

Stuttering: A Diagnostic Dilemma

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When an adult who has stuttered in childhood, experiences a sudden reonset of stuttering, the clinician has to clarify whether this "adult onset" stuttering is the result of brain damage, and thus will be labelled as a primary neurological stuttering. It also can be the reappearance of the "cured" developmental stuttering, which may be brought out by a special event, and therefore is mostly labelled psychogenic stuttering.

1. Introduction

The last decades of research on developmental stuttering have given birth to several definitions of developmental stuttering, some of which characterise developmental stuttering by "involuntary repetitions and prolongations of small linguistic elements (phonemes, syllables, monosyllabic words)" (Wingate, 1964; Andrews & Harris, 1964). Other authors speak of "a deviation of speech that effects adversely the speaker or listener because of an interruption of the normal rhythm of speech by involuntary repetition, prolongation or arrest of sounds" (Espir & Rose, 1970). These definitions essentially *describe* what every listener can observe when stuttering occurs, but do not refer to the underlying causes which still remain obscure.

On the other hand definitions of acquired stuttering (that mostly occurs in adulthood) are usually based on the same framework, even if the location of a cerebral lesion is clearly established. Helm et al. (1978) use the definition of Espir and Rose (1970), and Rosenbek (1984) follows the definition of Wingate (1964), when he states: "*Neurogenic stuttering is the repetition, primarily, of correct sounds and syllables*" (p.37). By using the same type of definitions, the authors indirectly accept the idea that the audible dysfluencies one can hear in the case of acquired stuttering are similar to those of the developmental stuttering, although these ideas were denied by Canter (1971). According to the last author, the symptoms of the two types of stuttering are different.

If there is a growing knowledge about the onset of acquired stuttering, thanks to the studies with PET scan and MRI (Braun et al., 1997; Demonet et al., 1992, 1994; Wise et al., 1991), we still know very little about the causes of the sudden recurrence of stuttering in adults. This may happen after brain damage, trauma or shock. The question then is to make clear whether this stuttering is neurogenic in its origin or

not. Indeed, if this « second manifestation » of stuttering is the clearly demonstrated consequence of brain injury, we can speak of primary acquired or neurogenic stuttering, even if the person presented with developmental stuttering in childhood. On the other hand, the reappearance of stuttering in adulthood can be a new manifestation of the « old », presumably cured, developmental stuttering, that suddenly breaks out under particular circumstances, and can be of psychogenic origin (Deal, 1982; Tippett & Siebens, 1991; Roth et al., 1989; Stewart & Grantham, 1993).

The following two case studies will be analysed in light of:

1. The circumstances in which the stuttering reappeared, and
2. The personal background of the patient.

2. Case reports

2.1. Patient M.M.

M.M. has suffered developmental stuttering and has come in for therapy for the first time for about two years. He then was 30 years old and had stuttered since his childhood, but he never had followed speech therapy. As to his personal background, he described his youth as unhappy with an aggressive, authoritarian father and an anxious mother. At the time of therapy, he himself had become an authoritarian husband and father. He lived with clichés about the social roles of men and women.

His stuttering is characterised by frequent repetitions of fist sounds: « p.t.k.b.d.g » (*p-p-petit; t-t-tout; q-quand; b-bon; d-d-dans; g-g-garage*) as well as by prolongations of initial consonants « s.m.f. » (*sssans; mmmais; ffour*). There are never more than three repetitions of the same sound. The prolongations are often accompanied by repetitions (*sss-ss-ssans; mmm-mm-mmmmais; ff-fff-ffour*); these two dysfluencies appeared to be intimately waved together in this patient. During the repetitions and prolongations, he showed concomitant behaviours: trembling of the right eyelid and the right corner of his mouth. He avoided eye contact during communication, and his speech output was very fast, so that it was sometimes hard to understand him. He only stuttered in front of his father, male superiors and men with a high social position. -He never stuttered with women. But he stuttered with me. The therapy was based on relaxation, cognitive approach of personality problems, and classical speech therapy with soft onset, easy speech flow etc. The cognitive approach and the soft onset gave good results and two years later M.M. can leave therapy.

March 1997 he suffered a road accident in which his motor was hit by a car that caused cranial trauma and a fracture of the shoulder. He was unconscious for 15 minutes. Two months later, M.M. developed stuttering symptoms. Neuropsychological examination reveals that his short-term memory is bad. The patient has difficulties in retaining what has just been said or planned (After having read a story, he is unable to recall what he just has read; repetition of short sentences is correct, but repetition of long sentences is hesitant). The patient suffers from concentration problems, when working for more than 15 minutes. On the other side, he is well-oriented. His sleep is

disturbed and he frequently wakes up during the night. He is complaining about diarrhoea and chronic headache. MRI examination doesn't reveal abnormalities. However, the difficulties in short-term memory, concentration problems and sleep disturbances are the cause that M.M. hasn't been able to go back to work since his accident.

His stuttering is characterised by the same repetitions of initial sounds as before, but there are more repetitions of the same sounds and in addition, he repeats parts of words (the first syllable) and small words as well (*ma si-situation fa-familiale; le-le-le but*). He also frequently pauses or stops when speaking (*ac// centué, et// je me rends compte// que... admini//stra//tif; tem//po//raire; je vous tiens//au courant...*), at places where normally one doesn't stop. A new aspect of his speech output is the strikingly monotonous and very slow speech; he seems to search for the right word.

Besides his complaints about his stuttering, he also suffers depression. He can't cope with daily problems any more. On the other hand it is not clear if the depression that he says to experience, is a direct consequence of the brain damage or the consequence of his incapability to cope with everyday life.

The linguistic impairments as monotonous and slow speech, aberrant pauses, word finding problems and difficulties with short-term memory, that clearly refer to aphasic elements remain present during the whole therapy that will go on for one year.

2.2. Patient R.K.

R.K. is a French-speaking woman of 46 years old, who is referred to me for stuttering diagnosis. She is born in Zaire, and was brought up in an orphanage of Lubumbashi, where she stayed until her 6th year. Her brother and sisters stayed with the grand parents. From her 6th to 14th year, she lived with her grand parents too, where she started to stutter. She doesn't remember why nor how she stuttered. The stuttering stopped without therapy (and without any objective reason) when she started to work at 14 years and when she gradually left the family. At age 17 she met a Belgian colonist (30 years older than she) whom she married. When she is 22 years old (1977), the family comes to Belgium, where she discovers that her husband already had a wife and two children. From 1977 to 1999 the marital situation worsens slowly, but suddenly considerably, because of the growing jealousy of the old husband. He becomes aggressive and beats his wife. In 1999 she discovers that her husband deceives her with several women while she is at work.

According to her own words, she started to stutter at that very moment, because of the stress caused by her husband who she can't bear anymore, who she is afraid of, but who is ill now (cancer) and who she morally can't leave. In the beginning of her trouble, she only stuttered with her husband, but gradually the stuttering became general, and she now stutters in all situations of everyday life and with everybody. She also is complaining about frequent headache.

The stuttering has a quite different pattern from what one generally observes in the clinic. She nearly stutters on every word. She shows blocks on consonants and vowels (*p, t, k, b, d, g, a, u*); she repeats initial consonants (*p, t, k, b, d, g, l, m, n, j*),

and she shows repetitions of syllables and one-syllable words (*mal-mal-mal à la Vvtête, à la nu-muque*). She frequently interrupts her sentences which are often incomplete or incorrect (*j'ai-j'ai-j'ai //ça-ça-ça //nani Vvtéléphone //di-di-dificile*). She also uses many interjections and expletives (*eh, nani, ça veut dire, oh là là*). These interjections and expletives can be repeated several times, and are manifest in nearly every sentence. Reading as well as automatic series (1-10, 1-20) are affected in the same way. The automatic series 1-20 is pronounced very slowly and with a constant effort. There is a block before « dix and quatorze » (10 and 14). She shows the same difficulties during rehearsal of 1-20, during telling the names of the days of the week, and the months of the year. However, neurological examination (January 2001, CT scan with and without contrast) doesn't reveal any abnormality.

3. Discussion

One of the intriguing aspects of stuttering is the fact that it can be worsened or alleviated by neurogenic disease, and these changes can be transient or permanent. Helm et al. (1986) describe a patient who ceased to stutter after a head injury, and Miller (1985) relates the case of the cessation of stuttering with progressive multiple sclerosis. The case of Vicky (Cooper, 1983) shows the transient but complete cessation of stuttering for 6 weeks, after brain-stem contusion. But when the injury gradually healed, the stuttering returned. Helm et al. (1986) describe another patient (left-handed) who suffered a CVA (lesion in the brainstem) after which his childhood stuttering came back. This case is similar to our first patient, but with the restriction that in our patient no brain lesions could be clearly detected. One of Rosenfield's (1980) patients, on the other hand, experienced an aggravation of his trouble after having suffered a lesion in the brainstem.

Monotonous slow speech (as was the case in our first patient) and concomitant acquired stuttering was described by Lebrun et al. (1986). The stuttering was the first indication of Parkinsonism. Downie et al. (1981) and Koller (1982) also mentioned acquired stuttering in the presence of Parkinsonism. But our patient didn't develop Parkinsonism during the time he came in for therapy.

The slow and monotonous speech output of our patient reminds me of another Dutch patient who was referred to me by Dr. H. van Dongen (Dijkzigt Ziekenhuis, Rotterdam). This man underwent two heart operations, one followed by a coma. After the operation he was tired, depressive and couldn't concentrate on his work anymore. He was complaining of headache as well. Two months later, he started to stutter, manifested aberrant pauses and had a slow, monotonous speech output. The neurological examination here was non-conclusive and showed no abnormalities. Six months later however, a MRI showed a loss of perfusion in the arteria cerebri media and posterior, but the scan was normal.

This last case might give us some insight in the possible origin of the recurrence of stuttering in our patient. It might be that he suffered a slight brain damage that was (or

is) not visible on the scan, but that caused his stuttering, i.e. primary acquired stuttering, with slow monotonous speech, frequent stops and pauses and some word finding problems. The typical stuttering pattern (repetitions and prolongations) was that of his original stuttering but aggravated, like it was the case for Rosenfield's (1980) patient.

M.M. is complaining about his impossibility to concentrate and work. Concomitant with his lack of concentration he feels depressive. The depression and anxiety is often mentioned as one of the consequences of brain lesion. Sapir and Aronson (1990), Roth et al. (1989), and Gainotti (1972) observed depression and anxiety in their patients with a lesion in the left hemisphere. For these authors it was clear that the depression and anxiety were a direct consequence of the brain damage and not the consequence of the existing stuttering. Sapir and Aronson stated: "*Depression and anxiety have been linked to disturbances of the basal ganglia, thalamus, limbic system and frontal lobe. These same anatomical structures are also important in voice, speech, language and non-speech motor functions. These findings suggest that depression and anxiety are likely to coexist with voice, speech, or language disorders as a result of central nervous pathology*" (p. 503), and they added: "*anxiety is known to worsen existing speech disorders, e.g. stuttering*". These statements clearly remind us to our patient, even if the scan and MRI couldn't reveal objectively brain damage. In light of all the mentioned elements, I would diagnose that M.M. experienced a primary acquired stuttering following unknown brain damage after a road accident. The manifestation of it was an aggravation of the symptoms of his old developmental stuttering syndrome, with slow, monotonous speech output and word finding difficulties that make us think of possible aphasic elements. Depression and anxiety became dominant symptoms of his trouble.

In the second case, my special attention was raised by the particular speech pattern the patient manifested at the beginning of therapy. Indeed, a stuttering person rarely stutters on nearly every spoken word. Roth et al. (1989), Sapir and Aronson (1990), Deal (1982, 1987), Weiner (1981), Tippett and Siebens (1991), as well as Stewart and Grantham (1993) describe sudden onset of stuttering in adulthood due to psychological stress. Some authors (Roth et al., 1989) stress the particular stuttering pattern in psychogenic stuttering. They also notice the possible dramatic changes that can occur when the causes of the trouble are disclosed. One of my own patients, a young lady of 21 year who had never stuttered in her life, experienced stuttering the very day that she was eliminated for the Olympic Games. When she came in for therapy, she stuttered on every word and she was badly oriented. After some sessions, she could express her anger and deception of not having been selected for the Olympics. The following sessions she did not stutter anymore.

When R.K. comes in for therapy, she can hardly be understood because of her constant repetitions, blocks, interjections and phrase revisions. This speech pattern and the circumstances in which her stutter re-appeared (after a shock and heavy marital problems), decide me to concentrate on psychological and relational aspects in therapy, besides the classical speech technics. The therapy based on relaxation, psychological approach of the marital problems, self-esteem, and speech therapy, give

very quickly good results. The changes in R.K's speech output are as dramatic as in the just mentioned case. After a few therapy sessions, when she has explained how she lives with her husband, and how he treats her, she seems relieved. The next therapy session, she relates the changes that have occurred in her life, i.e. she understands the role her husbands plays and the way he wants to keep her down. When telling this, she stutters slightly, but then the stuttering symptoms simply seem to fade away. During the following sessions, she doesn't stutter anymore. She sometimes stutters when entering the room, but she soon gets back to fluent speech. She now comes in from time to time, just to show me that she is fine.

According to the patient, she only stutters at home, with her husband, and when she is very stressed by a particular situation, but her stuttering does not last anymore. She feels free and she can decide for her life. She feels all right, and she doesn't stutter anymore. In light of mentioned elements, the recurrence of stuttering in this patient, that appears to be clearly limited to some few situations, seems to be of psychogenic origin.

4. General conclusions

Recurrence of stuttering in adulthood is not well studied until now. The few existing studies show the complexity of the trouble in function of the eventual neurological and psychological background history of each patient. This study shows the importance of careful diagnosis with all elements being taken into account.

Samenvatting

Bij het plotselinge terugkeren op volwassen leeftijd van overwonnen ontwikkelingsstotteren moet de vraag gesteld worden naar de eventuele neurogene of psychogene oorzaak van dit verworven stotteren. In dit artikel worden twee gevallen van terugkerend stotteren op volwassen leeftijd geanalyseerd in het licht van bovenstaande vraagstelling.

References

- Andrews, G., & Harris, M. (1964). The syndrome of stuttering. *Clinics in Developmental Medicine*, 17. London: Heinemann.
- Braun, A.R., Varga, M., Stager, S., Schulz, G., Selbie, S., Maisog, J.M., Carson, R.E., & Ludlow, C.L. (1997). Altered patterns of cerebral activity during speech and language production in developmental stuttering: An H² 15 O positron emission tomography study. *Brain*, 120, 761-784.
- Canter, G.J. (1971). Observations on neurogenic stuttering: A contribution to differential diagnosis. *British Journal of Disorders of Communication*, 6, 139-143.

- Cooper, E.B. (1983). A brainstem contusion and fluency: Vicki's story. *Journal of Fluency Disorders*, 8, 269-274.
- Culatta, R., & Leeper, L. (1988). Dysfluency isn't always stuttering. *Journal of Speech and Hearing Disorders*, 53, 486-487.
- Deal, J. (1982). Sudden onset of stuttering: A case report. *Journal of Speech and Hearing Disorders*, 47, 601-303.
- Demonet, J.F., Chollet, F., Ramsay, S., Cardebat, D., Nespoulos, J.L., & Wise, R. (1992). The anatomy of phonological and semantic processing in normal subjects. *Brain*, 115, 1753-1768.
- Demonet, J.F., Price, C., Wise, R., & Frackowiak, R.S.J. (1994). Differential activation of right and left posterior regions by semantic and phonological tasks: a positron-emission tomography study in normal human subjects. *Neuroscience Letters*, 182, 25-28.
- Downie, A.W., Low, J., & Lindsay, D. (1981). Speech disorder in Parkinsonism: use of delayed auditory feedback in selected cases. *Journal of Neurology, Neurosurgery, and Psychiatry*, 44, 852-853.
- Gainotti, G. (1972). Emotional behavior and hemispheric side of the lesion. *Cortex*, 8, 41-55.
- Helm, N.A., Butler, R.B., & Benson, F. (1978). Acquired Stuttering. *Neurology*, 28, 1159-1165.
- Helm-Estabrooks, N. (1986). Diagnosis and management of neurogenic stuttering in adults. In St. Louis, K. (Ed.), *The Atypical Stutterer* (pp. 193-217). Orlando: Academic Press.
- Helm-Estabrooks, N., Yeo, R., Geschwind, N., Freedman, M., & Weinstein, Ch. (1986). Stuttering: disappearance and reappearance with acquired brain lesions. *Neurology*, 36, 1109-1112.
- Koller, W.C. (1982). Dysfluency (stuttering) in extrapyramidal disease. *Archives of Neurology*, 40, 175-177.
- Lebrun, Y., Devreux, F., Rousseau, J.-J. (1986). Language and speech in a patient with a clinical diagnosis of progressive supranuclear palsy. *Brain and Language*, 27, 247-256.
- Miller, A.E. (1985). Cessation of stuttering with progressive multiple sclerosis. *Neurology*, 35, 1341-1343.
- Rosenbek, J. (1984). Stuttering secondary to nervous system damage. In Curlee, R.F., & Perkins, W.H. (Eds.), *Nature and Treatment of Stuttering: New Directions* (pp. 31-48). San Diego: College-Hill.
- Rosenfield, D.B., Miller, S.D., & Feltovich, M. (1980). Brain damage causing stuttering. *Transactions of the American Neurological Association*, 105, 1-3.
- Roth, C.R., Aronson, A.E., & Davis, L.J. (1989). Clinical studies in psychogenic stuttering of adult onset. *Journal of Speech and Hearing Disorders*, 54, 634-646.
- Sapir, S., & Aronson, A. (1990). The relationship between psychopathology and speech and language disorders in neurologic patients. *Journal of Speech and Hearing Disorders*, 55, 503-509.
- Stewart, T., & Grantham, C. (1993). A case of acquired stammering: the pattern of recovery. *European Journal of Disorders of Communication*, 28, 395-403.
- Tippett, D.C., & Siebens, A.S. (1991). Distinguishing psychogenic from neurogenic dysfluency when neurologic and psychologic factors coexist. *Journal of Fluency Disorders*, 16, 3-12.
- Weiner, A.E. (1981). A case of adult onset stuttering. *Journal of Fluency Disorders*, 6, 181-186.
- Wingate, M.E. (1964). A standard definition of stuttering. *Journal of Speech and Hearing Disorders*, 29, 484-489.
- Wise, R., Chollet, F., Hadar, U., Friston, K., Hoffner, E., & Frackowiak, R. (1991). Distribution of cortical neural networks involved in word comprehension and word retrieval. *Brain*, 114, 1803-1817.