Journal of Regenerative Biology and Medicine

ISSN: 2582-385X
Maciejewicz B, 2022- J Regn Bio Med
Review Article

Clinical Neurophysiology in the Prognostic and Diagnostic Evaluation of Disorders of Consciousness

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Abstract

According to its neurophysiological function, a disorder of consciousness (DOC) is a long-lasting change in the consciousness state of a patient that can be categorized as a coma, vegetative state, locked-in syndrome, or minimally conscious state. Although recent advances in neuroimaging and electroencephalography may help us better understand the brain networks involved in states of awareness and consciousness, the pathophysiology of DOC remains poorly understood. The main objectives of DOC rehabilitation programs are to maintain live support, to reduce the likelihood of a comatose patient developing new medical conditions, and to provide the affected families with the relevant information to make informed choices as to whether to continue the live-supporting programs. Therapeutic interventions can include both pharmacologic and nonpharmacologic therapies although at present there are no definite medical treatments for people with DOC. This research investigates and recommends numerous diagnostic methods and treatments that can be used to identify cognitive and neurobiological impaired states of consciousness, including coma, persistent vegetative state (PVS), also referred to as "unresponsive wakefulness syndrome" and other severe forms of awareness impairments, often brought on by acute brain injury but also other toxic or metabolic causes.

Keywords: Neuroscience of consciousness; Neurobiology; Coma; Minimally conscious state; Unresponsive wakefulness syndrome; Disorders of consciousness.

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Received Date: 07-14-2022

Accepted Date: 08-22-2022

Published Date: 09-14-2022

Introduction

After sustaining a moderate to severe traumatic brain injury, certain patients will experience profound and long-lasting consciousness impairments. In addition to other types of treatment and therapy used in traumatic brain injury neuro-rehabilitation, they often need treatments to help them regain states of awareness. Their

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DOI: https://doi.org/10.37191/Mapsci-2582-385X-4(4)-117

rehabilitation needs may be different from those of other patient groups. The primary objective of patients with DOC is generally considered to be the prevention of other, interconnected medical and neurological consequences, as there is currently no pharmaceutical treatment that has been shown to accelerate or improve recovery from consciousness disorders. Due to the difficulty in identifying diagnostic codes and the transient nature of certain consciousness diseases, there is no official registry of patients with consciousness disorders. Despite impairments in multiple brain regions, such as deficiencies in cortico-thalamic network activation, these patients frequently exhibit no other neurological symptoms.

Acute care hospitals, in-patient rehabilitation centers, and community or nursing homes are some of the care settings for patients with DOC. The facilities and medical staff trained in DOC care usually employ a multidisciplinary approach that involves the patient's family. The link between simultaneous wakefulness and awareness is broken in DOC and is distinct for each form of consciousness deficit, including coma, vegetative state, or minimally conscious state. Variable levels of consciousness and wakefulness are associated with each of these disorders of consciousness. Coma is considered the most dangerous of the four states because it is often characterized by a lack of consciousness and wakefulness.

The vegetative state, in which awareness is absent but moderate wakefulness is believed to occur, is the next state on the absence of consciousness spectrum. The minimally conscious state follows the vegetative state and is distinguished by partial awareness and a low level of wakefulness. Coma patients are unconscious, unaware of their surroundings, and unable to respond to stimuli. Depending on the location of the accompanying brain injury, the individual may awaken from a coma or develop a vegetative state. A coma patient requires complex and extensive care, some of which may include:

- A postural management program can prevent deformities, contractures, and pressure ulcers via posture, splinting, mobilization, and alternative seating arrangements.
- Bladder and bowel management
- Respiratory care includes secretion management (e.g. suctioning, tracheostomy management)
- Percutaneous endoscopic gastrostomy (PEG) feeding
- Management of infections like urinary tract infections
- Management or prevention of medical and neurological complications like seizures.

Methodology

Qualitative data was the primary type of information employed in this study of consciousness disorders. The research and in-depth data came from doctors and nurses being primary care physicians and primary care nurses for patients with consciousness disorders, reviewed after the patients have been evaluated.

In addition, the data was obtained from DOC cases relating to changes in the subject's conscious state, including interviews with subjects who had emerged from unconsciousness and family members

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relating changes and progressions of such states of awareness and their implications.

Unconsciousness and inability to respond are hallmarks of the coma state [1]. There are many different factors that can cause coma, ranging from minor metabolic abnormalities to extremely serious and life-threatening mass brain lesions. While it is possible for a coma to last for an extended period of time, the condition can also be temporary, and after a brief loss of consciousness, a full recovery can be achieved. The alerting and arousal mechanisms, consciousness, and conscious content of the brain are all affected [2].

Despite the fact that the COD terminology has explicit definitions in the medical literature, these definitions are frequently applied incorrectly, which leads to misunderstandings. The term obtundation is used to describe decreased sensitivity to the environment and muted awareness. Stupor is a more severe state from which a strong stimulus can only briefly rouse the patient. Instead of using potentially ambiguous phrases, healthcare professionals could ascertain a patient's reaction to stimulation and communicate that reaction instead. Coma grading systems may provide a simpler way to monitor a patient's level of consciousness over time and may be able to identify trends.

The clinician's first priorities are to stabilize the unconscious patient and identify and treat any reversible causes of coma, such as hypoglycemia. The subsequent step in the examination procedure is to determine the most likely cause of the coma and to perform the initial sorting of structural and nonstructural potential causes affecting the central nervous system, including neuronal malfunction [3]. A coma may be the result of

Research

widespread brain damage caused by metabolic or viral factors. Common toxic or metabolic causes of coma can include hypoglycemia, hyperglycemia, excessive alcohol consumption, drug overdose, and illegal drug use, to name a few. Although they are less common, meningitis and encephalitis are primary infections of the central nervous system that could induce comas. Mental status changes or comas may be caused by structural brain conditions like subdural or epidural traumatic hematomas, spontaneous intracranial hemorrhages, venous thrombosis, tumors, acute hydrocephalus, elevated intracranial pressure, anoxic brain injury, or brainstem strokes.

A mild or nonconvulsive form of generalized status epilepticus, also known as transformed or end-stage status epilepticus, may develop when unresponsiveness predominates the clinical picture and there is little to no concurrent motor activity. Due to the increased use of electroencephalogram (EEG) monitoring in intensive care units, patients are also being found to have very abnormal electroencephalographic readings that reflect nonconvulsive status epilepticus in patients who are clinically free of generalized seizures.

Retrospective reviews are difficult since there are numerous terminologies used to describe COD conditions, which leads to numerous coding alternatives. Without particular coding for coma, diagnostic coding typically just describes the cause of the altered mental status. The fact that hypoglycemia, a major cause of coma, is frequently treated by emergency medical service providers and resolved before the patient leaves the emergency room, contributes to the

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described coding lack of unification. Another functional definition some neuroscientists use involves the neuronal malfunction brought on by a reduction in the brain's supply of glucose or oxygen, which is the acknowledged pathophysiology of a coma state. A diverse array of etiologies can lead to disruption of the essential substrate, with coma as the most severe clinical condition and diffuse CNS dysfunction as the most severe outcome. For example, any clinical condition that leads to circulatory collapse or severe hypoxia can manifest as a coma. A circulatory collapse lasting as little as fifteen seconds can induce unconsciousness. If the cause of the circulatory collapse is minor and easily treated, such as a simple faint, consciousness is restored. However, the impaired state of consciousness may persist if hypotension or hypoxemia pose a risk of subsequent CNS injury. Increased intracranial pressure may also induce coma. The brain is protected by the closed skull and conditions that increase intracranial pressure may result in decreased cerebral perfusion. Cerebral perfusion pressure (CPP)=MAP (mean arterial pressure estimated as 1/3 systolic BP [blood pressure] +2/3 diastolic BP)-ICP is the equation that roughly represents this relationship (intracranial pressure). When ICP approaches MAP, cerebral perfusion declines. CPP is kept stable by lowering elevated intracranial pressure and avoiding hypotension [5,6].

When assessing a comatose patient, the fundamentals of emergency care-airway, breathing, and circulation-must be taken into account. During the initial physical examination for COD patients, the physical examination is geared toward looking for any evidence of trauma. The patient's reaction to pain, as well as their ability to open their eyes and speak, should be assessed during the initial neurologic evaluation. In a cranial nerve examination, extraocular movements, pupillary, corneal, cough, and gag reflexes should be evaluated. In the uncal herniation syndrome, which is indicated by the presence of ipsilateral pupil dilation, the third cranial nerve may be compressed by a mass [7]. The recording of the neurologic examination can be roughly quantified using the Glasgow Coma Scale, the four score, or other rating systems [8,9]. Although they have some disadvantages, brief rating scales are advantageous for sequentially monitoring the patient [10,12].

Such a scale is proposed as follows:

Eye response

4=eyes open spontaneously

3=eye-opening to verbal command

2=eye-opening to pain

1=no eye-opening

Motor response

6=react to commands

5=localizing pain

4=withdrawal from pain

3=flexion response to pain

2=extension response to pain

1=no motor response

Verbal response

5=oriented

4=confused

3=inaccurate words

2=incomprehensible sounds

1=no verbal response

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Four Score scale is recommended as follows:

Eye response

4=eyelids open or opened, tracking, or blinking to command

3=eyelids open but not tracking

2=eyelids closed but open to a loud voice

1=eyelids closed but open to pain

0=eyelids remain closed with pain

Motor response

4=thumbs-up, fist, or peace sign

3=localizing to pain

2=flexion response to pain

1=extension response to pain

0=no response to pain or generalized myoclonic status

Brainstem reflexes

4=pupil and corneal reflexes present

3=one pupil wide and fixed

2=pupil or corneal reflexes absent

1=pupil and corneal reflexes absent

0=absent pupil, corneal, and cough reflex

Respiration

4=not intubated, regular breathing pattern

3=not intubated, Cheyne-Stokes breathing pattern

2=not intubated, irregular breathing

1=breaths above the ventilator rate

0=breaths at ventilator rate or below

For all patients with impaired mental status, a point-of-care empiric glucose administration test is advised in order to determine the serum glucose level. Patients who exhibit symptoms of the narcotic toxidrome, such as slowed respiration, constricted pupils, and altered mental status, are occasionally administered naloxone. If a treatment plan to reverse the cause of coma cannot be determined based on the patient's medical history, physical examination, and statistical test results, neuroimaging is strongly encouraged [13].

Furthermore, patients suspected of malnutrition due to alcoholism, those who have had bariatric surgery, or those with persistent malabsorptive conditions were given thiamine [14]. One of the aspects of the management care strategy is to maintain the mean arterial pressure while maintaining the cerebral perfusion pressure. It is vital to continue providing supportive care while maintaining blood pressure and protecting the airways. If clinical symptoms or imaging indicate elevated intracranial pressure, hyperosmolar treatment could be considered in conjunction with further monitoring [15].

Patients with reversible causes of coma, such as hypoglycemia, may be discharged from the hospital after receiving the required treatment and determining if they have sufficient home care. Chronic coma patients require hospitalization for continuous monitoring, supportive care, and specialized treatment for the underlying cause of the coma. Patients with comas due to traumatic causes will require observation, necessary procedures for mass lesions, and supportive care to prevent further damage. The etiology of coma and the level of awareness at the time of admission are both important factors in the prognosis of patients with non-traumatic coma. Patients with a Glasgow Coma Score (GCS) of 3 to 6 at presentation had a higher hospital mortality rate than those with a GCS

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of 7 to 10 [16]. Multiple organ systems may require care during the initial treatment of these patients; therefore, a multidisciplinary team approach is essential. One European system has developed a "coma alarm" to allow a diverse team of specialists to begin their work immediately, including in the intensive care unit (ICU) since many coma patients require breathing assistance. Depending on the etiology, the short-term prognosis can range from favorable to poor, but full recovery can take months or years. The prognosis is influenced by the duration and severity of the coma.

The persistent vegetative state (PVS), also referred to as "unresponsive wakefulness syndrome," is a severe form of awareness impairment, often brought on by acute brain injury. The risk of misdiagnosis of the minimally conscious state (MCS), from which PVS can be challenging to distinguish, has been estimated to happen in 43% of cases. In contrast, brain death and PVS are not confused.

After an acute brain injury, a severe impairment of consciousness typically manifests as coma, a condition in which the patient does not open his or her eyes even in response to intense stimulation and displays no signs of psychologically interpretable contact with the outside world [17]. In patients with severe brain damage, coma can transform into a misleadingly wakeful state in which the patient's eyelids occasionally open, but there is no evidence of consciousness or the ability to communicate. Recently, the term "unresponsive wakefulness syndrome" has been used to refer to this alleged persistent vegetative state (PVS) [18].

A patient is no longer considered to be in a vegetative state and is instead considered to be in a minimally conscious state (MCS) when they exhibit any consistent behavior that demonstrates conscious perception of their surroundings [19]. Typical results of MCS include basic, non-reflexive behavior patterns such as fixation and pursuit movements (examined using a mirror; referred to as "MCS minus") or the ability to follow simple instructions (referred to as "MCS plus").

Accurately determining a patient's state of consciousness, and distinguishing PVS from MCS in particular, remains a significant challenge in routine clinical care. A rate of misdiagnosis in this field has been estimated to range between 37% and 44%; that is, many individuals are incorrectly diagnosed with other neurological conditions, whereas a thorough investigation would reveal abundant evidence of MCS [20]. Frequent clinical misdiagnosis is a serious problem because it can lead to the premature termination of life-sustaining care and can even cause emotional harm to patients who are barely conscious due to the lack of individualized care that results when their condition is misidentified.

The Glasgow Coma Scale (GCS) may not be able to distinguish between PVS and MCS clearly or practically [21]. The revised Coma Recovery Scale (CRS-R), a widely used tool for this purpose, allows for a clearer distinction between PVS and MCS. Its scale ranges from 0 (deepest possible coma) to 23 points (awake and fully capable of contact) [22].

Due to the prevalence of clinical misdiagnosis, efforts to develop technical methods for identifying individuals in a minimally conscious state have intensified. Every diagnostic test's sensitivity and specificity can only be assessed in comparison to a gold standard, which must be made clear

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right away. As a result, our evaluation of diagnostic techniques is limited by this fundamental methodological challenge. A classical clinical test such as when a patient is asked to "follow my finger with your eyes" shows that if there is no movement, it is assumed that the patient is unconscious and is therefore in a vegetative state. A classic clinical test, such as asking a patient to "follow my finger with your eyes," demonstrates that if there is no movement, the patient is presumed to be unconscious and therefore in a vegetative state. Nonetheless, it is still conceivable that a patient could be aware but unable to engage their motor system to comply (clearly) with the task. So, when we try to evaluate a diagnostic method, we run into the issue of not being able to tell for sure whether a test result is actually negative or not (or positive). When attempting to evaluate a diagnostic method, we are therefore unable to determine with certainty whether a test result is negative or positive. Therefore, the classic visual fixation test alone cannot serve as proof of loss of awareness.

Currently, the clinical standard used (CRS-R) may not be sensitive enough to detect all individuals with MCS, making the distinction between PVS and MCS difficult. According to recent research, 10 to 24 percent of patients who are unequivocally determined to be in a persistent vegetative state on clinical grounds (and who do not fall under the category of the 37 to 43 percent of patients who were given a incorrect clinical diagnosis) actually demonstrate clear signs of understanding and being able to follow instructions as detected in their fMRI or EEG readings [24]. When given instructions to envision doing certain movements (motor imagery), such as "Imagine you are on a tennis court and are hitting the ball," the fMRI can be used to identify patients in whom the same task-specific brain areas are activated as in normal control subjects [25].

Using EEG-based imaging techniques, similar results have been obtained. For example, a high-resolution EEG (HD-EEG; 64-256 electrodes) can be obtained by instructing the patient to clench his or her fist or move his or her toes in response to a specific cue. Following that, the EEG signal is examined for variations in bioelectric brain activity that occur frequently in a relevant brain area (for example, in the motor hand area, in the instance of making a fist) at the appropriate temporal interval after the signal. 10% to 24% of the PVS patients were able to complete the task in both the fMRI-based and EEG-based trials. Consequently, despite the fact that these patients had received an accurate diagnosis of PVS based on accepted criteria, testing with more sophisticated techniques and methods revealed that they maintained a significant and now detectable level of consciousness despite the loss of motor functions [26].

Both of these procedures are still being debated because they are methodologically quite demanding, especially given the statistical analysis and interpretations involved [27]. How can one determine if the new techniques provide genuine evidence of sustained consciousness, as opposed to merely artifacts or incorrect brain scan interpretations? To determine the answer, it would be necessary to conduct a long-term study using fMRI and EEG on a cohort of PVS patients, and then after recovery from the condition, to ask some patients if they remember the study and the tasks they were assigned. In practice, this would be extremely

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difficult because nearly all PVS or MCS patients who recover have long-term amnesia for the period during which their consciousness was compromised [28].

Nonetheless, there is substantial evidence that these techniques are effective. One PVS patient participating in fMRI research was able to correctly answer five out of six autobiographical questions by visualizing himself playing tennis for "yes" answers and touring an apartment for "no" answers [28].

For a long time, the accepted theory of prognosis in PVS was that if a patient remained vegetative for 12 months after sustaining a catastrophic brain injury or 3 months after sustaining non-traumatic brain damage, such as hypoxic brain damage, there was no reasonable chance that they would ever regain consciousness [29]. Recent research indicates that this criterion should be modified slightly, but in general, a PVS with a traumatic etiology has a better prognosis than one without. In one study, 50 patients with at least six months of persistent vegetative state were followed prospectively for two years. 10% of patients regained some level of consciousness, while 14% regained more [30]. The majority of patients who recovered from a vegetative state and regained the ability to communicate did so after more than a year had passed. This was true regardless of whether or not the underlying brain injury was trauma-related.

Independent of traumatic causes, younger age is also associated with a better prognosis for acclimating from a vegetative state to minimal consciousness. In the aforementioned study, those with a better prognosis were significantly younger than those who remained in a vegetative state (age 32 versus 54 versus 12 years old). However, sadly, all patients in that study group who regained consciousness endured severe functional limitations and required ongoing nursing care [31]. Currently, physicians assessing the prognosis of patients who are unconscious after an acute brain injury use a variety of "good prognosis" criteria in their evaluations. The spectrum ranges from a simple return to consciousness to total independence with mild disability. From the perspective of some doctors, policymakers, and family members, anything less than restoring an independent lifestyle may not be an acceptable state of being. Nevertheless, some severely physically handicapped and bedridden patients who have regained just the ability to communicate as described above assessed that their quality of life has improved [32,33].

Extreme recovery cases

Case: JW

The chances of surviving a severe disorder of consciousness such as a coma are low, but as was previously stated, there is still a chance that someone will recover unexpectedly and against all odds. JW, a 18-year-old victim of a car accident, is an example of such one case [34].

The 18-year-old patient, JW, suffered a severe brain injury as a result of an automobile accident. He displayed a GCS score of 4 at the scene of the collision, as well as large, fixed pupils and decerebrate rigidity in all four extremities. At the scene of the collision, he displayed a GCS score of 4, as well as large, fixed pupils and decerebrate rigidity in all four extremities. The patient was residing in the intensive care unit of Germany's "Universitätsklinikum Würzburg." CT scans revealed the presence of extensive cerebral

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edema and midline shift subdural hemorrhage. For the purpose of reducing cerebral pressure, an emergency decompression craniotomy was performed. Following surgery, JW continued to have fixed pupils, no corneal reflex, and no motor response to pain. The family was informed that brain damage was fatal and that survival was extremely unlikely. However, the family chose to continue the patient's life support. After 12 days in the intensive care unit, the case was presented with the following:

- Coma
- Acute subdural hematoma, right side
- Brain swelling
- Artificial respiration
- Abnormal pupillary functions
- Severe spastic tetraplegia
- Tracheal cannula
- Percutaneous gastrostomy
- Decerebrate rigidity of all extremities

1st month

The patient remained on a ventilator during the first month of rehabilitation and had autonomic instability, including tachycardia, and hypertension. No change in the mental state was observed.

2nd month

The EEG revealed a significant slowing and a predominance of delta-theta waves in the second month. Magnetic resonance imaging (MRI) revealed a right-frontal subdural hematoma, an asymmetric, right dominant ventricle system enlargement without midline displacement, parenchymal lesions with gliosis in the temporal regions, and white matter surrounding the posterior horn of the lateral ventricle.

JW was able to be weaned off of ventilation by the end of the second month, despite his cognitive disabilities remaining unchanged.

3rd month

By the third month, the patient was MCS-positive. However, there were no vocalizations other than groans in response to painful stimulation. No communication or command execution channels were able to be established.

4th month

After 4 months, a new MRI revealed significant brain atrophy, numerous parenchymal lesions, and diffuse shear lesions, but no periventricular cerebrospinal fluid transudation.

5th month

The skull was scheduled to be operated on five months after the accident. After the operation, additional complications arose, including an epidural hematoma that required additional surgery. There was no obvious improvement in clinical status.

7th month

JW regained the ability to scream in response to different stimuli after seven months. For more than four weeks, this continued. During his "awakening" moments, he yelled, and painkillers and other attempts at medication had little effect. However, JW 's EEG now exhibited a nearly typical alpha rhythm. Even though they were delayed in latency, the event-related potentials (ERPs) for tone discrimination and semantic speech processing could be easily distinguished.

8th month

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JW began closing his eyes on command eight months after the incident, clinically indicating progression to MCS+. Nine months after the incident, the patient made eye contact by blinking, which was then also followed by head nodding and shaking. It was presumed that the patient had progressed beyond MCS because communication was deemed to be increasingly adequate. After nine months of intensive care, he was at this point discharged for early rehabilitation.

Home recovery

JW resumed his intensive therapy regimen at home, with weekly sessions of two hours of occupational therapy, three hours of physical therapy, and two hours of speech therapy. After six months at home, the patient's body control had improved sufficiently for him to walk approximately 20 meters at a time, with noted difficulties. Despite continuing to experience aphasic symptoms, he regained the ability to speak and communicate effectively. Epileptic seizures continue to occur approximately once per month.

After six months at home, the patient was readmitted to the hospital for ten weeks in the rehabilitation center (Kliniken Schmieder, Germany) to improve his walking ability, memory, and overall independence. JW. received a neuropsychological evaluation at this time, one and a half years after the accident. He scored well on verbal intelligence, short-term memory, and executive function. In spite of this, he struggled to remember some words and geometrical figures, showed a below-average long-term memory, and processed information more slowly overall. Years after the fatal injury, he kept up with his therapeutic routine and kept rehabilitating.

Ten years after the incident, JW can now walk, use stairs, and even ride his bicycle without assistance. He is now completely independent in all aspects of daily life, including maintaining a job. Despite his persistent memory impairment, he has developed coping mechanisms. Despite his speech delay, he has a standard vocabulary and accent.

Conclusion

Instances in which patients were incorrectly diagnosed as brain-dead, as nearly occurred with the patient described above, posed significant medical and psychological challenges for physicians and patients' families. Due to a misdiagnosis of their state of consciousness, a patient in a coma or vegetative state has been known to perceive sensations, hear conversations between family members and medical personnel, and even feel every incision and every touch during surgery. Hearing discussions and suggestions to end their lives at their bedside, as well as experiencing excruciating pain and being unable to communicate it, subject these patients to psychological, emotional, and physical torture. Thus, progressing research on improving diagnostic tools and methods for disorders of consciousness patients remains both important and urgent.

Indeed, rapid technological advancements over the past two decades have resulted in the development of novel techniques for studying the structure and function of the human brain in real time. Today, a comprehensive picture of normal and abnormal brain function can be created by combining detailed anatomical images from computerized tomography (CT) and magnetic resonance imaging (MRI) with PET, functional magnetic resonance imaging (fMRI), quantitative electroencephalography

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(EEG), and magnetoencephalography (MEG). Functional neuroimaging has consequently emerged as the method of preference for neuropsychologists, cognitive neuroscientists, and researchers in the larger neuroscientific community with a focus on the connection between the brain, behavior, and consciousness.

These methods of research have mostly been applied as a correlational tool to map the brain's network connections to certain cognitive activities or functions, be it an action, a reaction (for example, to some type of external stimulus), or a thought process. Recent advances in imaging technology, specifically the ability of fMRI to reliably identify individual participants' neural responses in real time, are beginning to make it possible to infer a participant's thoughts or intentions based solely on the pattern of brain activity observed. The case of the vegetative patient discussed previously is a clear illustration of such an application [35,36]. Observing the patient's time-correlated and sustained fMRI responses in response to commands to perform specific mental activities was the only way to determine that the patient was consciously aware in the absence of any other motor functions on their part. On the basis of this, it was feasible to deduce (within the limitations of the cues assigned to them) not only that they were aware and thinking but also what they were thinking about during that assessment.

Conflict of interests

The authors declare no conflict of interest.

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Citation: Maciejewicz B. Clinical Neurophysiology in the Prognostic and Diagnostic Evaluation of Disorders of Consciousness. J Regen Biol Med. 2022; 4(4)1-12.
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