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Does Brain Stutter?

SUMMARY

Currently, stuttering studies focused on central nervous system pathology are one of those which develop most dynamically. Stutterers display various disorders within the anatomy of centres which control realization and abnormal lateralization of language functions, as well as functional disorders. Abundant research has proved that stutterers display disturbed functional brain asymmetry. The left hemisphere speech representation in a stutterer's brain is marked less clearly than in the case of fluent speakers. Speech fluency comes from realizing the motor plan, which is abnormally synchronised in a stutterer's brain. Similarly, stuttering people display incorrect cooperation between the language program and its motor performance. Dysfunctions are observed not only in the expressive speech areas, but also in the hearing centres, whose proper functioning is vital for normal course of speech. Research has showed the disturbed auditory feedback control of speech among stutterers, which is thought to be caused by anatomical and functional abnormalities within the brain hearing area. Results of studies concerning stutterers' brain structure and functioning point to numerous abnormalities observed in the whole functional system of speech. Importantly, these functional differences occur among stutterers during actual speech and even when speaking is only visualized in the stutterer's mind.

Key words: stuttering, brain, disturbances of the brain

Research on stuttering which is focused on nervous system pathology is currently one of the most dynamically developing. The key areas of investigation include differences in brain structure and functioning (particularly those parts which are responsible for speech), synapses and brain dominance as well as anomalies at the level of neurotransmitters. Research of this type came with the development of modern diagnostic methods. Initially, examination was performed with EEG,

then with computed tomography (CT and PET) and magnetic resonance imaging (MRI and fMRI).

People with stuttering (PWS) display various types of anatomies of brain areas controlling language functions, as well as functional disorders and abnormal lateralization of language functions. The research proved abnormal anatomy of cortical speech areas (Braun et al. 1997). Brain examination pointed to pathological patterns of brain area activation. When an ordinary speaker speaks, areas within their left brain hemisphere get activated. However, in the case of PWS, higher level of activity is observed in the right hemisphere (De Nil et al. 2000). Abnormal work of particular cortical and subcortical language areas are also observed (Heitmann et al. 2004). It has been identified that the cortical area activated, particularly hearing areas and motor areas which control lip and laryngeal movements, is larger among fluent speakers than among the disfluent ones (Foundas et al. 2004; Jäncke et al. 2003; 2004).

1. LATERALIZATION OF LINGUISTIC FUNCTIONS IN PWS' BRAIN

People display individual differences in the way language functions are organised in their brain. They differ with regard to both size of particular brain areas which are responsible for communication processes (structural differences), and the engagement of both hemispheres (asymmetry). In the case of most right-handed people and more than half of the left-handed, the left hemisphere is dominant in speaking. However, it is now common knowledge that the right hemisphere is also involved in performing linguistic tasks. In order to ensure proper course of speech, fundamental functions should be developed properly, individual areas should cooperate with one another to make a unified functional system, the left hemisphere needs to dominate and processes performed within both hemispheres should be integrated (Martin et al. 2004).

Numerous studies prove that the left-hemispheric speech representation of PWS is not so clearly marked as it is among fluent speakers. One of the first studies to confirm this thesis was related to binaural hearing. The study was performed among 20 PWS and 20 fluent speakers with the dominant right ear. It was assumed that right ear is the preferred one for the right-handed, which implies dominance of the left hemisphere for speech sound processing. This was confirmed for 75% of the control group and only 45% of the PWS (Curry, Gregory, 1969). These results have been confirmed many times and taken as the evidence of relatively less developed hemispheric dominance among PWS (Ingham 2001).

Also, certain anatomic differences have been identified to indicate reduced or reverse asymmetry and are regarded as markers of atypical brain lateralization in

PWS. In their research, Jäncke et al. (2003) used magnetic resonance to compare the total volume of brain hemispheres and the size of prefrontal areas as well as to compare prefrontal areas of both hemispheres in PWS and the control group. The size of hemispheres and the total volume of the brain were not different. However, while fluent speakers were identified to have a larger prefrontal area in the left hemisphere, adult PWS did not display such asymmetry. Severity of speech disfluency was not linked with individual anatomical configurations.

The model of brain dominance in PWS was also examined by testing PWS' skills and fitness. The patients were asked to perform various lateralization-related tests. It has been concluded that PWS' motor competence is lower for both the dominant and non-dominant hand (Loukas et al. 2007; Subramanian, Yairi 2006). In the research conducted by Saltuklaroglua et al. (2009), PWS were asked to speak and draw simultaneously. Statistically significant differences were observed with regard to performing the motor task, both when PWS were drawing and speaking at once, and when they were only drawing. Such a correlation was missing in the control group. PWS display weaker coordination and precision as well as delayed motor planning when two tasks are being performed simultaneously. In the authors' view, all the difficulties observed result from limited brain dominance.

2. THE FUNCTIONING OF AREAS RESPONSIBLE FOR SPEECH EXPRESSION IN PWS

2.1. Broca's area

Broca's area is located in the lower part of the frontal lobe, which stands for area 44 and 45 according to Brodmann's classification, and is responsible for controlling speech production and movements of articulatory muscles. It is a program of a kind which instructs the motor cortex as to how to manage the articulatory and phonatory system.

PWS display anatomical and functional disorders within the Broca's area. Foundas et al. (2001) concluded that there are numerous differences between many adult PWS with regard to how brain folds are configurated within this area and whether there occur any extra folds. Cykowski (2008) observed a slightly higher number of folds in the right Broca's area than in the left one. Beal et al. (2015) identified considerable differences in the structure and development of grey matter. Underdevelopment of the left part of Broca's area results in its insufficient activity, which has twofold consequences. Firstly, it causes too strong activation of the right part (Sommer et al. 2002). It should be noted that in fluent speakers speaking is always connected with activation of the left part of Broca's area. The right part becomes involved when one tries to stop the process of speaking. Increased activity of the right part of Broca's area was also observed in

attempts to stop the motor activity of the hand (Aron et al. 2003). This means that increased activity of Broca's area in the right hemisphere leads to stopping and inhibiting the process of speaking.

The other consequence of limited activity of Broca's area in PWS' left hemisphere is premature activation of left premotor cortex and primary motor cortex. Motor cortex activates before Broca's area sends instructions as to how to direct the articulatory system (Salmelin et al. 2000).

Moreover, it has been concluded that in the case of PWS, neurocommunication between the left part of Broca's area and the sensomotor area, which stores the representation of face and lips, is ineffective due to insufficient thickness of white matter (Jäncke et al. 2004; Sommer et al. 2002). Therefore, transmission of the code to motor areas by abnormally developed Broca's area is further delayed by reduction of white matter.

By observing how healthy people's brains work it has been concluded that stress causes dysfunction within the Broca's area. Individuals experienced it as impaired speech fluency which they observe in strong emotional tension. If the area does not function properly, stress suffered by a PWS will worsen the situation even further (Alm, Risberg 2007).

To sum up, it can be concluded that speech fluency is possible thanks to following a motor plan. However, in the case of PWS, it is synchronised improperly and cooperation between the linguistic plan and its motor performance is faulty (Bosshardt 2006).

2.2. Premotor area and supplementary motor area

In area 6, according to Brodmann's categorisation, there are two motor areas of associative type. The bottom part of area 6 includes a premotor area (PMA), while the upper part includes a supplementary motor area (SMA).

The premotor area is a place where models of motor activities are coded and advanced forms of speech transmission are coded. Exposing the area to electric shock results in a speech block (Duffau et al. 2003).

When fluent speakers speak, the premotor area and the Broca's area are activated simultaneously. In the case of PWS, premature activation of the premotor area is observed (Salmelin et al. 2000). Presumably, it is a way of compensating for the too late activation of the Broca's area. As a result, the motor cortex is activated in an unconcentrated way instead of selectively activating the representation of face muscles. Moreover, reduced connectivity between the Broca's area, premotor cortex and primary motor cortex has been observed (Joos et al. 2014; Kronfeld-Duenias et al. 2016).

Based on information sent from prefrontal and premotor area, a command to perform movement is created in the supplementary motor area and then sent to

the primary motor cortex. Damaging this area leads to difficulties with starting an utterance, voice modulation and temporary course of the utterance.

Supplementary motor area (SMA) of PWS does not work properly in either left or right brain hemisphere. The left SMA is not active enough, while the right one is hyperactive (Ingham et al. 2000). Neuropsychological examination confirmed that this disfunction of SMA is observed in PWS (Forster, Webster 2001). This hypothesis was also proven in research which made use of PET (Ingham et al. 2000). Hyperactivity of the right SMA probably results from the malfunctioning Broca's area being unable to stop it. Examination of patients with aphasia done with fMRI proved that left SMA damage results in hyperactivity of this area in the right hemisphere, and, consequently, serious problems with articulation (Martin et al. 2004; Naeser et al. 2004).

2.3. Other structures

The extrapyramidal system is a set of subcortical and cortical areas which perform numerous functions. It participates in all motor activities and can stimulate or hinder them. It also helps regulate muscle tension, controls the range, pace and precision of intentional movements as well as participates in movement automatization and developing motor habits (including spontaneous speech). The extrapyramidal system consists of cortical areas (mainly areas 6 and 4) as well as such subcortical nuclei as: basal ganglia (caudate nucleus, putamen, *globus pallidus*, amygdala), thalamic nuclei (ventral anterior nuclei, ventral lateral nuclei, dorsal–medial nucleus), subthalamic nucleus, mesencephalic nuclei (red nucleus, *substantia nigra*). Numerous neurotransmitters: dopamine, acetylcholine, serotonin and *gamma*-Aminobutyric acid (GABA) play an important role in the functioning of the extrapyramidal system (Alm 2005).

The role of subcortical nuclei in developing stuttering was first identified in patients whose stuttering came as a result of damage of these structures (Tani, Sakai 2011). It has been proven that putamen and caudate nucleus in the left hemisphere are less active. It should be noted that while the left caudate nucleus is directly linked to the Broca's area, the right one is not (Alm 2004). It has also been concluded that electric stimulation on the left subthalamic nucleus led to difficulties in articulation, whereas electric stimulation of the right one had no effect on speech (Santens et al. 2003). The diagnosed hyperactivity within *substantia nigra* and red nucleus was an additional evidence of the role of dopamine in stuttering (Watkins 2008). Treating fluent speakers with a medicine known as theophylline results in some of them becoming disfluent. Although this side effect is rather rare, it offers interesting hints as to the mechanism of stuttering. Theophylline improves dopamine transmission by blocking adenosine and GABA receptors, which is similar to what PWS are characterised by, i.e. excessive dopamine level

(Movsessian 2005). Some research indicates that dopamine level in PWS is even three times as high as it is in fluent speakers. Significant improvement of speech fluency observed after a patient has taken medicines which block dopamine receptors is the evidence of how dopamine level affects stuttering (Stager et al. 2005).

The fact that PWS find it difficult to learn automatic motor skills is an indirect proof that subcortical nuclei perform certain function in developing speech disfluency. When compared to fluent speakers, they differ with regard to not only amplitude and duration of movement, but also to acquiring habitual motor skills in terms of speed, stability and strength (Namasivayam et al. 2008). However, use of external help (the rhythm of a metronome, choral speaking) helps improve speech fluency. According to one of the hypotheses, it is so because external support helps to replace the faulty cortical-nuclear nervous road with the cortical-cerebellar one (Alm 2004).

Apart from the main areas responsible for speech which have been discussed above, there are also supplementary areas which may also function improperly in PWS.

The arcuate fasciculus is a bundle of association fibres which connect Wernicke's and Brock's areas. By containing fast transmitting association fibres, it enables quick combination of the perceptive and expressive elements of speech. Foundas et al. (2001) discovered atypical anatomical features of this structure in a fMRI examination, which, as anomaly, have been confirmed in research by Büchell et al. (2004), Cieslak et al. (2015), and Sommer et al. (2002).

Wernicke was the first to point to the role played by the insula in speech mechanisms. Insula cooperates with motor areas in programming activities of speech organ muscles and in producing words in the correct time sequence. Insula damage results in difficulties in speech expression and appropriate pronunciation of phonemic sequences (Dronkers et al. 2007). Patients who display impairment of this structure are aware of the mistakes they make and try to correct them by repeating the words pronounced several times. When compared to fluent speakers, PWS display hyperactivity of insula (Ingham 2001; Watkins 2008).

3. FUNCTIONING OF AUDITORY SPEECH AREAS IN PWS

Natural course of speech is conditioned by properly working auditory areas. While speech is being produced, the speaker's auditory feedback controls the process, and, consequently, speech sounds produced are understandable for other people.

It is scientifically proven that PWS' auditory feedback control of speech is disordered and anatomical and functional abnormalities within the auditory area are believed to be one of the causes (Jäncke et al. 2003; Lincoln et al. 2006). The

auditory area is larger in the left hemisphere of fluent speakers, unlike in the case of PWS. PWS display either symmetry of auditory areas in both hemispheres or atypically larger area in the right hemisphere (Jäncke et al. 2004; Foundas et al. 2003). As observed in an fMRI examination, there is also both-side deactivation of the associative auditory area while speaking (Fox 2000).

As a result, a PWS experiences difficulties in sound processing of his/her own speech, which disturbs the process of speaking as such. However, use of methods which change perception of one's own speech (delayed auditory feedback, white noise masking, the shadow method, synchronised speaking) improves PWS' speech fluency considerably (Howell et al. 2006; Howell 2007). Before neuroimaging techniques were applied, choral speaking had been believed to be able to change the model of brain processes during speaking. However, the well-developed neuroimaging techniques have proved that there are quite significant differences in brain activation models between PWS and fluent speakers. One of the key pieces of evidence is related to differences in brain activation during solo and choral speaking, and comes from a research done by Ingham (2001). He used an fMRI to examine 10 PWS and 10 members of the control group while they were reading a text on their own and then chorally (the patient would hear the text read in headphones and read it on his/her own at the same time). PWS display an outstanding activity of the right hemisphere within the area 6 of Brodmann's classification, which includes a supplementary motor area and a premotor area. When compared to the control group, there also exist differences with regard to auditory areas, insula and cerebellum. However, Wernicke's area is worth mentioning here as it was relatively passive in PWS. A similar effect is observed in primary auditory cortex, particularly in the right hemisphere. Choral speaking significantly changed the model of brain activation in PWS by reducing the hyperactivity of insula, premotor cortex and supplementary motor cortex in the right hemisphere. Still, cerebellum remained relatively active. Activity of the inactive Wernicke's area during choral speaking is similar to the one displayed by the control group, which leads to the fact that during solo speech, the inactive auditory area makes a PWS unable to monitor and control his/her speaking properly. Furthermore, it has also been proved that there is no difference in brain activity when a patient is not speaking, and differences are evident even when patients are asked only to imagine they are talking to someone.

It has been proved that, although PWS' language skills are normal, the brain processes which use of language skills is based on, are atypical for the majority of population (Weber-Fox, Hampton 2008). Some PWS display changes related to information (also the auditory one) processing, particularly with regard to complex sentences (Bosshardt 2006). Auditory processing deficit often results in disorders related to more advanced auditory and linguistic processes. Exam-

ining such auditory endogenous potentials as wave P300, N400 and P600, or MMN are a useful way to identify the mechanisms and pathologies of central auditory processes.

The P300 wave reflects reaction to the new and represents the complex neural processes which are responsible for identifying and distinguishing new stimuli. PWS and the control group did not differ in terms of wave P300 parametres (Furquim de Andrade et al. 2007). In their research, Morgan et al. (1997) noticed that the P300 wave in the right hemisphere of PWS displayed a higher amplitude. Furthermore, it has been observed that increase in P300 wave amplitude and decrease in its latency are a consequence of increase in speech fluency among PWS (Andrade et al. 2008). While the N400 wave, which is responsible for semantic processing, is similar both in PWS and fluent speakers, the P600 wave, which controls syntactic processing, is different in PWS (Usler, Weber-Fox 2015). Also, it has been proved that the amplitude of MMN wave (Jansson-Verkasalo et al. 2014) and the N1 wave (Wieland et al. 2015) during speech planning is lower among PWS.

The research results related to PWS' brain structure and functioning presented above point to numerous abnormalities which concern the functional speech system as a whole. It should be noted that functional differences become evident when PWS speak or even imagine themselves speaking. Although it is still debatable whether anatomical and morphological differences in brain structure are the cause or the result of stuttering, research conducted among children offers solid evidence that neurological differences occur even in early stages of speech disfluency.

Research conducted among 3- to 9-year-old stuttering children and their fluent peers have proved that stuttering children display weaker connectivity in neuron areas which are responsible for movement management. This may influence the process of speech planning and skilful managing speech-related motor activity (Chang, Zhu 2013). The researchers were observing fourteen right-handed stuttering boys aged 8–13 and identified that, compared to the control group, they displayed limited volume of caudate nucleus as well as atypical left asymmetry. These abnormalities may cause problems with motor automation of speech (Foundas et al. 2013). Results of research conducted among stuttering children aged 6–11 prove that they display diadochokinetic rate deficit, i.e. deficit in the speed which is supposed to stop a given motor impulse and replace it with its opposite. This results in little efficiency of motor control during speech production and articulation (Malek et al. 2013). Measuring the thickness of gray and white matter (VBM – voxel-based morphometry) among eleven children with stuttering and eleven fluent ones led to a conclusion that children with stuttering display lower volume of gray matter within the lateral part of the frontal lobe, in the left puta-

men and in both sides of corpus callosum, whereas the thickness of gray matter is higher in the right Rolandic fissure and in temporal operculum (Beal et al. 2013). When compared to fluently speaking peers, children with stuttering also display atypical asymmetry of the frontal and parietal-occipital lobes (Mock et al. 2012).

Analysis of the literature indicates that future research on PWS' brain will follow several strands. Firstly, comparisons will be made not only between PWS and fluent speakers, but also between PWS in moments of fluent and disfluent speaking (Belyk et al. 2015).

Secondly, we are observing an increasing number of studies on the influence of traditional speech fluency therapies on changes in brain functioning (Ingham et al. 2013; Toyomura et al. 2015).

Finally, further research will test effectiveness of different therapies which change PWS' brain functioning (Neef et al. 2015).

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