Acta Clin Croat 2020; 59:147-153
doi: 10.20471/acc.2020.59.01.18

Review

NEUROPLASTICITY AND BRAILLE READING

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SUMMARY – This article brings review of the studies and their findings about neuroplasticity of the brain and Braille reading, as well as some connections between the two. The goal of the article is to combine knowledge from different disciplines, thus enabling development of new efficient programs in rehabilitation. A lot of research has shown the possibility of brain reorganization (plasticity), indicating the creation of new neuron connections in people with vision loss which relate to Braille reading, especially in late-onset vision loss.

Key words: *Neural plasticity; Brain; Visual cortex; Vision disorders; Blindness – rehabilitation*

Introduction

When looking at the history of research about Braille reading, most researchers focused on reading achievement, reading rate, hand movement and dominance, learning media, reading process and assessment, tactile perception and contracted Braille *versus* uncontracted Braille reading. Development of neuroscience and medical technologies has resulted in new research findings becoming ever more important for workers in the educational field[1]. Multidisciplinary approach brings new and better rehabilitation programs as they offer better understanding of brain processes after vision loss and reveal a range of potential recovery and/or development in individuals taking into account congenital, early- or late-onset vision loss. Scientists are lifting boundaries in beliefs about brain development by constantly searching and learning new findings[2].

This article will describe the research in brain plasticity and its importance for visually impaired people. A description of processes in a Braille reader's brain and the way blind people 'see' will be presented, as well as some new findings from neuroscience and their application.

Anatomy behind the Process of Reading (Print and Braille)

The brain is divided into four main lobes. Each lobe (frontal, parietal, occipital and temporal) has been associated with different functions and purposes imperative to human living. Researchers examine neuron activity in visual and parietal lobes because of their importance in Braille reading[1,3]. In order to understand the research of the brain plasticity and tactile reading, it is important to understand the anatomy and neurology of visual and somatosensory pathways.

Visual pathway starts with the eye and transformation of light signals from the environment into neuron impulses in the retina. This visual information travels through the optic nerve, optic chiasm and optic tract to the thalamus, more specifically to two lateral geniculate nuclei (LGN), one in each hemisphere of the

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Received March 4, 2016, accepted July 15, 2018

brain. LGN are the information processing control centers where visual information is sorted into three brain areas: primary visual cortex (information about form, color, shape, dimension, orientation), superior colliculi (motion information) and pretectal area (photoreceptor information that helps regulate the circadian clock)[1].

Is visual cortex active in people without visual stimulation? Researchers have provided evidence regarding brain possibility to reorganize itself in the absence of visual stimuli or during new skill development (e.g., tactile reading).

According to Sadato, visual cortex of the blind during Braille reading has a role in tactile processing. It gives meaningful lexical and semantic properties to simple tactile information. Sighted people recognize and process letters by their visual characteristics in the visual cortex, whereas in Braille reading, the somatosensory system is responsible for letter perception. This ability of the primary visual cortex to additionally process tactile information provides evidence for the outstanding brain plasticity of early-onset blind subjects and therefore is functionally relevant to Braille reading ability[4].

The somatosensory path starts with sensory receptor cells (thermoreceptors, photoreceptors, mechanoreceptors and chemoreceptors). Neuron impulses travel in sensory nerves through the spinal cord to the thalamus and terminate in the ventrolateral posterior nucleus (VPL) or intralaminar nuclei. VPL is the control center for somatic sensory information processing (touch, temperature, pain, vibration and pressure). The VPL sends signals to the primary somatosensory area in the parietal lobe of the cerebral cortex, where tactile processing mainly occurs. After being sorted, information goes to the secondary somatosensory cortex or to other areas of the brain. Hannan describes moving fingertips over the tactile stimulus of raised dots as part of Braille reading. He states that isolating specific areas used for tactile perception during Braille reading movement is very difficult. The problem lays in brain scanning techniques that measure overall cortical activity and factors like number of fingers used, left or right hand use, and reading movement techniques[1].

Somatosensory path receives information from the skin, which is the largest organ of the human body. Mechanical stimulation of the skin will cause different conscious sensations. Four main types of mechanoreceptors, which differ in their ability to signal the speed and intensity of the stimulus, are located in the skin of palms and fingers. Fingertips and palms do not have identical mechanoreceptors. Fingertip skin is most sensitive to mechanical stimuli. It has 4 main types of mechanoreceptors, one type of rapidly adapting (RA) receptor and slowly adapting (SA) receptor in the skin and one type of RA and SA receptors in the subcutaneous tissue. Skin RA receptors are Meissner corpuscles (most numerous in fingertips), and skin SA receptors are Merkel tiles. Subcutaneous RA receptors are pacinian corpuscles, and SA receptors are Ruffini's corpuscles. RA receptors have great importance in Braille reading because they provide essential information about mechanical stimuli time line (sequence), which is important for the analysis of information during an active touch (palpation or finger swiping on a surface to determine if it is rough, smooth, etc.). If Braille readers are not allowed to move their index fingers from side to side between the letters, but only apply light vertical pressure, they are not able to read[5].

Neuroplasticity

Neuroplasticity is the ability of the neurons to create new connections and paths with new roles in the cortex. Neuroplasticity means reorganization of the brain. It is the ability of the brain to change its molecular, microarchitectural and functional organization or reorganize itself as a response to normal development/maturation of the body, experience, acquisition of new skills, sensory stimulation, deprivation and injury[6].

Several studies have provided evidence for neuroplastic changes in healthy human brains as a result of learning[7,8]. Damage to the brain breaks the original neuron network, which then strives to regenerate, i.e. reorganize[9].

Diamond and Hopson describe a research by neurologist Huttenlocher. He observed the visual cortex and discovered an increase in synaptic connections from the seventh month of pregnancy to birth and two months after birth. An expansion by tenth happened during second and fourth month, which the author connected to the improvement in child's vision in that age and the ability to create three-dimensional images. The number of synapses reaches its maximum around eight months. It persisted until the age of four years, after which a gradual decrease occurred, the density of synapses evened out and remained the same to maturity. This decrease in the number of synapses forms the visual cortex. The dendritic branches of the remaining synapses in the visual cortex keep extending and sprouting, therefore creating new synapses. They also describe another research of visual cortex development in kittens by Hubel and Wiesel, performed in 1978. By patching one of the kittens' eyes right after birth, for three months, they discovered the kittens were blind although the visual apparatus of the patched eye was not damaged. Visual deficiency in the beginning of life changed the cortex to receive visual impulses only from the unpatched eye. The mentioned principle applies to humans as well, although the critical period is longer. If the child does not receive any visual stimulation between the ages of eight and ten, it will result in blindness[2].

Cross-modal plasticity in the blind

The brain has the ability to efficiently reorganize and compensate for deficits in individuals with congenital or acquired absence of a sensory modality[10,11]. Blind individuals often demonstrate superior skills in their remaining senses compared to sighted individuals[12]. Areas of brain deprived of dedicated sensory stimulation can be used by other sensory modalities. Sensory-specific areas such as visual cortex receive direct (short-latency) inputs from other sensory modalities[10]. Many authors agree on visual cortex activation in the blind during Braille reading[13-20]. In children with total congenital blindness, the visual cortex is redistributed to process tactile, spatial and somatosensory information, and even language[13].

Pascual-Leone *et al.* describe a two-step process after vision loss. Firstly, the established somatosensory and auditory connections to the visual cortex reveal themselves. Secondly, new connections are established and general long-term plastic reorganization occurs[7]. Multiple pre-existing redundant pathways with the potential to take on similar function when needed may be the reason for cross-modal compensatory plasticity[21].

The brain has the ability to compensate for vision loss rapidly and reversibly. This similar cross-modal recruitment is seen in prolonged visual deprivation of sighted individuals (blindfolding for 90 minutes activates primary visual cortex during tactile discrimination tasks). The visual cortex of congenitally blind was researched in roughness or spatial distance discrimination of Braille dots during repetitive transcranial magnetic stimulation (rTMS). Results suggested that visual cortex preferentially processed macro-geometric information (spatial density). Its stimulation caused deficits in spatial discrimination, while somatosensory cortex stimulation preferentially disrupted roughness discrimination (micro-geometric judgment)[22]. Similar findings were confirmed by Hamilton *et al.* They studied a congenitally blind woman who developed alexia for Braille after having sustained extensive bilateral lesions in her visual cortex caused by stroke[23]. Despite an intact somatosensory cortex, injury of the visual cortex or temporary disruption by TMS impairs Braille reading ability[10].

Another research by Sadato *et al.* resulted in findings on the crucial age of vision loss onset in developing cross-modal plasticity. They investigated the reorganized neuron network with Tesla functional magnetic resonance imaging (fMRI) in 15 blind and 8 sighted subjects during passive tactile tasks to determine age dependency of the reorganization. Primary visual cortex was activated during a tactile discrimination task in blind subjects who had lost their sight before 16 years of age, but suppressed in those after. The first 16 years of life are critical for a functional shift of primary visual cortex from processing visual to tactile stimuli. Congenital and early childhood blindness commonly activates the primary visual cortex, but not in cases of blinding in adulthood and elderly life. They concluded that probability of visual rehabilitation success relates to the degree of visual competencies during the sensitive visual period[24]. The question remains whether the unclosed eye has better vision quality because of having at its disposal all of the synapses in the visual cortex? The scientists have not given an answer to that question yet.

The already developed visual network in early- and late-onset blindness performs perception for other senses but in congenital blindness, the visual network will develop and strengthen only if exposed to meaningful experiences and age-dependent activities, as well as modify other senses in compensation for blindness[14]. Visual cortical pathways are recruited differentially not only in sighted and blind individuals but also in early and late blindness onset. This suggests that plastic reorganization and its range varies among populations[16].

New research implies the need for a new model of neuroplasticity since reorganization changes in late-onset blind and congenitally blind reach beyond visual cortex and cross-modal plasticity into multimodal or multi-sensory integration regions, where interconnectivity between visual regions and multimodal/heteromodal integration regions was increased in the mentioned groups compared to sighted controls[25].

Research

Burton *et al.* researched visual cortical activation in early blind, late blind and sighted participants during a vibrotactile matching task. Results showed the greatest activation of visual cortex in early blind and some activation in several of the late blind participants (early blind had better response magnitudes). The evidence supported the thesis of decreased plasticity in visual cortical regions with later onset age of blindness[26]. A similar study agreed the congenitally and early-blind participants were better than the sighted ones on a vibrotactile discrimination task. Although in this research, duration of blindness did not predict task performance, congenitally blind participants were more accurate than the early-blind participants[12].

Gizewski *et al.* wanted to differentiate whether occipital activation of blind subjects during Braille reading is task-specific or only triggered by sensory or motor area activation. They conclude the brain differentiates between 'finger touching' and 'finger reading', thus not leading to the activation of the visual cortex by pure motor or sensory tasks. Braille reading is task-specific and not a combination of sensory or motor area activation. This indicates the activation of the visual cortex in blind subjects to higher and more complex brain functions[27].

Liu *et al.* researched the branching of altered functional synapse connections and their network in early childhood blindness using fMRI in the state of inactivity. Reduced functional connectivity in visual cortex was found between the visual areas in visual cortex and temporal multisensory area. The correlation coefficient between the reduced functional connectivity and Braille reading exercise increased if the blind person practiced Braille reading before or spent a lot of time practicing, especially in childhood. These findings may indicate that the general loss plasticity mechanism (less possibility of plasticity of the brain) and compensatory plasticity mechanism coexist in those with vision loss in early childhood. Changes in the functional connectivity in the steady-state people can be an integrated reflection (general or overall) of a general loss and compensatory plasticity in such deprived sensory modality. These authors investigated only one sensory modality deprivation. The plasticity is larger in those who very often practice Braille reading and those who started to read at an early age. In other words, they have a larger connectivity between the mentioned brain areas[28].

In sighted adults, a nine-month tactile Braille-reading training showed anatomical grey and white matter reorganization in the visual cortex, which is used for tactile discrimination of Braille characters, along with stronger functional connections to somatosensory and motor cortices[29]. Also, activity in the visual cortex during blindfolded Braille reading and the visual word form area (VWFA) observed with whole brain fMRI was noticed, as well as resting-state functional connectivity increase between the VWFA and somatosensory cortex and bilateral decrease with other visual areas. TMS disruption of VWFA impaired the accuracy of Braille reading. The results implied cross-modal plasticity as a result of long-term training in cases without sensory deprivation and injury[30].

How much time does it have to pass before Braille reading skill begins to atrophy when people are not exposed to Braille reading? In one of his research subjects, Hannan mentions a negative effect on Braille reading skill after a nine-week recess of Braille reading. After the recess, the subject started to read Braille again. She restored her Braille reading skill very quickly and her neuron connections regenerated. In efficient Braille readers, the Braille reading skill retains even if not used and is renewed when needed. However, the subject in question reported reading for approximately six hours *per* day. That is something we have to keep in mind, as people learning new skills or those who have not practiced and trained this much will not have the same retention or return of the skill in question[1].

Sadato *et al.* tried to exclude long-term learning of Braille as the reason of activated visual cortex during tactile discrimination (Braille reading) using fMRI. The visual cortex during the tactile discrimination task was activated in blind subjects who had recently lost their sight and never learned Braille, but not in sighted subjects. This finding suggests that the activation of the visual cortex of the blind during tactile discrimination task is due to sensory deafferentation, whereas sighted subjects with both modalities available favor tactile over visual modality. Deafferented areas of the visual cortex are recruited in the blind due to sensory influence and this tactile-visual cross-modal plasticity seen in the late blind is task-dependent but not learning-dependent. Long-term visual deafferentation can improve tactile acuity independently of prior Braille learning due to plasticity in the occipital cortex, but this study indicated no such tactile advantage in the recently blind, although some activity in response to tactile stimuli in visual cortex was already showing[31].

Does passive exposure to tactile stimuli in the blind result in increased visual cortex activity? Ortiz *et al.* studied visual cortex in blind teenagers and adolescents who had been exposed to passive tactile stimulation 3 hours *per* day for 3 months. The activity in visual cortex grew as the exposure continued. Although the stimuli in this research were not Braille dots but vertical, horizontal and oblique tactile lines, the study opens the question of similar passive program usefulness in later active tactile discrimination tasks and Braille reading ability[32].

Pascual-Leone and Torres associate reading Braille with the expansion of sensorimotor cortical representation of the reading finger. They came to this conclusion by studying the organization of the somatosensory cortex in proficient Braille readers, recording somatosensory-evoked potentials in blind and sighted subjects[33].

During Braille learning, neuroplastic changes happen because learners must acquire the ability to extract spatial information from subtle tactile stimuli. The sensorimotor cortical area devoted to representation of the reading finger enlarges. This enlargement suggests initial unmasking of the existing connections and establishment of more stable structural changes. In addition, Braille learning appears to be associated with recruitment of parts of the occipital cortex for tactile information processing. The occipital cortex can be critical for reading accuracy. Several studies suggest the possibility of applying noninvasive neurophysiologic techniques to guide and improve functional outcomes of these plastic changes, thus accelerating functional adjustment to blindness[34].

Ptito *et al.* stimulated the occipital cortex using single pulse transcranial magnetic stimulation in early blind subjects and blindfolded sighted control subjects. Some of the blind subjects felt tactile sensations in the fingers. The visual cortex was somatotopically organized for each finger. Blindfolded control subjects did not report any tactile sensations. Those who spent more hours *per* day reading Braille, as well as those who had better reading speed and dexterity reported more tactile sensations. A polysynaptic cortical pathway between the visual and somatosensory cortex may be responsible for these results[35].

Cohen *et al.* examined activation of the visual cortex of late blind individuals in somatosensory processing. They concluded that late blind participants may have been using visual imagery to assist them in task performance because of left precuneus activation, which is thought to have a visual imagery and memory recall function[36].

Studies have shown greater plasticity in people with significant visual impairment than in those with a less significant visual impairment, although further research is needed to determine the effects of Braille reading training[1]. Although various different methods (imaging, TMS, and psychophysiologic recordings) used in congenitally blind people have confirmed functional activation of the visual cortex, they have not extensively explained functional significance of these activation patterns[16].

Conclusion

Development of neuroscience has provided new information that is useful in rehabilitation, especially information about plasticity, brain areas and their functioning. Researchers have begun examining ways to harness neuroplasticity to promote healing and recovery. Changes in brain cell function occur throughout lifetime because of learning experiences and in response to nervous system injury. Visual stimuli are the main source of receiving information from our environment. Blind people use other senses to perceive their surroundings and this new usage implies anatomical (significant differences in volume and thickness of some brain regions) and functional (e.g., tactile stimuli while Braille reading activates visual cortex) changes. Evidence shows new neuron connections being formed in the visual cortex of people with patches over their eyes during an extended period of time, and disappearance of these connections after patch removal. As ever more is being learned about what causes neuroplasticity of the brain, we hope that new implications will be developed for educational and rehabilitation practitioners who work on developing Braille literacy in the visually impaired. Taking into account the potential for neuroplasticity in congenital, early- or late-onset vision loss, we can determine the range of success in learning Braille reading, which is important to create an individualized program for Braille literacy. The length, intensity and approach in the program need to be adjusted or they will discourage the student. A student who is prepared and knows what to expect is less likely to lose motivation during the literacy program.

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Sažetak

NEUROPLASTIČNOST I ČITANJE BRAILLEOVA PISMA

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U ovom radu prikazuju se studije i njihova saznanja o (neuro)plastičnosti mozga i čitanju Brailleova pisma, kao i poveznice između njih. Cilj je ovoga rada spojiti znanja različitih disciplina koja će zatim omogućiti razvoj boljih rehabilitacijskih programa. Brojna su istraživanja pokazala mogućnosti reorganizacije mozga te ukazuju na stvaranje novih veza u mozgu nakon gubitka vida kod slijepih osoba. Važnost spomenute plastičnosti može se povezati s čitanjem Brailleova pisma, osobito kod kasnije oslijepljelih osoba.

Ključne riječi: *Neuralna plastičnost; Mozak; Vidna kora; Vidne smetnje; Sljepoća – rehabilitacija*