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Psychogenic Stuttering

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Differentiating between various types of late-onset stuttering is not always straightforward. Especially the differential diagnosis between psychogenic and neurogenic stuttering may be difficult, as the case of I.V. illustrates. I.V. was a 15-year-old girl who started to demonstrate dysfluent speech after she suffered a cerebral trauma. Although the hypothesis that I.V.'s stuttering was of neurogenic origin was quite plausible, there were reasons to believe that her dysfluency was not an instance of neurogenic stuttering but rather had a psychogenic origin.

1. Introduction

Stuttering is a speech disorder that is defined by the World Health Organization as a disorder "in the rhythm of speech in which the individual knows precisely what he or she wishes to say but at the time is unable to say it because of an involuntary repetition, prolongation, or cessation of a sound" (World Health Organization, 1977). There is ample evidence that stuttering usually has its onset in childhood and in most cases manifests itself for the first time somewhere between the ages of two and five (Silverman, 1992). Van Riper (1971) even claimed that the fact that stuttering usually begins in childhood is "one of the few solid bits of information we have about stuttering" (p.62).

Yet, dysfluent speech may appear also for the first time later in life, beyond the typical childhood period. While not everyone seems to agree that a dysfluency that is observed for the first time in adolescence or adulthood should be called stuttering (see for instance Culatta & Leeper, 1988) it is common nowadays to recognize the existence of at least two varieties of stuttering. Stuttering that originates in childhood is then called "developmental stuttering". Stuttering that originates after the typical childhood period is called "late-onset stuttering" or sometimes also "adult-onset stuttering" or "acquired stuttering".

Late-onset stuttering may have various causes and different types of late-onset stuttering can be distinguished accordingly. Of course there is always the suspicion in cases of late-onset stuttering that the dysfluency is actually a recurrence of childhood stuttering, and some persons who start to stutter for the first time in adulthood may have been interiorized stutterers, who under great stress could no longer hide their disorder. Apart from these possibilities, already considered by Van Riper (1971), there would seem to be four different types of late-onset stuttering.

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Most often stuttering which presents for the first time after childhood derives from damage to the central nervous system. One speaks then of neurogenic stuttering. Neurogenic stuttering does not appear to be linked to any specific lesion side. The damage that lies at the origin of the dysfluencies in neurogenic stuttering may be unilateral or bilateral, focal or diffuse, cortical or sub-cortical, situated in the right or in the left hemisphere; within one hemisphere it may be localized in the frontal, temporal or parietal lobe. The symptoms in neurogenic stuttering are often quite similar to those of developmental stuttering and on the basis of symptomatology only, distinguishing between neurogenic and developmental stuttering may be very difficult, even for speech language pathologists experienced in dealing fluency disorders (Van Borsel & Taillieu, 2001).

Late-onset stuttering may also be a side effect of medication, in which case it can be called pharmacogenic stuttering. Three types of drugs in particular have been reported to possibly elicit stuttering: antidepressants, neuroleptics and anti-convulsants. In addition, there are a few cases of stuttering following the administration of theophylline, a broncho dilatator (McCarthy, 1981; Gérard et al., 1998; Rosenfield et al., 1994). Looking at the symptoms in cases of pharmacogenic stuttering, it is hard to discern any particular patterns of dysfluency associated with any particular type of drug. Even one and the same drug may apparently elicit a heterogeneity of symptoms. There seems to be a tendency, however, for repetitions to be the most frequently occurring symptom in cases of pharmacogenic stuttering, whereas secondary symptoms would not seem to be typical of pharmacogenic stuttering (Beck, 2000).

Another type of stuttering of late-onset is malingered stuttering. Cases of this type of stuttering have been reported by Shirkey (1987) and by Bloodstein (1988). In each case the stuttering presented in a forensic context. As the latter author pointed out, establishing positive evidence of malingering may be difficult and a satisfactory means of detecting malingering in stuttering remains to be found.

Finally, late-onset stuttering may be of psychogenic origin related to some form of psychological stress or emotional trauma. The existence of psychogenic stuttering was already recognized by Head in 1922 but compared to other types of late-onset stuttering, psychogenic stuttering has received far less attention in the literature. And although a number authors have reported on the characteristics of psychogenic stuttering, diagnosing psychogenic stuttering is not so straightforward. Especially the differential diagnosis with neurogenic stuttering may be difficult, as the following case of I.V. illustrates.

2. Case presentation

I.V. was 15 years old when she was overthrown by a motor vehicle. Upon admittance to the University hospital a cerebral trauma, a fracture of the acetabulum and the pubis and a hemopneumothorax were diagnosed. A initial CT scan showed a small intracerebral bleeding near the left cerebral peduncle. A control CT performed a few

days later revealed also a hyperdense area at the crus posterior of the internal capsula on the left with surrounding oedema. After being comatose for about five weeks I.V.'s condition gradually improved and she was transferred from the neurosurgery ward to the hospital rehabilitation center. A neurolinguistic examination about 5 months after the accident showed that comprehension was fairly good, with perfect ability to execute simple and complex commands in either oral or written form. Yet I.V.s score on the Token test (Van Dongen et al., 1976) was slightly reduced (49/61). Oral expression was virtually impossible. I.V. could not produce any audible sound, respiratory control was very poor, and volitional oral movements were very slow (to wiggle one's tongue, to lick one's lips, to pucker one's lips, to show one's teeth) or impossible (to click one's tongue, to kiss). Neither did I.V. manage to hum, clear her throat or cough on request. Written expression, on the other hand, was quite possible by means of a letterboard. Using this means, I.V. could produce complete and correctly spelled answers in reply to questions. On the basis of the above findings, it was concluded that I.V. was primarily dysarthric.

I.V. was again referred to us more than half a year after the first examination. During that time she had been receiving speech therapy focusing on improvement of respiratory control, on the elicitation of phonation and on improvement of articulation. In addition she had received occupational and physical therapy. As far as her communication problems was concerned, I.V. had made considerable progress. She was no longer dependent on a letterboard to express herself. Her voice was still weak but clearly audible and articulation was slow but otherwise correct. Her score on the Token test (56/61) now definitely ruled out an aphasia. Verbal fluency, however, as measured by the subtest "diernamen noemen" from the S.A.N. test, a Dutch standardized aphasia battery (Deelman et al., 1981), appeared to be reduced (7th percentile). The reason for referral this time, however, had not to do with the communication problems discussed so far. I.V. was referred now because she had developed a marked dysfluency.

An analysis of the dysfluency, based on video-recordings revealed that the main type of dysfluency demonstrated were blocks. In a sample of conversational speech, blocks accounted for over 80% of the dysfluencies. The remainder of the dysfluencies were all prolongations. Other types of dysfluency did not occur. The blocks and prolongations only affected consonants in syllable-initial position. Vowel sounds and syllable-final consonants were never affected. In polysyllabic words, it was most often the initial syllable of the word that was affected. The blocks and prolongations further appeared to occur primarily but not exclusively on lexical words and were produced without any accompanying accessory features such as facial grimacing, movements of the head, blinking of the eyes or quivering of the nostrils. Duration of the blocks tended to be somewhat longer than that of the prolongations (respectively up to five seconds versus one to two seconds). When blocks occurred on alveolar plosives, the inferior surface of the tongue would often protrude visually between the incisors while the tip of the tongue made firm contact with the alveolar ridge and upper incisors. I.V. appeared to be dysfluent not only during conversational speech

but also during serial and imitative speech, when reading aloud, or when singing. In these speech modes too, blocks and prolongations were the only symptom with blocks being predominant. It was also found that repeated reading of the same passage did not evidence an adapatation effect and that reading under masked feedback did not result in an improvement of fluency.

On a second evaluation of the dysfluency, about one and half month after the first one, a similar picture was observed. I.V. Was still highly dysfluent demonstrating blocks and to a lesser degree also prolongations in each of the conditions sampled previously.

Still three months later we saw I.V. for a third assessment. Now a dramatic change was observed. Analysis of a sample of conversational speech revealed not a single dysfluency. During serial and imitative speech and during reading aloud, only a few occasional blocks occurred. These blocks were of short duration, without protrusion of the tongue when affecting alveolars. It seemed as if the dysfluency had almost completely cleared up.

3. Discussion

One may wonder what type of stuttering was demonstrated by this patient. It is not very likely that we had to do with developmental stuttering or a recurrence of developmental stuttering. I.V's mother denied any history of stuttering in her daughter. Neither was there a family history of stuttering as is often seen in developmental stuttering. And I.V. was more than 15 years old when she started to demonstrate dysfluent speech for the first time, which is far beyond the typical childhood period.

It is not very likely either that her stuttering was drug-induced. During her 11 months' stay at the hospital rehabilitation center I.V. was not administered any antidepressant or anticonvulsant medication nor any neuroleptic drug.

What about the hypothesis that I.V. was malingering? On the one hand, there was some change in I.V.'s condition in the period that she started to stutter that could perhaps have given rise to malingering. In that period individual speech therapy had been replaced by group therapy and I.V. had uttered some complaints about not receiving more individual therapy. It is not unthinkable that she should have started to stutter in order to obtain again the individual attention she was receiving before she was enrolled for group therapy. On the other hand, however, the nature of her dysfluency was very consistent with patterns of genuine stuttering. She stuttered on consonants, not on vowels; as a rule she stuttered on syllable-initial consonants, and in polysyllabic words it was most often the initial syllable of the word that was affected. Moreover, she did not produce repetitions of any kind. Yet, repetition is the symptom of stuttering that is probably best known to non-professionals and as such the symptom that one would certainly expect in malingered stuttering. Therefore, it is in our opinion not very likely that I.V. should have been malingering.

Another, more likely possibility is that I.V.'s stuttering was of neurogenic origin.

After all, she became dysfluent after a motor vehicle accident resulting in documented brain damage. Moreover, I.V. showed several of the features which, at least according to some authors (see for instance Helm et al., 1978; Helm, 1993), are typical of neurogenic stuttering. Among these are the observation that the stuttering occurred across speech tasks, that the stuttering was unaccompanied by accessory behaviour such as facial grimacing or eye blinking, that dysfluencies occurred on both function words and lexical words, that speaking under loud masking noise did not result in more fluent speech, and that there was no clear evidence of an adaptation effect.

And yet, there is some doubt whether the dysfluency observed in I.V. was definitely of neurogenic origin. It would seem that a diagnosis of psychogenic origin is more plausible. To begin with, we should remark that some of the features which are considered indicative of neurogenic stuttering by some investigators, have also been reported to be typical of psychogenic stuttering. According to Deal (1982), for instance, symptoms such as the occurrence of the dysfluency across different speech conditions and the absence of secondary features are also typical of psychogenic stuttering.

A finding that at any rate is not really typical of neurogenic stuttering, is the delayed onset of the fluency disorder. In neurogenic stuttering the occurrence of dysfluency is usually more directly associated with the neurological insult. Findings by Market et al. (1990) suggest that the large majority of neurogenic stutterers (more than 80%) start to stutter within one month of their insult. I.V. first started to stutter almost a year after the accident.

Among the features that definitely suggest a psychogenic origin are the sudden onset of the dysfluency and the coincidence with psychological stress. An interview with I.V.'s mother revealed that I.V. had developped her marked dysfluency from one day to the next. And this happened in a stressful period. At the time I.V. developped her stuttering, she was still an inpatient at the hospital rehabilitation center but her discharge from hospital was pending. There was some discussion, however, whether it would not be more beneficial to delay discharge until after an operation which I.V. still had to undergo to improve her mobility. As mentioned already above, at about the same time, individual therapy had been replaced by group therapy, a change about which I.V. uttered complaints. Moreover, I.V. was apparently not very motivated any more, and several times she did not turn up for therapy. Clearly, this period was fraught with psychologically significant events for I.V.

A characteristic that according to Baumgartner and Duffy (1997) and Baumgartner (1999) is an important clue to the identification of psychogenic stuttering, is the presence of a "bizarre" speech pattern. It is perhaps hard to clearly define what constitutes "bizarre" behaviour. At any rate, the pattern of blocks displayed by I.V. did not compare to any of the patterns we saw before in our clinical work with developmental stutterers.

Another feature listed among the characteristics of psychogenic stuttering by Tippet and Siebens (1991) is that a decrease in the dysfluency is usually accompanied by a general improvement in the patient's outlook, attitude and self-concept.

Interestingly, when we saw I.V. for the third time she looked strikingly well and was pleased with her new situation.

The strongest suggstion for psychogeneity in I.V.'s case is probably the pattern of recovery. Neurogenic stuttering is often reported to persist for a long time and often seems to have a poor response to treatment (see for instance Andrews et al., 1972; Helm et al., 1978; Fleet & Heilman, 1985; Ardilla & Lopez, 1986; Lebrun et al., 1990; Stewart & Grantham, 1993). By contrast, reversibility of symptoms is considered one of the main characteristics of psychogenic stuttering. For instance, in 70 % of the 69 cases of psychogenic stuttering studied by Baumgartner and Duffy (1997) one or two treatment sessions were sufficient to obtain a dramatic improvement to normal or near-normal. I.V.'s speech improved quickly after the second evaluation. Her mother related the improvement to the discussion we had at that time with I.V. on her stuttering. What we did was to state that her speech would become better and encourage her to use light consonant contacts as substitutes for blocks. Optimism on part of the therapist and a symptomatic approach have been found useful by other investigators too (see Tippet & Siebens, 1991) in cases of psychogenic stuttering. Whether it was really the discussion we had with I.VK. that was responsible for the improvement is, however, hard to say. It was also at the same time that I.V. was finally discharged from hospital and that she started special education. Perhaps her improvement was also related, then, to this change of environment that had been pending for several weeks.

4. Conclusions

As the above case illustrates, differentiating psychogenic stuttering from other types of late-onset stuttering is not always straightforward. Although the hypothesis that I.V.'s stuttering was of neurogenic origin was plausible, there were still reasons to believe that her dysfluency was not an instance of neurogenic stuttering. From cases like that of IV it becomes clear that several hypotheses should be considered in the diagnosis of a dysfluency that developed beyond the typical childhood period. Psychogenic stuttering is certainly one of them. As psychogenic origin should even be considered in cases with demonstrable neuropathology. As Baumgartner (1999) stated it, "The presence of neuropathology, alone, should not exclude psychogenicity form differential diagnostic considerations" (p. 286).

Samenvatting

Het is niet altijd eenvoudig om de verschillende vormen van verworven stotteren van elkaar te onderscheiden. Vooral de differentiële diagnose tussen psychogeen en neurogeen stotteren kan wel eens moeilijk zijn, zoals het geval van I.V. illustreert. I.V. was 15 jaar oud toen ze begon te stotteren na een hersentrauma. Hoewel de hypothese

dat het om neurogeen stotteren zou gaan zeer plausibel leek, waren er toch argumenten om te besluiten dat haar stotteren niet een voorbeeld was van neurogeen maar wel van psychogeen stotteren.

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