaching our hospital. However, at the time of surgery both patients were optimized to have full knee ROM without any effusion, so that post- operative function is not biased.

In a recent study by Lavery et al [3], described a new technique of making 5- strand hamstring autograft by attaching the extra strand to the suture button loop. This extra

strand of 5-HT autograft was expected to increase the diameter of the graft and possibly graft strength also. They used EndoButton for femoral fixation and screws for tibial fixation.

We found significant differences in the IKDC, IKDCE scores and Lysholm scores between pre-operative and post-operative period. We found that mean IKDC score pre-operative was 43.22(SD=9.91) while post- operative 6 months follow up mean score was 92.6 (SD=1.85). this result was similar to the retrospective study done by Calvo et al [4] in which they found mean IKDC score was pre-operative 61.02 and post-operative 96.8.

We found that Mean Lysholm score pre-operative was 49.07 (SD=8.93) while post-operative 6 months follow up Lysholm scores was 96 (SD=3.16). this result was similar to the retrospective study done by Calvo et al [4] in which they found pre-operative Lysholm score was 59.1 and post-operative Lysholm score was 97.1 in 6 months post-surgery follow up.

Table - 5: Comparison of pre-operative and post-operative IKDC and LYSHOLM scores in five strand hamstring graft

Study	Mean IKDC score pre-operative 5-strand	Mean IKDC score post-operative 5-strand	**Mean** LYSHOLM Score pre-operative **(**5-strand**)**	**Mean** LYSHOLM Score pre-operative **(**5-strand**)**
Calvo et al.[75]	61.02	96.8	59.1	97.1
Current study	43.22	92.6	49.07	96

IKDCE group pre-operative was C and after post-operative 6 months

follow up, it was A. Similar study by Patel et al [8] on 25 patients, where they reconstructed ACL with help of four-stranded ST-G autograft fixed with bio absorbable interference screw. In this study, they found that the mean Lysholm knee score was 91.2 (range, 63 to 99) and the mean IKDC score was 90.7 (range, 60 to 98) at 6 months follow up after surgery.

In the present study, Patients had pre-operative 1RM max ranging from 5-10 kg. The mean pre-operative 1RM max was 6.4 kg with 2.3 standard deviations. Patients had post-operative 6 months 1RM max ranging from 10-15 kg. The mean post-operative 1RM max was 11.4kg with 2.3 standard deviation. The Patients had pre-operative VAS score range 2-6. The mean pre-operative VAS score was 4.35 with standard deviation 1.21 and Patients had post- operative 6 months VAS score range 0-3. The mean post-operative 6 months VAS score was 1.07 with standard deviation 1.49. Similarly, Soon et al [10] study on 76 patient and found no anterior knee pain at rest. A study was done by Spicer et al [11], on forty- four patients found that almost 89 % patients were asymptomatic or experienced little pain with daily normal activities.

We found that all patient who was in group C pre-operatively where anterior laxity was 6-10 mm improved to group A post-operatively where the anterior laxity was 0-2 mm. Donor site sensation pre-operatively was normal in all patients and in post-operative 6 months follow up we found that donor site sensation was decreased in 1 patient. Similarly, Soon et al [10], a study on 76 patient, they found that only 3 patients had less sensation at the donor site. In contrast to our study Sgaglione et al [12] found that 37% patients had paraesthesia after reconstruction of ACL with semitendinosus tendon. However, it was argued that paraesthesia was because of arthrotomy rather than the tendon harvest.

We found intra-operative hamstring graft diameter range from 8.5- 9.5mm. In 6 patients graft diameter was 9.5mm, in 6 patients graft diameter was 9 mm and in 2 patients graft diameter was found 8.5mm. In a retrospective study by Calvo et al [4] where they compared

outcomes of Five-Strand Hamstring Autograft and Quadruple Hamstring Autograft and they found average diameter in five strand hamstring graft was 9.2mm. In a study by Magnussen et al [13] on 256 patients who were operated by hamstring autograft found that 7 % of the patient need for a revision surgery at 14 months post-operative follow up. They found that patients with less graft cross sectional area and age were having more failure rate and underwent revision surgery. In patients with graft cross sectional area was more than 8 mm, revision surgery rate was 1.7% and cross-sectional area between 7.5-8mm had revision surgery rate of 6.5 %, cross-sectional area less than 7 mm had revision surgery rate of 13.6.

CONCLUSION

To conclude the present study showed that there are good clinical outcomes of five strand hamstring autograft at short-term follow-up.

We found that at 6 months follow up post-operative almost all of our patients regained normal strength without any decrease in donor site sensation. None of them had a complaint of subjective knee laxity. The pain was significantly reduced. Not a single patient required revision surgery due to laxity, this may be because of larger graft diameter of five strand autograft. The clinical outcome scores as measured by IKDC score, Lysholm score were significantly improved at short-term follow up periodof 6 months.

We found that pre-operative Mean IKDC scores were 43.22 +/-9.91. IKDC scores at 6 months follow up were 92.6 +/-1.85 with significant differences (p- value <0.001). We found that pre-operative Lysholm score was 59.1 and follow up at 6 months post-operative the Lysholm score was 97.1. We found that Intra-operative hamstring graft diameter range was found in between 8.5-9.5 mm and mean was 9.1 mm 1RM max for hamstring pre-operative was 5-10 kgs. and after 6 months follow up 1RM max was 10-15 kgs. Patients had pre-operative VAS score range 2-6. The mean pre-operative VAS score was 4.35 with standard deviation 1.21. Patients had post- operative 6 months VAS score range 0-3. The mean post-operative 6 months VAS score was 1.07 with standard deviation 1.49.

LIMITATIONS

There are some limitations to our study. We included only 14 patients in our study. All patients completed 6 months follow-up. The small sample size is the limitation of our study. The short-term follow up is also another limitation. A larger study with longer follow-up could probably shed more light on the long-term outcomes of five strand hamstring autograft.

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Effect of Mobile Phone Usage on Median Nerve Conduction Velocity.

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INTRODUCTION

Mobile phones are quintessential pocket devices in our daily life. They are an extensively used mode of communication. The advancing technology has nearly made smartphone to replace even laptops. Also the Covid pandemic has lead to further dependency on smartphone both in office works and leisure activities. This increasing dependency, raises the question of possible health effects particularly on neural functions.

Dr. Sreethu Syam, did MBBS from T. D medical college Alapuzha, currently doing MD PMR in GMC Trivandrum. She has special interest in pain rehab & has presented free papers and posters for national and state conferences. She got best poster award in national conference and best free paper award in state conference.

Studies shows that, people who use mobile >3.5 hours/day , can have pain at the base of the thumb.Nerves can be damaged by the repeated use of thumbs for mobile usages. This is because users often hold the device with a single hand, which forces the thumb to use the keys. It compels individuals to engage in repetitive flexion/extension of the wrist and repetitive stress on tendons.

Recent evidence has indicated that frequent smartphone users are more susceptible to have swollen median nerve. Median nerve is quite susceptible to be damaged in view of its precarious location in the carpal tunnel. This can be studied by assessing conduction velocity of median nerve.

There is a flurry of patients with wrist pain and paresthesia of hand in OPD and in most of these cases there is no implicatory factors other than over use of smartphones. Some patients raised the concern whether it is a result of over use of smartphones. This prompted us to investigate the matter. So in this study, our aim is to find out the effect of mobile phone usage on median nerve.

We hypothesized that overuse of smartphones may affect the flexor pollicis longus tendon (FPL) and the joints of the thumb as well as the median nerve, due to wrist posture.

OBJECTIVE-

To find out the effect of mobile phone usage on conduction velocity of median nerve.

MATERIALS AND METHODS:

This was a Cross sectional study conducted with 30 Mobile phones users attending Department of Physical Medicine and

Rehabilitation, Govt Medical College Thiruvananthapuram from a study period of December 2020 to May 2021 allotted using Consecutive Sampling. After getting an informed consent from all participants, pre procedure assessment of the patient including history, general examination,systemic examination with all required investigations were done and recorded.

INCLUSION CRITERIA:

1. Age between 18 to 40
2. Patients using mobile phone continuously in one hand method for more than six months
3. Patients consenting to the study
4. Stable vitals

EXCLUSION CRITERIA

1. History of diseases in the muscles and nervous system
2. History of any injuries, fractures or pain in the wrist area
3. Any pathology affecting wrist or anatomical variance (arthritis, leprosy,square wrist)
4. Uncontrolled diabetes, CAD, seizure, unstable patients, hypothyroidism
5. Pregnant females
6. History of chronic tobacco or alcohol consumption

STUDY INSTRUMENTS;

1. Nerve conduction study using Natus Keypoint NCS machine

Nerve conduction studies were carried out in the carpal tunnel protocol in antidromic method. Sensory and motor nerve conduction studies were done for median nerve in both hands.

2.Smartphone addiction scale- short version (SAS-SV)

Smartphone Addiction Scale- is a scale for smartphone addiction that consisted of 33 questions with a six-point Likert scale (1: ‘‘strongly disagree’’ and 6: ‘‘strongly agree’’) based on self-reporting. Its short-version scale as developed in order to evaluate the degree of addiction in teenagers as well as to suggest a cut-off value by gender using the previous scale Based on Number in Experts’ Agreement-Content Validity Index of SAS. - final set of 10 questions. the cut off value was 31for boys, and for the girls, the cut-off value was 33.

Slno	ITEM	SCORE-6-point Likert scale
1	Missing planned work due to smartphone use	
2	Having a hard time concentrating in class, while doing assignments, or while working due to smartphone use	
3	Feeling pain in the wrists or at the back of the neck while using a smartphone	
4	Won’t be able to stand not having a smartphone	
5	Feeling impatient and fretful when I am not holding my smartphone	
6	Having my smartphone in my mind even when I am not using it	
7	I will never give up using my smartphone even when my daily life is already greatly affected by it.	
8	Constantly checking my smartphone so as not to miss conversations between other people on Twitter or Facebook	
9	Using my smartphone longer than I had intended	
10	The people around me tell me that I use my smartphone too much.	

STUDY PROTOCOL

After taking consent of participants, and baseline evaluation, structured profrma was filled .SAS-SV was administered for addiction scale.Their Median motor and sensory NCV studied in both hands using NatusKeypoint NCS machineand compared.

METHODS AND ANALYSIS:

Data collected and converted into tabular form. Data analyzed using statistical software SPSS version 2.5 and compared using Chi square test.

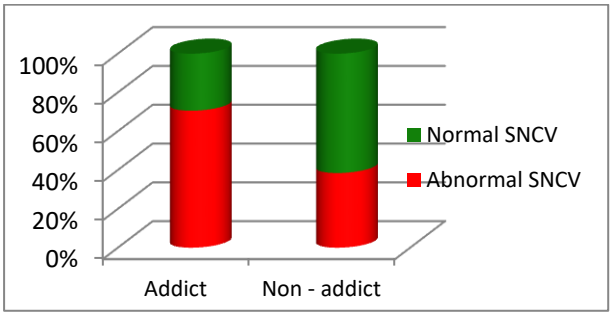
RESULT:

Totally ,30 participants were included in the study,10 of them (33.3%) were females and 20 of them (66.6%) were males. 19 (63.3%) were 30-40 years of age. The mean age of total participant was 32.5.28 participants (93.33%) were right-handed and 2 (6.66%) were left-handed. All the participants were using mobile phones with their dominant hands. NCV of non-dominant hand was found to be normal in all participants.11 participants (36.66%) were in a habit of using mobile phones continuously for the past 1 to 1.5 years.Main purpose of mobile phone usage for 20 participants (66.6%) was texting, browsing and selfie taking, of which 7 of them use mobile continuously for more than 2 hours.

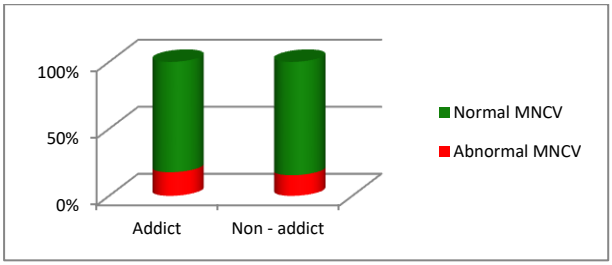
23 participants (76.6%) of the population were asymptomatic. Out of the 7 (23.3%) symptomatic patients, all 7 were having wrist pain, 4 had associated paraesthesia, 2 had positive Phalen's test. Median nerve conduction velocity was reduced in 6 (85.5%) of the 7 symptomatic.

According to SAS-SV addiction score 17(56.6%) participants were mobile addicts of which 15 (88.23%) were male. The mean SAS value of total participants was 33.5.

Of the 17 mobile addicts 12 (70.6%) had abnormal sensory nerve conduction velocity (< 50 m/s) and of the 13 non addicts 5 (38.5%) had abnormal SNCV. No significant difference in MNCV noted in the group.Of the 17 mobile addicts 3 (17.6%) had abnormal motor nerve conduction velocity (< 49 m/s) and of the 13 non addicts 2 (15.4%) had abnormal MNCV.Only 5 (16%) of total subjects were found to have abnormal CMAP



- The Chi-square statistic is 3.0964 (p = 0.078).



- The Chi-square statistic is 0.0271 (p = 0.742).`

DISCUSSION

Majority of the total participants were males and most were in the age group of 30 to 40 years with a mean of 32.5. Overwhelming majority were right handed.36.7% participants used mobile phones continuously in the past 1-1.5 year period.Main purpose of mobile phone usage for the majority was texting ,browsing and selfies.

Majority of the participants were asymptomatic. Among the symptomatic subjects wrist pain was present in all. Majority of the subjects had abnormal SNCV. Very few had abnormal MNCV.

Majority were mobile addicts as per SAS-SV. Most of the addicts than non addicts had abnormal SNCV but the Chi-square value (3.096) had an insignificant p value (p = 0.078).There was no significant difference in MNCV of addicts and non addicts.

There is reduction in conduction velocity of sensory component of median nerve in chronic mobile phone users. CMAP was found to be normal.there was no significant association with mobile phone addiction.

CONCLUSION:

In most mobile phone users there was abnormal median sensory nerve conduction velocity eventhough there was no significant association with mobile phone addiction.

Continuous mobile phone usage for long hours which involved repetitive movement of thumb, can affect median nerve.

LIMITATIONS

1.Low sample size

2.Muscle changes not included in the study
3.Confounding factors can affect results
4.Addiction tool was short version

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A Case Report on Radiation Myelopathy

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INTRODUCTION

Myelopathy is a rare but serious complication of radiotherapy. Radiation myelopathy is the white matter damage to the spinal cord developed after a certain period of ionizing radiation. Factors affecting the severity of radiation myelopathy are the dose, duration and frequency of radiation.

Dr. Aneesh Nalinakshan did his MBBS from Government Medical College, Trivandrum in the 2010 batch. He is currently doing his PG in PMR from Government Medical College, Kozhikode.

CASE DESCRIPTION

40 year old male presented with complaints of pain , progressive weakness and tightness of left lower limb of 8 months duration. He developed weakness and tightness of right lower limb of one week duration. Neurological examination showed: Grade 3 spasticity of left lower limb & Grade 2 spasticity of right lower limb. Power was Grade 0 at left Hip and knee, & Grade 1 at the ankle and EHL. Grade 3 power was noted at right hip and knee; grade 4 at ankle and EHL. DTR were exaggerated bilaterally & Plantar was extensor bilaterally. Sensory impairment from below T10 was noted. He had history of surgical resection for ependymoma located at the conus 8 months back. Post surgery there was improvement in sensory symptoms with little improvement in weakness. He underwent postoperative radiotherapy, 45Gy-25 fractions for 1 month. Six months post radiation he noticed weakness of right lower limb. He had reduced frequency of micturition with leaks during straining. BCR was sluggish, anal tone reduced,VAC was present.

IMAGING

MRI Spine: Conus at L1, distal aspect of cord(from D8-D12) appears bulky. On DWI the area shows diffusion restriction likely cord ischemia. Altered signal intensity noted involving cord in D5-D7 vertebral level and at conus, no evidence of diffusion restriction likely cord edema.

COURSE IN THE HOSPITAL

The patient was treated with high dose methyl prednisolone 1g/day for 5 days followed by oral methylprednisolone 40mg/ day for a week, tapered over 3 weeks. Injection LMWH 40mg subcutaneous twice daily for 5 days was given and thereafter the patient was put on Aspirin 75 mg OD. Simultaneously rehabilitative measures given .After the administration of steroids patient got significant improvement in power and sensory symptoms. Power of left lower limb improved to 2/5,power of right hip 4/5,knee ,ankle and EHL 5/5.

DISCUSSION

Myelopathy is a rare but serious complication of radiotherapy. It is characterized by demyelination depending on the white matter damage caused by ionizing radiation after a certain latent period. Most commonly affected structures are myelinated fibres and blood vessels. Oligodendrocytes and endothelium have been interpreted as the major target cells of radiation myelopathy. According to glial theory radiation induces damage in oligodendrocytes and its progenitor cells resulting in myelin breakdown and destruction of white matter. According to vascular hypothesis vascular injury secondary to irradiation causes circulation disturbances in the spinal cord and induces white matter lesion. Atrophy and necrosis can also develop in the spinal cord depending on vasculopathy. As damage in radiation myelopathy is irreversible treatment is usually supportive .Some patients may benefit from steroids and anticoagulation therapy. In this case significant clinical improvement was observed with steroid and anticoagulation therapy.

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Study of Acceptance of Wheelchair and Awareness about Advanced Wheelchairs among Paraplegics and Quadriplegics

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INTRODUCTION

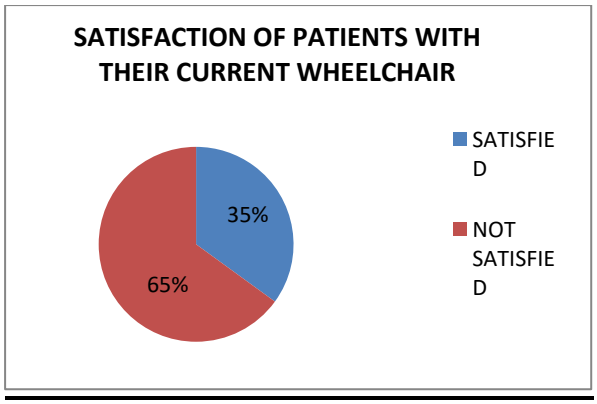
Freedom of movement is an essential component of human independence. Wheelchair and its components are fundamental for altering the interaction of a person with a mobility limitation has with the environment. Quality of life of an individual is reflective of the overall effectiveness of the wheelchair and seating system when considering activities of daily living. Enquiring about the patients history with wheelchair and seating technology can provide an indication of what has and has not worked for the patient. Understanding wheelchair users satisfaction levels may help the healthcare professionals to increase wheelchair users performance in the activities of everyday living, facilitate social and community participation and increase indoor and outdoor mobility

Masna Majeed did her MBBS in the 55th batch from Calicut Medical College. She is a second year Junior Resident in the Department of PMR, at Calicut Medical College. She won the gold medal for paper presentation in the IAPMR National Midterm CME.

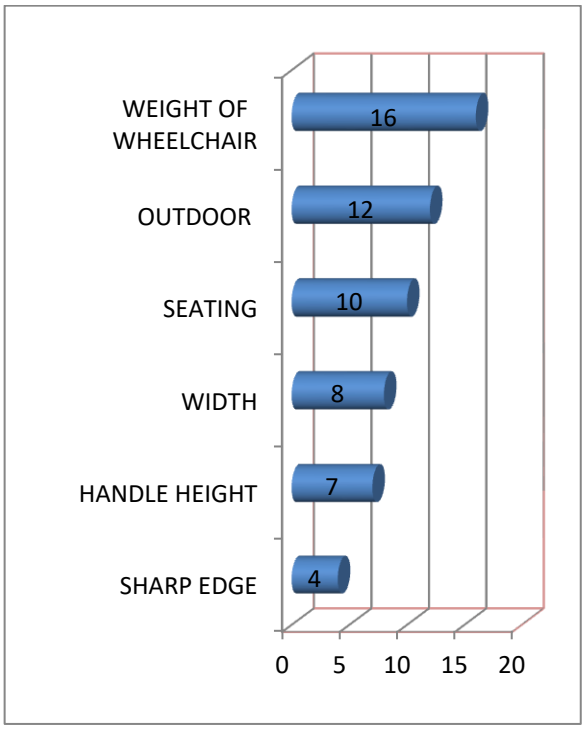
STUDY DESIGN

Cross Sectional . 20 spinal cord injury patients who were using wheelchair attending PMR OPD and ward were given prepared questionnaire. Data was assessed and quantified.

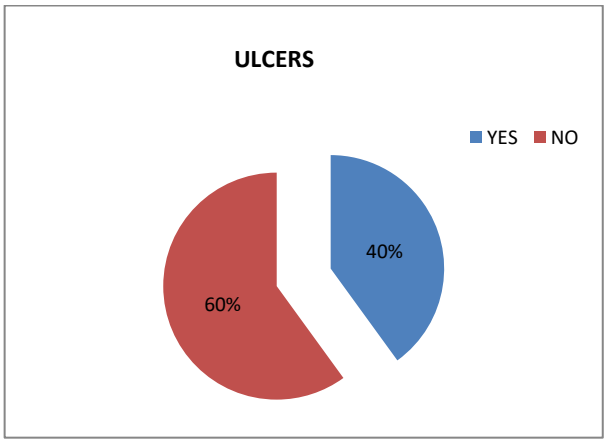
RESULTS



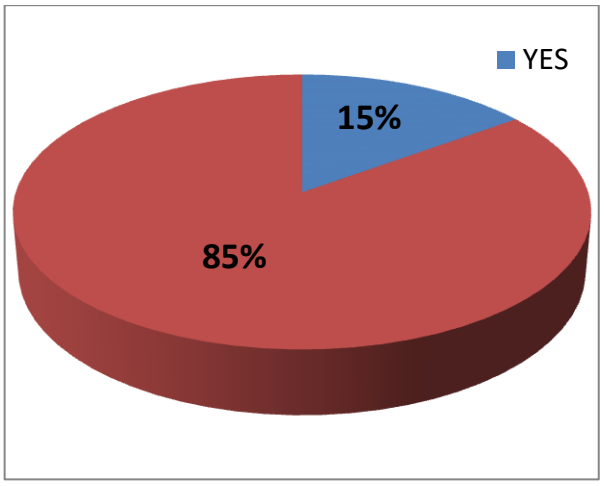
DIFFICULTIES FELT WHILE USING CURRENT WHEELCHAIR



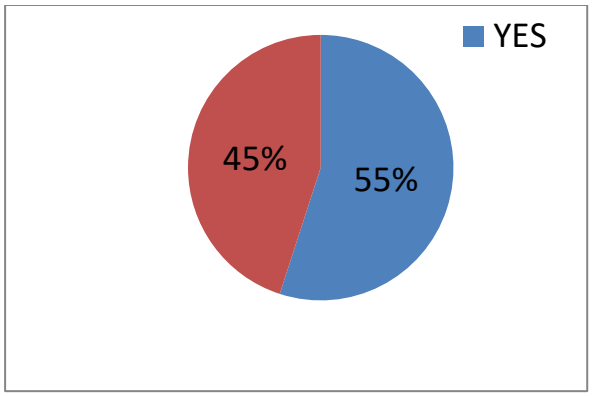
PRESSURE SORE AND OTHER ULCERS DUE TO WHEELCHAIR USE



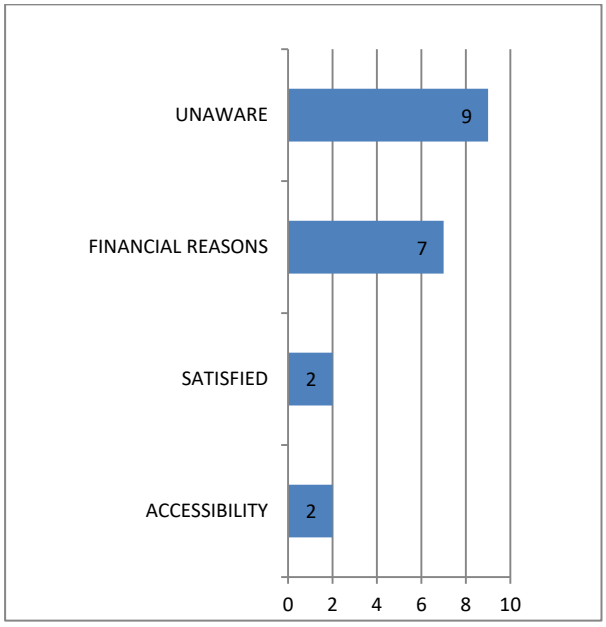
AWARENESS OF PRESSURE RELIEF TECHNIQUES



PSYCHOLOGICAL BARRIER IN USING WHEELCHAIR



WHY NOT A BETTER WHEELCHAIR?



ANALYSIS

Out of the 20 patients, 13 were unsatisfied with their current wheel chair.Out of the difficulties, weight of the wheelchair was the problem with majority. Most of the patients are unaware of pressure relief techniques and 40% people developed pressure ulcer due to improper usage. Also unawareness of advanced wheelchairs and financial limitations stopped them from getting an appropriate wheelchair. This study shows there is a great need for proper awareness and training for wheelchair among spinal cord injury patients.

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