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Approaches of the intensive neurofunctional physiotherapy: A short review

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Abstract
The context of the neurocritical patient is complex and the early intensive physiotherapy approach and during the hospitalization is of extreme importance to the prognostic and to evolution sensorimotor of these patients, or to prevent the relevant complications of immobility, managing the mechanical ventilation or encouraging the neuroplasticity. It is presented in this work the more common neurologic alterations and it particularities. Also, the normotensive physiotherapeutic techniques are showed to provide information about them and meliorates it provokes. The recent theories about the Bo bath method, the neuroplasticity theory, and the theory of restriction movement are showed. In conclusion the importance of the work of physiotherapist is to obtain quality of life for the patients in intensive unit.

Keywords: intensive therapy; neurofunctional physiotherapy; bo bath; neuroplasticity; quality of life

Introduction

The neurologic alterations caused by trauma or by diseases normally in a first moment need to the intensive vigilance, like this the patients are admitted in the intensive therapy unit, where a multi professional team interacts to diagnosis and treatment of the earlier form avoiding other complications.

The essential support measures to the neurocritical patient are: sedative, analgesic, positioning, ventilatory assistance, and encephalic monitoring.

The monitoring of the intracranial pressure and the computed tomography are common vigilance vectors in the neurologic complications, but other forms have been proven and utilized in the clinical practice, such as the bulb-jugular monitoring and the transcranial Doppler, turned to the analysis of the cerebral perfusion.

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More common neurologic alterations

Encephalic vascular accident (EVA)

Of the cerebrovascular diseases, the EVA is the neurologic disease that more assaults the nervous system, being the principal cause of physical and mental incapability [1]. The EVA is characterized for one interruption of blood flux to the brain, could be for arterial obstruction, ischemic EVA, or for vessel rupture, hemorrhagic EVA [2].

The nervous tissue is devoid of reserves, being totally depending of circus contribution, because thanks to this are that the nervous cells stay actives, being their metabolism depending of oxygen and glucose. In the case of interruption of this contribution, a determinant area of the brain generates diminish or stop of the functional activity of this area [3].

The cause more common of cerebral vascular accident is the obstruction of one of the important cerebral arteries (media, posterior and anterior) or of minor piercing that go to the deeper brain parts. The cerebral vascular accidents of the brainstem, provokes by disease in the cerebral and basilar arteries, are less common.

An adult brain weight about 1500g, needs of a constant supply of 150g of glucose and 72 liters of oxygen every 24 hours, not having a good reservation to stock these substances [4].

Cerebral vascular accidents types

Cerebral infarction

It Occur infarcts when a plunger or thrombus is trapped in a vessel, obstructing the blood flux. Typically, a plunger abruptly deprives an area of blood, resulting in the almost immediate appearance of disabilities. The obstruction of the blood flux in deep minor gauge artery results in lacunar infarctions [5].

Hemorragy

A Hemorragy blood deprives the vessels in front of, and the extravascular blood exerts pressure on the surrounding brain tissue. Usually, the hemorragic accidents presented with the deficient more intense hours after occurrence, it is following improvement measure that the edema regresses and the extravascular blood is moved [6].

Subarachnoid hemorragy

The bleeding to the subarachnoid space cause excruciating headache, with abrupt begins, with moment loss of consciences. Differently of the other types of hemorragy, the initial data not is, with frequency, focal. The deficiencies, resulting of subarachnoid hemorragy, are progressive, due to the continued bleeding or to the hydrocephaly. Vasospasms and infarction are common sequels of the subarachnoid hemorragy [7].

Cranioencephalic trauma - CET

The cranioencephalic trauma is defined as a cerebral aggression, no of degenerative or congenital nature, but caused for an external physic force, that can produce a decreased or altered state of conscience, that results in compromise cognitive abilities or of the physic functioning. It can also result in the emotional disturb or compartmental functioning, could be temporary or permanent and provokes total or partial functional compromise [8].

The major of the trauma lesions of the encephalic occur as result of accidents with motorized vehicles. The impact tends to damage the orbitofrontal region, the anterior or inferior parts of the temporal lobe and to cause diffuse axon damage. The axon damage reaches primary the base ganglion, top cerebellar pendulum, the corpus callosum and the midbrain. As the frontal, temporal and limbic areas are typically damage, the people present compromise of the capability of judgement, reducing of the executive functions, man sic deficiency, processing slowed down of the information, attention disturbs and compromise of the capability of complex problem solution [9].

Spindle trauma (ST)

The trauma damages of the spinal cord are, in general, caused by motorized vehicles accidents, by lesions occurred in the sports, falling, or penetrating wounds. The three types of lesion not section the spindle. On the other hand, the damage is due of crushing, hemorragy, edema and infarction. The penetrate wounds by knife or projectile section directly the spindle neurons. Immediately after the trauma damage of spinal cord, the spinal functions below of the lesion level they are depressed or lost. This condition, known as spinal chock, is due of the interruption of the descendent tracts that produce the medullary neuron ionic facilitation [10]. During the spinal chock they are compromised: somatic reflex, autonomic reflex, autonomic regulation, sweating control and of the piloerection.

Various weeks after the lesion, most of the people experiment recuperation of part of the medullary function, with return of the reflex activity below of the lesion level. In some people, the spinal neurons they are excessively excited, leading to hyperreflexia [11].

Cervical medullary lesion cause tetraplegia, with compromised of the arms functioning, of the trunk, of the legs and of the pelvic organs. People with lesions above of C4 need invasive ventilatory assistance, because the diaphragm has his innervation between C3 and C5. The paraplegia is caused by medullary lesion below de cervical level, preservation the arms function [12, 13].

Medullary lesions classification

The medullary lesions are classified according two criteria: if the lesion is complete or incomplete and when to the neurological level of the lesion. A complete lesion is defined as a lack of the sensorial and neurological functions in the inferior sacral segment. An incomplete lesion is defined as sensorial function preservation and/or motor function in the more inferior sacral segment. The neurologic level is the level more caudal with sensorial and motor functions normal bilaterally. Yet, the motor functioning could be compromised in level different of the sensorial functioning and the losses could be asymmetric. In these cases, up until four distinct neurologic segments could be descripted in a same patient: right sensorial, left sensorial, right motor and left motor [14].

Neurointensive physiotherapeutic techniques

Ventilatory assistance

The need of mechanical ventilation is indicated for patients with fall of conscience level (Glasgow below of 10) and those who that presented alteration in the intracranial pressure (ICP) [15], being the normal value between: ICP< 10 mmHg – normal; ICP between 10 and 20 mmHg – slightly increased; ICP between 21 and 40 mmHg – moderately increased; ICP > 40 mmHg – severely increased.

The ventilatory strategy walks in the control of the level of PCO_2, until that when the vascular tonus regulation is responsive, to each 1 mmHg of increase of the $PaCO_2$ propitious an increase of 4% in the cerebral blood flux and consequently causes an increase of the ICP.

The called hyperventilation of prophylaxis is utilized in the first 24 hours, objectifying: maintenance of $PaCO_2$ around 28 to 33 mmHg, levels that margin the vasodilatation (increase) and vasospasm (decrease). The action desire is reached in the respiratory frequency addiction; surveillance about the relation inspiratory/expiratory and auto-PEEP, could be changed by anterior item; Surveillance of the intrapulmonary pressure, so that not alter the intracranial pressure (ICP); Head positioned in the medium line and headboard to 30º, favouring the cerebral venous return, beyond other complications as the bronco aspiration [16]. This strategy objective the maintenance of the cerebral perfusion pressure (CPP) = medium arterial pressure (MAP) – intracranial pressure (ICP). Other care during the mechanical ventilation should come back to the moment of aspiration of the airways, this must be criterions and indicated when necessary, should be think together the team the indication of neuromuscular blockers or changed in sedation.

The actual literature argues the not need of viewer as protocol to all patients, should this proceeding be judged of criterions with finality of avoid other clinical alterations due of this strategy. In an initial moment the great preoccupation is avoid complications due of the base problem, therefore, the preoccupation of the physiotherapist if it gives in the managing of ventilatory assistance if it is indicated or of the oxygen therapy and adequate bed positioning [16-19].

The neuroplasticity and the functional movements are study centre when we show the treatment progress, after the initial phase serving as base to application of physiotherapeutic techniques.

Neuroplasticity

The plasticity concept not is unique, although it will be older in the scientific literature, there is not unification theory of the neuroplasticity phenomena, the experimental approaches are multiple and the results oftentimes conflicting. It is considered as the nervous system tendency and to adjust environmental influences during the development, and to establish or to restore disorganized functions by experimental or pathologic conditions [20-23].

It can observe a link of plastic phenomena with the autogenetic development of nervous system, like this as the compensatory answer capability to lesions and other external influences, therefore, we can face the neuroplasticity in various angles, depending of the approach that if you do (morphological, physiological or psychophysical) and of how each mode react: motricity, perception and language.

The literature show works when considered doted of great plasticity the embryonic brains or neonate [24], if compared with the mature individuals' brains. The confirmation of relative loss of plasticity of the nervous system of adult's mammals it took to a great interest about the relation between the plastic phenomena and those that determine the normal ontogenetic development of the nervous system.

In the morphological plane, a lot of works of cellular alterations, dendritic and principally axonal, it has been descripted, being development many theories to explain how the nervous system submitted to destructive lesions it is capable, in determinate conditions, of to recover functions previously lost.

Hierarchy Representation Theory: each functional group of nervous system would be organized vertically in ascending levels of increase complexity. Like this, the levels, immediately inferiors to one lesion site assumed, although imperfectly. Good theory when applied to the motors system, but relatively week to explain plastic phenomena in the superior system sensorial and functional [25].

Delectable Theory: one focal lesion destructive of the nervous system provokes, besides functional loss by define destruction region, it will have a specie of chock or functional depress (delectable) in regions related to lesion zone. The deficit observed right after the lesion it will be the association of the symptoms provokes by tissular destruction with that provokes by delectable, however this would disappear, returning to the normal the functioning of the depress regions [26].

Doctrine of substitutionism: propose that before the lesion other regions of the nervous system assumed the altered function, "replacing" the destroyed region, when the more important factor that determines the temporal evolution of resulting deficits of lesions it will be "the quantity" of hit tissue, soon, how much minor the lesion, more easy the reminiscent regions could to assume the hit function [27].

A lot of suggestions were proposal, including the orientation of the fibres in growing according the straight lines of electoral fields, or according the lines of mechanical tension coming of the grown and nervous system folds, or still according chemical gradient of substances capable of "to attract" the growing cones of the axons in direction to their targets. In second place comes up the question of how is stablished the precise topographic relations between the pre-synaptic population and the respective post-synaptic targets [28]. In this case, the postulation of some factors that operate in this construction of the coupling must the evidences obtained of plastic systems, as the competition by post-synaptic sites, when the two populations of growing axons establish competitive interactions by "conquer" of the available terminal space; "Compensating bud", in this case, each axon had a volume of arborization pre-terminal, when a part this arborization is destroyed, the loss is compensated by buds that occur in other points of the growing axons, could be disorganized by mechanical disturbs of the target tissue [29].

It know that with the neural plasticity can stablish the lost part function and/or lesioned, being the microscope prosses that take part of the neural plasticity: the priority inhibition of the connections immediately before the lesion and local budding of both involved axons and neighbouring axons, that take to the formation of new synapses correct and incorrect, this mechanism of repair or budding of axons occur principally the sensitive-motors nerves [30].

Other points latter can be emphasized in these patients as the increase of the respiratory capability that normally is deficient, by the situation of immobility in the bed or by alteration in the muscular information.

Therapy of restriction and induction to the movement (TRIM)

The function recuperation of the superior members, promote by plasticity, is difficulty by a phenomena known as "not use learned". With the loss of the function of one area of the brain target by VCA, the region of the body that it was close to this area is affected lost its capability of movement. As the patient not move the affected member, it compensate using other, like this, before certain time, when the effects of the lesion not be more presents and occurred re-adaptations in the brain, the movements could be recovered, while, the patient "learned" that this member not is more functional.

The therapy of restriction and Induction to the movement (TRIM) is a technique that involves repetitive training of motor activities, that it can be better the function and the use of the hemiparetic superior extremity resulted of a chronic, acute or subacute VCA, it was it is believed that this improvement occurs through two separate but closely linked mechanisms: overcoming of the "not use learned" and induction of an use-dependent cortical reorganization.

The objectives of the TRIM are based on researches previously made in primates, whose the somatic sensations of a one superior extremity were surgery abolished through dorsal rhizotomy. The monkeys cessed the use of affected superior extremity immediately before the deafening and never recover exponentially their use. However, the use of the member deafening can be induced both by immobilization of intact superior extremity and by training in a period of days. The result was an extensive reutilization of the superior extremity deafening persisting by the rest of life of the animal, reflecting in gain motors in the functional activities as in the nutrition.

The somatosensory deafening and the VCA obviously involve different types of lesion. However, the nature of the mechanism involved in "not use the affected superior extremity" is one that this comes in function always there is damage to the nervous system, resulting in an initial amplified deficit followed by a prolonged recuperation period. As the patient or the animal before the VCA utilized compensatory strategies, to realize activities of diary life, using, the not affected superior extremity, in the majority of their activities the intrinsic recuperation that occur is "mascaraed". Therefore, though the motor function return gradually as a result of the spontaneously and rehabilitation, the use actual of the hemiparetic superior extremity often look like much smaller that the potential use.

Bo bath concepts

It is a treatment based in the neuro evolution theory. It was much utilized in individuals with central nervous system physiopathology. It has the objective to modify the movement abnormal and tonus postural standard, facilitating more correctly movement motors standard, preparing the patient to a variety functional abilities, always realized by a specialized physiotherapist [31].

The physiotherapeutic intervention evolve a process of direct handling to the inhibition and facilitation of techniques

application, with the objective of function perfect, including the vary systems interaction [32, 33].

The neuroevolution treatment recognizes that a continuous inadequate behavior can result in increased deficiency, new deficiency and other physiopathology, such as the compromised of the cardiorespiratory function or contractures and deformities of the muscles and articulations [34, 35].

The typical motor professional knowledge and how it varies throughout life, provide the embassy to a functional evaluation and interventional planning, fits to the physiotherapist to recognize that there are standards generic of time and abilities acquisition during the development and maturation as well as the loss of some abilities in the aging. These standards provide a reference model to an efficient human motor function and enable the individuals' difference identification, normal and atypical deviations, and the inadequate motor control.

Proprioceptive neuromuscular facilitation (PNF)

The techniques of PNF trust mainly in the stimulation of the proprioceptors to increase the demand made to the neuromuscular mechanism, to obtain and simplification theirs answers. It was developed by Dr Herman Kabat e by Ms Margareth Knott, in the Kabat Kaiser Institute, between 1946 and 1951 [36].

These techniques and the treatment method in what are used aims to obtain the maximum quantity of activity that can be achieved in each volunteer effort and the major number of repetitions of this activity to facilitate the answer. The physiotherapist need of expertise in the execution of the techniques and a total understand of the treatment method to obtain the best results of the patient [37].

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