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Neuroanatomical causes of stuttering

SUMMARY

The article discusses the results of the research team of American researchers from the University of Michigan: Soo-Eun Chang, David C. Zhu, Ai Leen Choo and Mike Anstatt (2013, 2015), which were published in the prestigious journal *Brain*. Studies were carried out in connection with the exploration of structural and functional differences in the brains of people who stutter, compared with people who speak fluently. The results provide evidence of multiple changes in the connections of the brain as the cause of stuttering. The discussion verified the existing theories of the origin of stuttering in comparison with the presented research and indicated the prospects of further investigations, concerning both theory and practice.

Keywords: stuttering, neuroimaging, auditory-motor integration, fluency of speech

INTRODUCTION

The studies by the U.S. University of Michigan team of researchers: Soo-Eun Chang, David C. Zhu, Ai Leen Choo and Mike Anstatt (2013, 2015) that were published in the prestigious *Brain* journal may be of fundamental importance for understanding the causes of stuttering. The investigations were carried out in connection with a search for structural and functional differences in the brains of stutterers in comparison with fluent speakers (nonstutterers). This direction of research, made possible owing to advanced neuroimaging techniques, began in the 1990s (Fox et al. 1996) and brought in the early twenty-first century significant discoveries in the studies by Martin Sommer and et al. (2002) on disorders of connections within the grey matter in the speech area of the left hemisphere

in developmental (stuttering onset in childhood) stutterers (see also Woźniak 2002, 2015).

The abovementioned and many other research results pointed to subtle functional and structural differences in the cortical areas supporting the auditory-motor integration of processes of utterance production, and to differences in the involvement of ganglia in the basal-ganglia thalamocortical loop in stutterers and nonstutterers. The observations interpreted with reference to the treatment of stutters enabled explanation of some interesting phenomena associated with stuttering, such as, for example, improvement of speech fluency in the cases of auditory masking (jamming with white noise), manipulation with auditory feedback (echo correction), the use of external auditory stimulation (metronome). A significant limitation to the generalization of conclusions formulated based on the investigations was the fact that it focused on studying adult stutterers whereas the onset of stuttering can be sought already at two- to four-year-old children. In adults, there may have already developed compensatory strategies and changes caused by neuroplasticity as a consequence of years of coping with stuttering. Most of the studies showed deficits in the connections of basal ganglia with supplementary and primary motor cortex as well as disorders of connections within the auditory-motor network covering Broca's center (particularly the Brodmann area 44), the adjacent inferior frontal gyrus, and superior frontal gyrus. The studies also indicated the heightened activity and volume of structures in the areas of the right hemisphere, which may be a secondary effect of compensation. The only way to decide whether we are dealing with primary changes or a consequence of other phenomena was to examine the subjects closer to the moment of occurrence of stuttering, i.e. children. Only such investigations were able to show whether disorders in cortical and subcortical connections could be the internal cause of the pathophysiology of stuttering (Chang, Zhu 2013).

Along with functional and structural disorders of connections in the left hemisphere, the previous theories of the evidence-based origin of stuttering also named:

a) Disorders of the temporal aspect of utterance perception (Szeląg 1999)

b) Disorders in the lateralization of auditory control of utterances (Kurkowski 2013).

It can be assumed that the foregoing theories are not only true but also interrelated, to the effect that they indicate different disorders in the functioning of the brain contributing to the rise of speech disfluency. All the above-mentioned phenomena may be somehow dependent on the common neuroanatomical basis.[1]

[1] For a broader discussion of the presented theories of the origin of stuttering, see the article by Tomasz Woźniak, *Diagnoza i terapia osób z zaawansowanym jąkaniem,* [in:] S. Grabias, J. Panasiuk, T. Woźniak (eds.), *Logopedia. Standardy postępowania logopedycznego*, Wydawnictwo UMCS, Lublin 2015, pp. 797–835.

However, to provide an answer about the causes of stuttering certainly requires answering many additional questions, for example: why does stuttering start in early childhood in some people and in others later – at the beginning of school or during adolescence? What about post-traumatic stuttering in adults. What is the contribution of emotions to the origin of the phenomenon? Why do stutterers sometimes speak fluently? Why do some persons have spectacular successes in overcoming their disorder while others fail despite many therapies? The answers to the foregoing questions can be provided only by reliable basic research based on neuroimaging or other objective methods of observing physiological processes underlying the functions associated with speech or hearing, otherwise we will still continue to multiply speculations.

When deciding to conduct this kind of investigations, the authors of the publications discussed in this paper formulated two preliminary hypotheses, which were verified in the next publications:

1. Stuttering children may, in comparison with their non-stuttering peers, show weaker functional and structural connectivity both in neural networks of the auditory-motor areas and in the connections of basal ganglia with thalamocortical areas, first of all in the left hemisphere of the brain (Chang, Zhu 2013).

2. In stuttering children there may occur a difference in the organization of the white matter in the brain, particularly in the above-named areas, but also in other cerebral regions (the right hemisphere, corpus callosum), which is caused by subtle differences in the development of the white matter between three and ten years of age (Chang, Zhu, Choo, Anstatt 2015).

The studies in 2013 and 2015 were carried out using the methods of functional magnetic resonance imaging (fMRI) and diffusion tensor imaging (DTI) – tractography, utilizing fractional anisotropy (FA). The employed methods allow functional and structural assessment of cortical and subcortical structures, both of the grey and white matter. For example, FA measures the directions of water diffusion as an indicator of the organization of the white matter. This enables determination of, for example, the degree of myelination, the cohesiveness of the courses of axon bundles in contrast to their chaotic tangling, as well as the integrity of the cell membrane of axons. This method is assisted with computer programs using spatial statistics. The investigations were carried out with a strict observance of meticulous experimental procedures, paying attention not only to technical and statistical requirements but also to procedures, which took into account for example the earlier acquaintance of the subjects with the conditions under which the tests were carried out.

The study groups were stuttering children (SC) and non-stuttering ones (NSC), aged between three to ten years (respectively: 27 SC/29 NSC in the 2013 study and 47 SC/42 NSC in the 2015 study), selected on the basis of standardized tests for the assessment of language utterances. In the case of SC the degree of

disfluency was defined as moderate. The children were right-handed, monolingual (English-speaking), without developmental disorders, at comparable age and with the comparable economic status, without any additional speech disorders. Attempts to maintain a gender balance were also made, but it was not entirely achieved, particularly in the group of the stuttering children, where there was a majority of boys, for example in the 2015 study there were 28 stuttering boys and 19 girls. The average time of stuttering in the studied children in both studies was about three years.

RESEARCH RESULTS

In the 2013 study significant differences were found between stuttering children (SC) and non-stuttering children (NSC) with regard to functional and structural connections in the brain.

The results formulated based on the data from functional magnetic resonance (fMRI) allow a conclusion that in SC there is weaker structural and functional connectivity in neural networks of the left cerebral hemisphere that make up the connections of the basal ganglia with the thalamocortical circuit, including connections with the left putamen and the supplementary motor area. Interestingly, SC had more connections with the putamen in the right hemisphere. This region is connected by descending motor and sensory pathways and it itself sends ascending pathways to the premotor cortex and supplementary motor areas. Functional studies also show deficits in SC as compared with NSC in the left basal dorsal putamen, in the left cerebellar hemisphere, the left insula, and in the right superior temporal gyrus.

The tractography (DTI) data demonstrate that NSC have a larger number of connections between the left putamen and the left middle frontal gyrus, the left middle temporal gyrus and the left insula. Furthermore, stronger connections are observable in the white matter associated with the supplementary motor areas, parietal lobes (on both sides), the left gyrus of the cingulated cortex (the region in the medial part of the brain, above the corpus callosum, which is of significant importance in planning and controlling behaviors), and with the left putamen.

In contrast, SC show the weakening of functional and structural connections within the network in the auditory-motor area of the left hemisphere, especially the connections of Brodmann area 44 (Broca's center) with the superior temporal gyrus (Wernicke's area). Interestingly enough, smaller differences are found in girls whereas stuttering boys exhibit a distinct functional decrease in connections as compared with nonstuttering boys.

Moreover, in NSC larger structural connections are reported between the left posterior region of the frontal gyrus and the left superior frontal gyrus. NSC showed greater connectivity of these areas with the putamen.

The presented results explicitly show the stuttering children's functional weakening of connections between the principal cerebral areas within the basal ganglia loop, in their connections with the thalamus, the supplementary motor area, and with the putamen. These areas are essential in explaining the potential pathophysiology of stuttering because they support the timing of self-initiated motor sequences, they are responsible for the complex motor control, including the inhibition of involuntary movements. They are also responsible for sensorimotor learning (apart from other functions). The putamen and the supplementary motor area may interact with the cortical auditory-motor system which is fundamental to the production of speech.

Among the studied children only the stuttering boys exhibit weaker functional connections between the internal premotor areas (pars opercularis) and superior temporal lobes in both hemispheres. This observation can be additionally interpreted with regard to the difference in the brain structure depending on gender, and also with regard to the more frequent occurrence of stuttering in boys and the more frequent recovery from stuttering in girls in the course of individual development. It is a fact that more girls grow out of stuttering.

Functional disorders in SC are confirmed in structural disorders of the white matter, which is manifest in the significantly lower number of connections. This applies mainly to connections of the cortical auditory and motor areas with the putamen in the left brain hemisphere.

These results are solid proof that there are the earlier assumed differences in brain connectivity, the differences that are of fundamental importance for the emergence of stuttering in children, from the moment when disfluency symptoms were observed. To sum up, it should be said that in stuttering children the neural networks, which support the timing of begun motor activities/sequences, are affected and function at a lower level.

One more conclusion that stems from the studies in question is that SC have weaker connectivity within the network of basal ganglia with the thalamocortical areas. The thalamus and the supplementary motor area are part of the network which also includes the primary motor cortex and the superior temporal gyrus. The functions of this network also assist the self-initiated motor sequences. But the networks are more active in the case of such sequences rather than in the case of externally triggered sequences (stimulated, imitated). This may explain some well-known stuttering-related phenomena, for example the fact that stuttering mainly occurs during spontaneously formulated utterances while the utterance of a person can be entirely fluent when it is formulated in synchrony with an external situation: rhythmization or metronome pacing.

The studies by A. Toyomura et al. of 2011 referred to by the above-cited authors showed that the cerebral activity of stuttering adults assessed using fMRI changes depending on whether they speak solo or in chorus, or to the external

rhythm. While speaking solo, the stuttering persons had lower activity in the motor regions as compared with the nonstutterers. This activity increased with the used methods of inducing fluency (i.e. speaking in chorus and rhythmically), especially while speaking to metronome beats. The activity of the auditory cortex became significantly heightened at the time. The observed differences concerned the left brain hemisphere.

Fluency induced by through externally guided patterns may be interpreted in stuttering like the effect of utterance improvement in patients with Parkinson's, in whom the external pacing signals allow them to initiate correct articulatory movements: cortical-cerebellar connections are utilized as compensatory in order to overcome deficits in the operation of the basal ganglia thalamocortical network. In stutterers the lateral premotor cortical-cerebellar network allows them to compensate for the weaker striatal-cortical system of connections (stutterers exhibit hyperactivity of lateral premotor regions). The heightened cerebellar activity correlates with increased speech fluency in stutterers. With time this hyperactivity becomes normalized with therapy but not entirely. In the course of inducing fluency by means of such methods as rhythmized utterances, or choral speech, the connections in the premotor cortex may possibly allow the stutterer to overcome difficulties caused by defects in the network based on connections between basal ganglia with the thalamus and cortex, and to introduce the needed timing for fluent speech. This happens owing to the use external sensory (auditory) cues. This may be why, despite the defects presented by the foregoing study, the "tricks" used in therapy methods can result in speech fluency.

The scholars in question also refer to the research results that show that in many patients with neurogeneric stuttering, basal ganglia and thalamus lesions were reported. But many of them also had a previous history of early childhood stuttering.

The reports on the use of medication were also discussed in the two studies. Many stutterers (not all) reported beneficial changes after the administration of dopamine antagonists (haloperidol, risperidone) whereas the administration of drugs enhancing dopamine levels (theophylline) intensified stuttering.

The interrelation between dopamine and stuttering was studied by O. Civier et al. (2013). A too high dopamine level in the brains of adult stutterers may the consequence of excessive stimulation of the thalamus and cause disorders in information transmission from the basal ganglia to the internal premotor cortex (especially affected by dopamine are the putamen cells). The motor cortex loses the ability to fluently pass from one motor sequence involving a syllable to the next one. Then what occurs is blocks, prolongation or repetition of the already said syllable. Production of syllables is delayed and stuttering occurs. The authors of that study tested their hypotheses using computer simulations of neural networks responsible for speech. They concluded that they had too few data to completely

corroborate the validity of their model but they believe with absolute certainty that stuttering cannot be explained only with one cause – a decrease in connectivity in the white matter in the basal ganglia-thalamocortical networks. They insist that this is indicated by for example the observable change in speech fluency of stutterers.

However, as has been said above, the studies by S. Chang and D. Zhu (2013) also show other deficits in cortical connections, particularly differences in connections supporting auditory-motor integration, which is necessary for fluent speech. The research by their predecessors did not take the auditory cortex into account: as has been earlier, emphasis was placed mainly on the abnormal anatomy of the left motor region and hyperactivity of the right hemisphere (which was explained as the result of the compensatory strategy for the left-side deficit). Meanwhile, the studies presented by the authors reported weaker connections between the posterior superior temporal gyrus, the insula, the supplementary motor area and the inferior frontal gyrus of the left hemisphere. The deficit was found only in stuttering boys and decreased with age (although the authors admit that greater deficit in smaller boys may have also been an artifact, which needs to be confirmed anyway). Disorders affect the white matter – long tracts of the connections of the frontal motor cortex with the auditory areas of the left hemisphere. This may result in the insufficient ability of the brain to compare a sound with motor performance (articulation) as part of feedback. If a mismatch occurs between the predicted auditory execution and the actual feedback, the auditory cortex sends corrective signals to the motor system to modify subsequent articulations, and the auditory cortex heightens its activity, the authors reported. This is an evident "hallmark of auditory influence on motor output" (cited after Chang and Zhu 2013) – deficit within this phenomenon results in disfluency.

In S. Chang and D. Zhu's investigations (2013) the fact of the occurrence of deficit in connectivity between the basal ganglia-thalamocortical "motor circuit" and auditory-motor areas is thoroughly documented. If these connections do not develop well in childhood, the timing of motor sequences is disrupted, which results in disruptions in auditory-motor planning, execution and control of utterances. This may further cause observed changes in adult stutterers that are the result of neuroplasticity: discrepant activity patterns in the left brain hemisphere, general heightened activity in motor areas, or hyperactivity of the right brain hemisphere during speech.

In the abovementioned 2015 study, differences within the white matter in the whole brain were assessed. The main objectives of the investigations were the following:

1. Verify the previous reports on differences in the white matter in the group of children with stuttering onset;

2. Determine possible differences in the white matter in stuttering children as compared with their nonstuttering peers;

3. Investigate specific areas of the white matter in which significant changes were reported that correlated with the severity and duration of the disorder.

Of special interest to the researchers were the areas defined in the previous study: the internal premotor cortex, premotor cortex, motor cortex, temporoparietal-occipital junction, arcuate fasciculus, and the posterior superior temporal gyrus. The investigations were meant to answer the question whether developmental changes in these regions also appear in the right hemisphere and whether they are associated with the corpus callosum, which underlies the connections in interhemispheric communication.

In comparison with the control group, SC exhibit reduced values of anisotropy along the left superior longitudinal fasciculus, including the inferior frontal gyrus (Brodmann area 44), premotor area (Brodmann area 6), motor cortex, and the inferior parietal lobe – the regions of the temporoparietal-occipital junction (Brodmann area 39 and 40). Interestingly enough, a right-side decrease in anisotropy value was reported, but smaller than the differences found in the left hemisphere. Reduced values were also reported in the structures of the cerebellum, brainstem, and the corpus callosum.

Interesting results are obtained from observations of the temporoparietal-occipital junction. It is the area where the sensorimotor cortex borders on the parietal cortex. The two regions are connected with the areas responsible for the motor activity of the articulatory organs, throat, breathing and larynx (during voice production). It was found that a larger number of connections of the white matter in this region correlate positively with speech fluency, and negatively with the severity of stuttering. This is also the case in the left longitudinal fasciculus. Negative correlations between the severity of stuttering and the number of connections in the areas in question were found mainly in boys except for the left longitudinal fasciculus, in which the correlation was greater in girls. Interestingly, the researchers observe that in the studied girls the symptoms of stuttering were not, however, as frequent and severe as in boys. They suggest that when diagnosing the degree of disfluency it is necessary to assess the frequency and duration of the three longest blocks and accompanying symptoms, taking into account the variables: the day, interlocutor and the speaking situation. They also point out that stuttering frequency may correlate with anatomy to a greater extent than with situational variables, which is a new conclusion.

Most cases confirmed the left-side deficits of connections of the inferior frontal gyrus, supplementary motor area, premotor cortex, temporoparietal-occipital junction, and the inferior parietal part. These areas are crucial to speech fluency, especially to feed-forward planning of utterances and control feedbacks. At the beginning of speech development, sensory feedback is more important, but after it

has developed, speech planning processes become more significant; these use auditory feedbacks. That is why delayed auditory feedbacks associated with a smaller number of connections or a slowdown in information transmission manifest themselves in speech fluency disorders. But weaker auditory-motor connectivity is only part of the picture of the phenomena that underlie stuttering.

The cortical connections involved in utterance forming are also influenced by subcortical connections and homologous areas of the right brain hemisphere. The authors found surprising facts: in the right hemisphere, reduced activity and a smaller number of connections is observed in the above-named regions, as is a smaller number of connections in the callosal commissure. This observation is puzzling in the context of the earlier observations of hyperactivity in the right hemisphere in adults because a greater number of connections and a greater corpus callosum could be assumed. In contrast, the reported right-hemisphere deficit in children regarding the connections in question can additionally cause disorders of prosody and rhythm, these being functions associated with the right hemisphere. With age, an increase in the number of connections in the corpus callosum and in the right hemisphere is observed, but at the same time there is an observable lack of the age-related left-side lateralization for language functions (which corroborates the observations of the weaker lateralization for language functions in stutterers). This may indicate a persistent left-hemisphere deficit of connections, which underlies stuttering, including also the growing age-related changes in the cerebral structure.

The 2015 study did not confirm changes in the putamen, but it reported disorders in cerebellar activity: lesser activity in the left cerebellar hemisphere, greater in the right.

DISCUSSION

The studies presented in the article support the thesis of the multifocal structural and functional disorder of brain connections, which underlies stuttering. Moreover, anatomical determinants of some therapeutic measures were explained, for example the fact of inducing speech fluency to the externally paced rhythm. Conclusions can also be drawn about the positive effect of musical training on brain development. Other conclusions concerning therapy may stem from the fact that despite persistent structural disorders, stutterers sometimes speak fluently. There are known cases when this state lasted even several weeks. This raises hopes that other systems allowing control of speech fluency can be used and that a permanent change in the dynamic course of programming and controlling functions will be achieved. In some cases this may mean that speech motor control will be based on the use of proprioreceptive rather than auditory feedback.

An interesting, in a way secondary, observation made during the investigations is that the authors reported a subtle decrease in the results of SC as compared with NSC in the standardized language use. The observation, interpreted from the perspective of the found anatomical differences, may correct the present classificatory and therapeutic approach to stuttering. Perhaps stuttering is not only a production disorder? Perhaps treatment should also take competence building and developing exercises into consideration? At any rate, this observation opens a promising research perspective.

Finally, it is essential to consider the above-mentioned theories of the origin of stuttering in the context of new discoveries.

Firstly, the findings confirm the interpretation of the phenomena, described by E. Szeląg (1999), of the origin of disfluency caused by disorders of timing integration as a delay in the "closure" of the control loop and comparison of input information with output information (cf. Woźniak 2015). Studies by S. Chang, D. Zhu et al. (2013, 2015) provide strong evidence of disorders in the left hemisphere connections between the auditory cortex and motor areas responsible for utterance planning and execution. Weakened functional and structural connections between these areas significantly impact the time of information processing and cause disfluency. Furthermore, it should be concluded that a similar influence can also result from problems in forward planning of utterances which pertain to the motor and premotor cortex in the left hemisphere or connections of the basal ganglia, thalamus and the cortex. The disorders of the "brain clock", described by E. Szeląg are only a symptom of structural and functional brain disorders, and in my present interpretation they (the former) can be entirely explained by the latter**.** Therefore, the theory of disorders in the "brain clock" as the cause of stuttering is modified: both timing disorders and fluency disorders arise as a result of weakened connections between various links in the speech chain in the brain.

Secondly, a separate consideration is needed for the theory of the origin of stuttering due to disordered auditory lateralization. Z. M. Kurkowski's studies (2013) on audiogenic conditions of speech disorders provided substantial arguments to support the thesis of A. Tomatis, who in the mid-twentieth century pointed out the disorders of auditory lateralization as the fundamental cause of stuttering. The results of Z. M. Kurkowski's investigations show that 65% of stutterers show the left-ear advantage in auditory self-control (taking into account the kind of conduction of speech signal, in this case: bone conduction). At the same time 75% of those tested showed the right ear dominance for understanding speech.

What does the lack of harmony between the right-ear predisposition to receive speech sounds and the left-ear dominance in the self-control of one's utterance mean? Left-ear hearing causes a signal to pass first to the right hemisphere, and then information is transmitted through the corpus callosum to the left hemi-

sphere (Kurkowski 2013: 219–220). The consequent prolonged time of auditory self-control may become a cause of disfluency.

Moreover, left-earedness can explain the growing difficulties in situations producing negative emotions: they activate the right-hemisphere and additional disorders of the control signal. The conception of the contribution of auditory lateralization disorders to the origin of stuttering helps explain: the occurrence of stress-induced stuttering (stress factors may impact listening strategies, and, during the period of up to five years of age, even the model of auditory lateralization), relations between stuttering and lateralization disorders in handedness (it is probable that switching a left-lateralized person to the right hand can sometimes produce a similar effect if the motor area develops in the left hemisphere) (Woźniak 2015: 804)

In light of the presented data concerning structural and functional disorders in the brains of stutterers, possible variants of the occurrence of left-earedness in stutterers should be taken into account.

Variant One assumes that left-earedness occurs as a compensatory strategy aiming to level out deficits within motor and auditory cortex interconnectivities in the left brain hemisphere. The increased activity of the right hemisphere in the auditory cortex is confirmed by the presented studies. The weakened brain lateralization in the area of speech in stutterers is also confirmed; there is a continuous left-hemisphere dominance of language functions, with hyperactivity of the right hemisphere and its gradual structural increase. In this variant, disorders of auditory lateralization are also a consequence rather than a cause of stuttering. With time, however, the left-ear dominance may become a factor that intensifies disfluency, for example in stress situations. This would occur in children, in whom symptoms of disfluency already appear very early.

Variant Two should be considered in a situation when stuttering occurred at later age: in childhood but not early childhood, or during adolescence or adulthood. There are two possibilities then:

1. The disorder of auditory lateralization is the main cause of disfluency;
2. The disorder of auditory lateralization is a factor that triggers stuttering, active in the context of other functional and structural deficits of connections in the brain, for example the number of connections in the corpus callosum.

The second variant appears more probable because 17% of the population with speech located in the left hemisphere show the left-ear dominance in auditory control (Kurkowski 2013: 138) whereas stuttering is reported in less than 1% of the population (and for various reasons). It is certainly necessary to additionally explain the links between auditory self-control strategies and environmental factors (e.g. stress), and structural and functional connections in the brain.

Further studies on stutterers, taking into account speech therapy assessments, the time when stuttering occurred, results of neuroimaging examinations, auditory

processing tests and anxiety scales, could find the answer to the foregoing questions. It is very unlikely that emotional factors will be completely excluded from the etiology of stuttering and replaced with anatomical factors only.

In light of the studies by S. Chang, D. Zhu et al. (2013, 2015) it is also justified to seek genetic correlates of the presented neuroanatomical changes. Most probably, it is unlikely that the multifocal structural and functional disorder of connections in the brain does not have genetic causes.

The presented studies therefore largely verify the current views on the causes of stuttering and open new research perspectives.

BIBLIOGRAPHY

Civier O., Bullock D., Ludo M., Guenther F., 2013, *Computational modeling of stuttering caused by impairments in a basal ganglia thalamo-cortical circuit involved in syllable selection and initiation*, "Brain and Language", 126 (3), pp. 263–278

Chang S., Zhu D., 2013, *Neural network connectivity differences in children who stutter*, "Brain", 136, pp. 3709–3726

Fox P., Ingham R., Ingham J. C., Zamarripa F., Xiong J. H., Lancaster J. L., 1996, *A PET study of the neural system of stuttering*, "Nature", 382, pp. 158–161

Kurkowski Z. M., 2013, *Audiogenne uwarunkowania zaburzeń komunikacji językowej*, Wydawnictwo UMCS, Lublin.

Sommer M., Koch M. A., Paulus W., Weiller C., Büchel C., 2002, *Disconnection of speech-relevant brain areas in persistent developmental stuttering*, "Lancet", 360, 8, pp. 380–384

Szeląg E., 1999, *Nowe metody terapii wyzwaniem XXI wieku, "*Logopedia", 26, pp. 215–224.

Woźniak T., 2002, *Przyczyny i terapia jąkania w świetle nowych badań,* "Biuletyn logopedyczny", 3 (9), pp. 52–54

Woźniak T., 2015, *Diagnoza i terapia osób z zaawansowanym jąkaniem,* [in:] S. Grabias, J. Panasiuk, T. Woźniak (eds.), *Logopedia. Standardy postępowania logopedycznego*, Wydawnictwo UMCS, Lublin, pp. 797–835