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Understanding cortical neuroplasticity in visual impairment

Compreendendo a neuroplasticidade cortical na deficiência visual

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ABSTRACT

Introduction: The continuous process of brain change, of "reorganization" of neural circuits, and the reception of new attitudes or thoughts, is what is called neuroplasticity. A better understanding of how the brain adapts to vision loss and what the functional consequences are for the remaining sensory information could result in a better quality of life for the visually impaired.

Objective: Discuss the mechanism of cortical neuroplasticity in visual impairment, and the importance of this mechanism for patients with low vision and blindness.

Method: The research was carried out by selecting articles from the PubMed and Scielo databases, covering publications in English and Portuguese. The following descriptors were used: neuroplasticity, plasticity of the visual cortex, neural plasticity in blindness, neuroplasticity in visual impairment.

Conclusion: The results of the various studies show that the great "selectivity" of the visual system for the processing of light stimuli arises mainly from the methods usually used to study this system. In the absence of vision, the visual cortex can respond to tactile and auditory stimuli, because the absence of vision allows the unmasking of latent connections, thus revealing metamodal organization.

KEYWORDS: Neuroplasticity. Neural plasticity. Visual cortex. Blindness.

RESUMO

Introdução: O processo contínuo de mudança cerebral, de "reorganização" dos circuitos neurais, e da recepção de novas atitudes ou pensamentos, é o que se chama neuroplasticidade. Um melhor entendimento de como o cérebro se adapta à perda da visão e quais são as consequências funcionais para as informações sensoriais restantes podem resultar em melhor qualidade de vida para os deficientes visuais.

Objetivos: Discorrer sobre o mecanismo de neuroplasticidade cortical na deficiência visual, e a importância deste mecanismo para pacientes com baixa visão e cegueira.

Método: A pesquisa foi realizada a partir da seleção dos artigos nas bases de dados PubMed e Scielo, abrangendo publicações em inglês e português. Utilizou-se os descritores: neuroplasticidade, plasticidade do córtex visual, plasticidade neural na cegueira, neuroplasticidade na deficiência visual.

Conclusão: Os resultados dos diversos estudos mostram que a grande "seletividade" do sistema visual para o processamento de estímulos luminosos decorre principalmente dos métodos usualmente empregados para estudar esse sistema. Na ausência de visão, o córtex visual pode responder a estímulos táteis e auditivos, porque a ausência daquela permite o desmascaramento de conexões latentes, revelando assim a organização metamodal

PALAVRAS-CHAVE: Neuroplasticidade. Plasticidade neural. Córtex visual. Cegueira.

Central Message

The word plasticity derives from the Greek "plastikos", which means moldable, and neural plasticity refers to the ability of the nervous system to reorganize during development and, in adulthood, in response to environmental challenges. It is an intrinsic property of the nervous system through which learning and compensatory changes occur after neural tissue injury. Discussing the mechanism of cortical neuroplasticity in visual impairment and raising the importance of this mechanism for those with low vision and blindness is important in order to update those involved in the subject.

Perspective

Studies on blindness and subsequent plasticity show that the extent of cortical reorganization is correlated with the age of onset of deficiency. A different pattern of cortical activation has already been identified between the early blind and those who lost their vision late. In the absence of vision, it allows the unmasking of latent connections, and the visual cortex can respond to tactile and auditory stimuli, thus revealing metamodal organization. In addition, this process generates expansion of the cortical areas involved with touch and hearing, explaining the refinement of the auditory and somesthetic functions of the blind. Understanding these physiological changes is essential to optimize the rehabilitation process after visual deprivation.

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INTRODUCTION

Vision is the sense in which sensorimotor integration is most evident, due to eye movements that project the region of greater visual acuity of the retina to points of interest in the outside world.[1] In 2002, it was estimated that more than 162 million people worldwide were visually impaired, and more than 37 million were completely blind.[2]

The word plasticity derives from the Greek "plastikos", which means moldable. Neural plasticity refers to the ability of the nervous system to reorganize during development and in adulthood in response to environmental challenges. Plasticity is an intrinsic property of the nervous system, in addition to being the mechanism by which learning and compensatory changes occur after neural tissue injury.[3]

Neuroplasticity, also known as neuronal plasticity or brain plasticity, refers to the ability of the central nervous system to adapt and shape itself to new situations. The central nervous system is an ever-changing structure, of which neuroplasticity is an integral property, and the consequence of every sensory input, motor act, association, reward signal, plan of action, or consciousness. Thus, neuroplasticity is not an occasional state, but rather the normal, continuous state of the central nervous system throughout life. It refers to the capacity of the central nervous system to undergo structural and functional reorganization in response to the environment, its afferent stimuli, and efferent demands. Neuroplasticity is expressed at all organizational levels and spans different time scales, ensuring the ability to adapt and learn throughout human life.[3,4] Neuroplasticity does not occur only in people with severe neurological damage; it occurs all the time, in all people around the world. It is an involuntary process of our body and very beneficial for our daily lives. To be able to exemplify we can take into account an experiment carried out by the scientist Pascual-Leone.[3] It consisted of blindfolding adults with healthy vision for 5 days. During this time, blindfolded people lived and acted like blind people, reading Braille and performing auditory discrimination activities. After performing magnetic resonance it was observed by the scientist that the visual cortex began to be activated by hearing and touch. The result proves that even after adulthood, our brain can adapt to changes (drastic or not) and reorganize itself based on our greatest needs.[5]

The pioneering work of important groups, such as those of Mountcastle (1957) and Hubel and Wiesel (1959)[6,7], revealed a complex pattern of ultra-specialized cortical circuits in analyzing stimuli of different sensory modalities, such as touch and vision, respectively. Recently, however, other groups have been demonstrating that this specialization is more flexible, with several cells located in primary sensory areas responding to stimuli from 2 or more sensory modalities. Although there is evidence that some of these modalities are processed in a neuro-specific manner, most of our perception of events in everyday life is recorded simultaneously by more than 1 sensory modality in an integrated and unified manner, with no apparent discontinuity, optimizing the detection and recognition of objects and also our response to them.[8] For example, when planning a movement, it is common to integrate visual, somesthetic, and auditory information to optimize the sequence of movements necessary to achieve the goal.[9]

Thus, the objective of this review was to discuss the mechanism of cortical neuroplasticity in visual impairment, describing what exists in the literature on this topic, detailing the peculiarities and controversies, and to discuss the importance of the mechanism of neuroplasticity for the rehabilitation of patients with low vision and blindness, according to data in the literature.

METHOD

The search was carried out based on the selection of articles in the PubMed and SciELO databases, covering publications in English and Portuguese. The following descriptors were used: neuroplasticity, visual cortex plasticity, neural plasticity in blindness, neuroplasticity in visual impairment, citing the existing definitions of neuroplasticity, and their correlations with visual impairment. For a better understanding, each type of neuroplasticity and its peculiarities were described. This paper presents a synopsis of the main studies described in the literature on the mechanism of neuroplasticity of the visual cortex and its implications for blindness.

DISCUSSION

Eye blindness has served as a unique model to investigate developmental neuroplasticity, helping to uncover the neurophysiological mechanisms that characterize the brain's potential to be shaped and adapt to sensory deprivation. In fact, individuals living with blindness and visual impairment typically develop compensatory behavioral strategies (i.e., rely on non-visual modalities, including hearing and touch) to gather relevant sensory information and maintain functional independence in a highly vision-dependent world.[10,11] The types of neuroplasticity mentioned in the literature are described below.[3]

Axon plasticity

This is the initial (and most fundamental) plasticity of brain development. Occurs between 0-2 years of age. Within it there is the critical period, which consists of the moment when there is more action of neuroplasticity in the central nervous system. It occurs in children, as it is the phase where there are more discoveries about life, the environment and their own body.

Dendritic plasticity

Characterized by changes in the size, length, arrangement, and density of dendritic spines, dendritic plasticity occurs mainly in the early stages of development. Dendritic spines are the "little wires" that connect and transmit information between neurons. The axon is the "tail of the neuron" and the dendrites are the "branches" that are both at the head of the neuron and at the end of the axon. They are the ones who receive and release the neurotransmitters, which make the real communication between the neurons, while the rest of the neuron passes on the message electrically.

Somatic plasticity

It refers to the ability to regulate the proliferation and death of nerve cells. This is a capacity present only in the nervous system of the embryo and is not influenced by the external environment.

Synaptic plasticity

It is equivalent to the ability of synapses to strengthen or weaken in response to external and internal stimuli.

Regenerative plasticity

It is the regeneration of affected axons. It has a greater action on the peripheral nervous system, which is responsible for connecting the central nervous system with other parts of the human body.

Neuroplasticity and visual impairment

Changes in peripheral stimulation patterns in any neural system lead to reorganization of the system's synapses, which is most evident during the critical period of development, characteristic of each modality. This plasticity is demonstrated at various levels of analysis, from the molecular to the behavioral.

Currently, plasticity is no longer considered an extraordinary state of the nervous system, but rather a latent mechanism capable of generating continuous changes throughout life.[3]

Most studies on blindness and subsequent plasticity show that the extent of cortical reorganization is correlated with the age of onset of deficiency. Several researchers have identified a different pattern of cortical activation between the early blind and those who lost their vision late. Cohen et al.[10] reported activity in the visual cortex in tactile discriminative tasks in congenitally or precociously blind people, but not in late blind people. Sadato et al.[2] showed similar results, i.e., greater activity in the primary visual cortex (V1) of early blind (before 16 years of age) than in late blind people, and similar activation in extra-striated areas in the 2 groups. Burton et al.[12] showed activation in V1 during Braille reading in both early and late blind people. However, in contrast to most other studies, Büchel et al.[13] described V1 activation in late blind people, but not in congenitally blind people. These authors suggested that the activation of V1 observed in late blind people would be related to previous visual experience. These discrepant results reveal the need for further studies on intermodal plasticity as a function of age at onset of blindness.

Reading in Braille is an example of sensory substitution in the blind. The pioneering work of Wanet-Defalque et al.[14] demonstrated that there is activation of occipital visual areas during the performance of tactile tasks in visually impaired people. Subsequently, Sadato et al.[2] demonstrated that activation of the primary visual cortex occurred only when the task was to discriminate Braille words, and not for other tactile stimuli. This result suggests a relationship between metamodal activation of the occipital cortex and semantic cognitive function. The importance of the occipital cortex is even more evident in cases of injury to this region in blind people. Hamilton et al.[15] reported a case of braille alexia after a stroke that affected the occipital cortex bilaterally. In this case, the patient was blind since birth and a professional reader of Braille. After the accident, she was able to identify everyday shapes and objects by touch, but she was completely unable to read Braille words or even identify a single letter in Braille.[15]

Cohen et al.[10] used transcranial magnetic repetitive stimulation (rTMS) to verify the functionality of visual cortex activation in individuals who have been blind since childhood, in a task of identifying braille letters and embossed Roman letters. rTMSr was used because it is a non-invasive technique that reversibly blocks the functioning of a small cortical region through electromagnetic pulses that cross the skull. In this way, a reversible virtual lesion can be created and the effect of the absence of function in that region can be analyzed.[1] Transient inactivation of the occipital cortex caused more errors in both tasks (braille and Roman letter readings) and distorted tactile perception in the blind. In contrast, in sighted people, this inactivation had no effect on tactile performance, but it impaired visual perception.[10] In another recent study, activation of the occipital cortex using single pulses for transcranial magnetic stimulation (TMS) caused tactile sensations in blind Braille readers.[16] Experimental evidence obtained from brain imaging studies has shown that these compensatory behaviors in blind people are associated with the mechanism of neuroplasticity and reorganization within the brain.[3,17-20] Arguably, the most striking finding was the observation that the visual cortex (normally associated with visual processing) is functionally recruited for the processing of non-visual sensory information and cognitive tasks such as memory and language.[21-23]

As mentioned earlier, there is growing evidence that the brain undergoes neuroplastic reorganization in the case of eye blindness. A key advance in understanding neuroplasticity in this condition has been the demonstration of how associated changes in the brain correlate with various behavioral measures of performance. For example, occipital cortical thickness has been shown to correlate with performance on auditory discrimination tasks.[23]

The learning of new skills is initially done through the reinforcement of pre-existing connections, resulting from the genetically controlled and different neural system between individuals, this reinforcement is possible through environmental influences, afferent input and efferent demand. Behavior is the manifestation of the coordinated functioning of the entire nervous system. As long as the exit pathway responsible for manifesting behavior is preserved (even if alternative pathways need to be unraveled or facilitated), changes in distributed neural network activity may be able to establish new patterns of brain activation and sustain function.[1]

The notion that vision loss is partially compensated for by other sensory modalities is quite popular and serves to explain the use of behavioral alternatives by the visually impaired (e.g., Braille reading).[24] In terms of the individual contribution of other sensory modalities to this compensation, hearing plays a prominent role due to its capacity for spatial analysis of the environment, similar in several aspects to vision. In sighted individuals, the spatial analysis of the environment is naturally dominated by vision, but in blind individuals, hearing plays a prominent role in navigation, allowing the location of objects and obstacles. In fact, it has been shown that blind people have above-normal abilities in auditory tasks, such as sound localization based only on monaural cues.[25]

The idea that primary cortical areas are specialized in exclusively analyzing each sensory modality is not new. Recently, however, other groups have been demonstrating that this specialization is more flexible, with several cells located in primary sensory areas responding to stimuli from 2 or more sensory modalities.[7] Although there is evidence that some sensory modalities are processed in a neuro-specific manner,8 most of our perception of events in everyday life is recorded simultaneously by more than 1 sensory modality in an integrated and unified manner, with no apparent discontinuity, optimizing the detection and recognition of objects and also our response to them.[9] For example, when planning a movement, it is common to integrate visual, somesthetic, and auditory information to optimize the sequence of movements necessary to achieve the goal.

Better understanding of how the brain adapts to vision loss and what the functional consequences are for the remaining sensory information may result in a better quality of life for the visually impaired.

CONCLUSION

Most studies on blindness and subsequent plasticity show that the extent of cortical reorganization is correlated with the age of onset of deficiency. Several researchers have identified a different pattern of cortical activation between the early blind and those who lost their vision late. In the absence of vision, the visual cortex (including the primary visual area) can respond to tactile and auditory stimuli, because the absence of vision allows the unmasking of latent connections, thus revealing metamodal organization. In addition, this process generates expansion of the cortical areas involved with touch and hearing. This explains the refinement of auditory and somesthetic functions observed in blind people. Understanding these physiological changes is essential to optimize the rehabilitation process after visual deprivation.

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