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Contemporary clinical conversations about stuttering: What does brain imaging research mean to clinicians?

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Abstract
Purpose: To discuss among neuroscientists and community speech-language pathologists what brain imaging research means to clinicians.
Method: Two university neuroscientists and two speech-language pathologists in private practice discussed the matter. Written conversational turns in an exchange were limited to 100 words each. When that written dialogue was concluded, each participant provided 200 words of final reflection about the matter.
Result: For now, neuroscience treatments are not available for clinicians to use. But sometime in the future, a critical mass of neuroscientists will likely produce such treatments. The neuroscientists expressed diverse views about the methods that might be used for that to occur.
Conclusion: Neuroscience does have practical clinical application at present and, in a way, that does not exclude a concurrent influence of the social model of disability. As such, the current practices of the clinicians are supported by basic neuroscience research.

Keywords: stuttering; brain imaging; community clinicians

Prologue
During the past decade brain structure and function have become among the most researched aspects of stuttering, with an edition of the Journal of Fluency Disorders specifically assigned to the topic in 2018 (Neumann & Foundas, 2018). To date, more than 100 reports about this matter have been published in peer-reviewed journals, with regular neuroimaging additions each year. Meta-analyses and reviews of this literature have established that children and adults who stutter differ from their peers in both brain structure and function (Belyk et al., 2014; Brown et al., 2005; Budde et al., 2014; Chang, 2014; Chang et al., 2018; Etchell et al., 2018; Neef et al., 2015; Zhang et al., 2022). As is the case for most central nervous system disorders, it is increasingly being reported that stuttering is associated with circuit-level disruptions along major brain networks that support speech motor control. Deficits in both structural connectivity (white and grey matter volume) and functional connectivity (brain activity occurring in grey matter areas) have been reported. Broadly speaking, white matter is involved in transmission of information and grey matter is involved with information processing. Recurring research findings have implicated two prominent white matter structures in atypical neural speech processing: the corpus callosum and the arcuate fasciculus. The corpus callosum is white matter connecting the two brain hemispheres, and the arcuate fasciculus is white matter connecting parts of the brain associated with speech planning, production, and auditory processing. Grey matter structure as well as functional differences have been...
reported in structures along the basal ganglia-thalamo
cortical loop, which supports crucial functions
such as initiation, timing, and sequencing of speech
sounds.

This body of basic research raises the question of
what it might mean to professional speech-language
pathologists who provide healthcare to those who
stutter. Consequently, the present contemporary clin-
cial conversation about stuttering deals with the
application of those research findings to clinical prac-
tices. Participating in this conversation are two uni-
versity researchers who have conducted brain imaging
research: Soo-Eun Chang is Associate Professor
of Psychiatry and Director of the Speech Neurophysiology Lab located at the University of
Michigan, and Eric S. Jackson is Associate Professor
of Communicative Sciences and Disorders and
Director of the Stuttering and Variability (savvy) Lab
located at New York University. The two professional
speech-language pathologists involved in the conver-
sation are Gissella Santayana, a private practitioner in
Montreal, Canada, and Gillian Zavos, a private prac-
titioner in Sydney, Australia; both work with stutter-
ing clients of all ages. The conversation is moderated
by Mark Onslow, Director of the Australian
Stuttering Research Centre, Sydney, Australia.

Participants were limited to 100 words during
each of their conversational exchanges. When the ini-
tial dialogue was concluded, each participant pro-
vided 200 words of final reflections about the matter
in an epilogue. These final reflections were not seen
by the other participants until they were all
completed.

Dialogue

Mark: Soo-Eun, you publish brain imaging research. What is the reason you do that?

Soo-Eun: I've been curious about how the brain works since I was a child. When I was six, I moved to the United States from Seoul, speaking only my native language. Within a year, I was speaking freely in English without any trouble. But my parents never achieved this proficiency. I wondered, what is so special about the child's brain that makes it possible to acquire and produce speech and language so effortlessly? And what’s going on in those cases—a disorder or just older age—where this process is not as easy?

Mark: Eric, you also research brain function for those who stutter. For what reason do you do it?

Eric: Being a stutterer has taught me that most of stuttering occurs beneath the surface. This includes thoughts and emotions related to the experience of living with stuttering, as well as neurobiological processes more closely associated with stuttering events. Given their covert nature, these processes are often difficult to observe, but for the stutterer, learning to cope with them fundamentally shapes one’s experience of stuttering. I use functional neuroimaging to make these difficult-to-observe features more observable, to better understand the events that lead to overt stuttering behaviours, which I believe will benefit stutterers both in the long- and short-term.

Mark: Eric has raised clinical application of brain imaging research. Gissella and Gillian, you are professional speech-language pathologists. Have you applied this research to your clinical practices?

Gillian: I find neuroimaging research helps clients understand that stuttering is physical. I explain that fibre tracts connecting areas involved in spoken language seem to be less efficient. I discuss how this is consistent with the age when stuttering begins, its variable nature, and how it could explain spontaneous recovery. I explain that with a caveat that the neural basis for stuttering is not fully understood. So far, I don’t think neuroimaging research has had much bearing on treatment. Some of my clients seem to approach therapy practice tasks more readily when they are linked to the idea of neuroplastic change.

Mark: Do you apply brain imaging research to clinical practice?

Gissella: Brain imaging research enhances my response to the very first questions parents and people who stutter ask clinicians: “What is stuttering? Why is my child stuttering? What treatment options are available?” Providing this information allows better informed choices. For example, I believe that current research supports a recommendation to start treatment in the preschool-age years, when neuroplasticity is greatest. Additionally, it supports consistent and frequent treatment in out-of-clinic situations, emphasising the crucial role parents play in the therapy of young children. A better understanding of causality can contribute to the reduction of the stigma that may be associated with stuttering.

Mark: So, Eric and Soo-Eun, we have two clinicians who apply this body of literature to their clinical practices in ways that help their clients. From your perspectives as university researchers, is this what usually occurs?

Eric: In my experience, yes. Clinicians and student clinicians use neuroimaging research, along with genetics research, to help explain how stuttering is a neurobiological condition and to reinforce that it’s not the stutterer’s fault that they stutter. I have seen in my own practice and through supervision that this can help to reduce feelings of guilt and shame and reduce the stigma associated with stuttering. However, I agree with Gillian that neuroimaging research to date hasn’t had much bearing on treatment. I think that it can but that our research questions need to get better.

Soo-Eun: I am impressed to hear from both clinicians that neuroimaging findings are applied to their clinical work! More commonly, I feel that there is a substantial gap between science and clinical practice in our field. It is difficult for full-time clinicians to keep abreast with current research, let alone neuroimaging research, because most speech-language
pathologists are not used to consuming this type of literature.

Mark: Gissella and Gillian, as full-time generalist clinicians, how do you keep abreast of current brain imaging research so that your practices are up to date with emerging research?

Gissella: Currently most of my caseload is in stuttering; this has clarified my focus when reading research. I remember having less time as a generalist clinician, reading various topics concurrently. I listen to podcasts, webinars, interviews, and conferences, where I hear firsthand from the authors. This makes the information more accessible. I subscribe to alerts that notify you of new publications authored by my selected researchers. When reading an article, I save papers of interest, peruse only the abstract, and, when possible, read the papers later. Finally, discussing new findings with colleagues helps me integrate this new information.

Gillian: I work part-time as a clinician, exclusively with clients who stutter. I follow neuroimaging research because I did a PhD in this area 20 years ago. Owing to my background, it’s not too daunting to read those papers, but I’m not doing so for therapeutic insights. Clinicians spend most of their time with clients; they also have administration, which leaves limited time to read research. Neuroimaging papers tend to be written for fellow researchers. Clinicians might prefer a brief review paper or podcast discussion of clinically relevant findings.

Eric: I agree, neuroimaging papers aren’t written for clinicians, but I don’t know how much they’d help if they were! Very few clients I’ve worked with have been interested in the details of this past work, “there are differences in brain structure and function, particularly in speech areas.” Knowing the latter is in my view enough to help reduce shame and guilt. I agree with Soo-Eun that there is a large science-practice gap because we are only beginning to understand the brain, much less the stuttering brain.

Mark: Soo-Eun, do you agree with Eric’s statement that all that clients need to know from this body of research is that “there are differences in brain structure and function, particularly in speech areas?” I hope you do because that would make generalist clinical life a lot simpler …

Soo-Eun: Such bite-sized review summaries can certainly help alleviate stigma associated with stuttering. That said, what Gillian mentioned before struck me, that some clients seem more motivated to engage in therapy if it is linked to the concept of neuroplasticity. Clients may benefit from understanding that having differences in brain structure and function does not necessarily mean that these differences are set in stone. Our brains have a remarkable capacity to mould and adapt in response to environmental stimuli, and this can be leveraged during therapy. This is particularly true for children, but it is also possible in adults.

Mark: Indeed, there is evidence (Nock et al., 2007) to support the commonsense notion that compliance will be enhanced if a treatment makes basic theoretical sense to clients. So, with neuroplasticity in mind, let me ask our researchers how neuroscience might develop treatments in the future. Or perhaps neuroscience can be utilised more than is occurring at present with stuttering treatment?

Soo-Eun: A challenge is that compared to neighbouring fields, only a tiny number of researchers are investigating the neurobiological bases of stuttering. Neuroscience-based treatments that target alleviation of core symptoms must be preceded by years of basic science to understand causal factors, physiology, and mechanisms underlying differences we observe in the brain and behaviour. Then comes translational studies and clinical trials. We are at the start of this long process. In dyslexia, for instance, basic science has led to treatments that follow the principles of neuroplasticity, promoting meaningful gains in reading and associated strengthening of neural connectivity (Donnelly et al., 2019; Huber et al., 2018).

Eric: Soo-Eun’s right, we need to better understand the neurobiological bases of stuttering before neuroscience can have an impact on stuttering treatment. For example, neuromodulation might have a significant impact, but a better understanding of the stuttering brain is first required. I think we can achieve this understanding faster if we focus our questions, for example, on how the brain processes actual stuttering. Stuttering is intermittent by nature, and learning to cope with this intermittency is in my view central to the experience of stuttering. Yet, most functional work focuses on the brain in the absence of stuttering, during fluent speech.

Soo-Eun: Previous studies have indeed mostly examined brain function during perceptually fluent speech in stutterers. One reason is that in the moment of stuttering, concomitant activity associated with hyperactive motor and emotional responses can occur, which vary widely across individuals. So, in my view, initial studies would need to home in on core brain differences present across stutterers even when they are fluent, which could then tell you something about the underlying trait of the condition. Examining why stuttering is intermittent is no doubt a clinically and theoretically important question. A more fundamental question, however, is why and how does stuttering occur at all?

Eric: Twenty-five years and many functional studies reveal some trait differences, but also inconsistencies. Soo-Eun raises an important methodological concern about concomitant activity, but we’re learning to address these issues. A more serious confound with functional trait research is simply studying fluent speech when the goal is to learn something about stuttered speech. Stuttered and fluent speech are wildly different experiences for the speaker and, therefore, determining why stuttering events emerge
is, I think, the fundamental question. In my view, functional trait research has run its course. Genetics and structural imaging can inform why some people are stutterers but not why and how they stutter.

Soo-Eun: Studying fluent speech could provide critical clues to how the speech motor control function differs in stutterers. It might be subtle timing differences or less efficient integration of key brain regions within a network, for example, that are present even during non-stuttered speech. Distinct neural mechanisms observed during fluent speech in stutterers could be associated with why they are more prone to stutter. But Eric is right that current trait research cannot inform how an individual stutters. Future therapeutics will be increasingly individual-specific, and this will require a deeper understanding of how a specific person experiences their own stuttering.

Mark: So now, considering that our conversation is about what brain imaging research means to clinicians, we should hear from our clinicians about what all this means to them. Junior clinicians and students of speech-language pathology will be reading this to learn something. So, Gillian and Gissella, what would you like to say to them?

Gillian: As a clinician, I would like to understand how effective treatments for early stuttering (such as the Lidcombe Program) change the brain. I mentioned neuroplasticity earlier, but, in the clinic, I refer to it in general terms because it’s assumed, not studied. Also, clinicians need improved treatments for clients of all ages. Treatments for early stuttering fail some clients and are time onerous in the context of modern life. Speech-pathology treatment doesn’t offer adults who stutter the goal of authentic stutter-free speech. I think clinicians would like to see neuroimaging research refocus, to the extent that it’s critical clues to how the speech motor control function differs in stutterers. It might be subtle timing differences or less efficient integration of key brain regions within a network, for example, that are present even during non-stuttered speech. Distinct neural mechanisms observed during fluent speech in stutterers could be associated with why they are more prone to stutter. But Eric is right that current trait research cannot inform how an individual stutters. Future therapeutics will be increasingly individual-specific, and this will require a deeper understanding of how a specific person experiences their own stuttering.

Gissella: I believe researchers’ continued efforts to synthesise their findings, while making suggestions for possible clinical applications, will nourish our curiosity as clinicians, creating more avid interest in this process. This would stimulate a more dynamic dialog between researchers and clinicians regarding clinical-relevant questions: How does this therapy work? What causes variability? What happens in the brain following treatment? Are current therapies compatible with imaging research findings? For example, current research on the role of the basal ganglia gives a rationale for therapies based on syllable-timed-speech. As a clinician, I feel more confident recommending therapy that I can link to ongoing research.

Mark: Now let me press for immediate and practical guidance to junior clinicians and students about clinical application of brain imaging research. Our conversation has touched on neuroplasticity, stuttering onset, early intervention, and the clinical benefits of a simple neuroscience explanation of stuttering. So, how can all that be unified when a clinician explains to a parent why a preschool child has begun to stutter? Eric is well qualified to provide input here because he is a researcher, a practicing clinician, and has lived experience of stuttering. So, Eric, what would you say to such parents?

Eric: I don’t think neuroscience is so relevant, but a basic neurobiological explanation can be helpful: Stuttering is associated with brain structure and function differences, most stutterers are predisposed, and stuttering emerges after a period of extensive learning and neural development. I avoid buzz words such as neuroplasticity due to a limited understanding of how it works in stuttering. My discussion focuses on developing a strong alliance and trust, alleviating concerns, and presenting the science with objectivity. Parents should know that no therapy offers a cure, but therapy can help develop positive attitudes, agency, and easier ways of communicating to set the stage for success if the child’s stuttering persists.

Mark: So now, Gillian and Gissella, what guidance would you give to junior clinicians and students about how to explain to a parent why a preschool child has begun to stutter?

Soo-Eun: I would explain that stuttering is categorised under the umbrella of childhood-onset conditions that include dyslexia and ADHD; these emerge when certain aspects of neurodevelopment are disrupted due to a complex interplay between genetics and the environment. Research shows that subtle differences exist in how brain circuits supporting speech initiation and timing develop in stutterers during childhood. Speech and language regions are among the most “plastic,” or changeable, in the human brain, which means that they can change in response to training, stimulation, and therapy. Relatedly, research has shown that neural connections that were initially weaker develop in a more typical manner as children recover from stuttering.

Mark: So now, Gillian and Gissella, at the coalface of clinical practice, tell us verbatim how you would explain to a parent why a preschool child has begun to stutter.

Gillian: The cause of stuttering is not completely understood. We know stuttering is physical, not psychological. Brain imaging research suggests stuttering is due to a glitch in the brain’s processing for speech. Your child didn’t stutter when younger because your child hadn’t yet developed the language to make speech complex. Typically, stuttering begins around the time that children are putting a few words together. You might know of another family member who stutters. Often this is the case. Although there’s not one gene responsible for stuttering, it seems that a single gene or combination of genes can be involved.

Gissella: The cause is not crystal clear at this time. So far, researchers have observed minor differences in the way some areas of the brain communicate between each other and treat information. These areas are involved in ensuring the production of
continuous fluent speech. It is assumed that many genes are associated with stuttering and researchers have discovered a few, but at this moment we don’t know exactly how they cause stuttering. Some factors may increase stuttering, but they are not the underlying cause. Starting treatment in the preschool years is best. You have done the right thing seeking services!

Mark: In their explanations to parents of why their children have begun to stutter, our clinicians have raised something that I want to throw back to our researchers: the cause of stuttering. Is the evidence to date convincing enough to convey to clients that brain network disruptions are part of the cause of stuttering? Our clinicians seem not convinced. I can relate to their reticence, considering that the only independently-replicated observations of such disruptions are after stuttering onset. Therefore, can we be confident that they are part of the effects of stuttering, not part of its cause?

Soo-Eun: Neuroimaging data cannot definitively tell us about the cause or aetiology. It can, however, provide crucial information that can bridge between aetiology and symptoms of a disorder. In other words, it can give us insights on how the aetiology disrupts the normal function of the brain to produce stuttering. From a clinical perspective, I’m not sure if knowing the “cause”—which may later be explained on a cellular/molecular basis—is terribly useful. What both clinicians said about how the cause is unclear, but that there are brain network differences associated with having stuttering, seems perfectly valid to me.

Mark: Eric, what is your take on this?

Eric: I completely agree with Soo-Eun. Perhaps, we as scientists who study the brain and stuttering need to better communicate these ideas. It may help to ameliorate recently emerging tensions between the various stakeholder groups in stuttering, such as between neuroscience and the neurodiversity movement. I think more pressing questions about cause relate to discovering the processes that underlie stuttering (social-cognitive, linguistic), which will happen through theory development.

Soo-Eun: Eric brought up a hot topic in the field: the tension between medical, disorder-based and neurodiversity-based perspectives. The former focuses on understanding the biological bases of the dysfunction so that they may be targeted for treatment, leading to alleviation of symptoms and impairment. The latter focuses on the neurodiverse person’s physical and social environment that limit and constrain, leading to adversity and undermining of well-being. In my view, the two perspectives are complementary, sharing the goal of alleviating negative impacts experienced by stutterers. I am curious to hear from clinicians who treat stuttering what they have heard and felt about these perspectives.

Gissella: I agree with Soo-Eun, that both perspectives are valuable in treating stuttering. Clients often identify reduction of stuttering as a goal at the start of therapy. This leads to a more nuanced conversation and a plan that includes realistic and attainable goals. While the current literature focuses on acceptance and neurodiversity, acceptance is a lengthy process, and I believe both perspectives can be combined successfully. For many young children, evidence-based treatments reduce stuttering severity to extremely low levels. Parents request this stuttering reduction. In my experience, it’s possible to both achieve this reduction and see them become confident communicators.

Gillian: As a clinician, I’m coming at this from the perspective of helping people to communicate. I agree, Soo-Eun, that a neurodiverse perspective can exist alongside a goal of stuttering reduction. Recently, treatment helped a 12-year-old client become aware that not talking at school, for fear of people noticing the stuttering, was more detrimental than stuttered speech. Feeling free to talk at school benefitted the client. Choosing to speak despite stuttering is courageous in a culture where it’s stigmatised. I see the neurodiverse and disorder-based perspectives as complementary.

Eric: The tension is that searching for cause or what goes wrong in the brain could imply that there is something wrong with people who stutter. This is perpetuated when we use terms like “impairment” and when we talk about “reducing stuttering.” Critically, there’s no way to directly target fluency in older kids and adults. Even with fluency-focused treatments, fluency is only indirectly targeted. This may be different in young children for whom studies show reductions in stuttering frequency due to treatment. However, I’m very sceptical of whether these reductions are practically meaningful, not just statistically meaningful, and also whether they truly outpace natural recovery.

Soo-Eun: While some may not experience stuttering as an impairment, many others do, enduring substantial psychosocial consequences. Ultimately, having more options for treatment, such as towards achieving authentic stutter-free speech, have greater value than what terms are used. I agree that current treatments targeting “fluency” are unsatisfactory and can even be harmful. That doesn’t mean that novel, effective treatments can’t be developed in the future, guided by an advanced neurobiological understanding of stuttering. Concurrently, educating society to become better listeners and advocates, and helping stutterers to be more confident communicators regardless of how they speak, can significantly help improve their wellbeing.

Mark: So, it seems, as was the case with a previous discussion in this Clinical Conversations about Stuttering series (Shenker et al., 2023), a medical and social perspective of stuttering may coexist comfortably in clinical practice.

Epilogue

Soo-Eun: I have tremendous respect for clinicians working tirelessly to treat and support clients who
sutter. I wish the research was more advanced to offer more evidence-based treatment options for adults and children who express their wish to speak effortlessly, naturally, and fluently. Our understanding of neural mechanisms underlying stuttering is still evolving. To date, efforts to develop treatments designed to stimulate neuroplastic growth that supports effortless speech have been lacking. The pool of researchers investigating the neural bases of stuttering is extremely small. Neuroimaging research to date has generated at times equivocal findings, which has also been amply experienced in other fields and overcome by forming consortiums among researchers, clinicians, and stakeholder groups that allow larger-scale studies guided by theoretically and clinically meaningful aims. Lessons from these neighbouring fields suggest that there will be no easy path for developing interventions for complex disorders such as stuttering. I have hope, however, for encouraging developments and breakthroughs in the next several years. It is one of the scientist’s jobs to disseminate their research findings. I am reminded by this discussion that I should do so not just with other researchers in mind—the clinicians inspire me to do better.

**Gillian:** Clinicians can explain to clients that stuttering is associated with different processing in speech and language areas in the brain. The processing differences may be due to weaker connections between speech and language areas. Around the age stuttering emerges, such connections are expected to strengthen instead. This likely has a genetic basis. Discussing this lets clients know that the stuttering is nobody’s fault, that the brain of someone who is stuttering is neurodiverse, and also that change might be possible. Speech and language areas of the brain are highly plastic—able to change in response to activity—particularly in children. Neural plasticity can explain why treatment outcomes for early stuttering are superior to those for advanced stuttering. I hope future brain imaging research will study children prior to the development of stuttering, so that we understand how it presents at the outset. I would like to understand what successful early treatment does to brain structure and function because I think it would be insightful for clinicians and motivating for parents. Such knowledge could also guide researchers in the development of novel treatments for adults, perhaps using techniques to increase neuroplasticity, such that authentic stutter-free speech is possible.

**Eric:** It seems that, for the most part, we agree that brain imaging research on stuttering can be used to help explain stuttering to clients and parents, which may help reduce shame, guilt, or other related feelings. We also agree that the scope of the impact has been limited due to the science-practice gap. Regardless of whether neuroimaging articles are written in a more accessible manner or more clearly summarised for lay audiences, the science is not advanced enough to directly impact treatment at this time, such as with neuromodulation, pharmacology, or neuroscience-guided treatments (Chester et al., 2017; Garnett et al., 2019). Moreover, whether the brain can change via neuroplasticity as a result of treatment to the extent that it helps stutterers is an open question. For brain imaging to be most useful, we must develop research questions based on the stuttering experience, the hallmark of which is the intermittency with which stuttering events occur, whether these events are observable or not. This is neither a neurodiversity nor medical perspective. It’s hard to argue against the long-term goals of the medical perspective (e.g. authentic stutter-free speech), but I disagree with the path that many neuroscientists are taking to get there; for example, studying differences in fluent speech between stutterers and non-stutterers. I don’t think that this will get us any closer to a neurobiological understanding of stuttering.

**Gissella:** Currently, brain-imaging research provides clinicians with information that we can use to help the reduction of stigmatisation that is often associated with stuttering. Future neuroscience research has the potential to help us understand the components of successful therapy and, therefore, directly impact treatment goals. This clinically meaningful potential is the motivation for clinicians to familiarise themselves with the brain-imaging research. Although there are some differences of opinion between medical and neurodivergence perspectives, I believe that they can both inform us in the treatments that we offer to persons who stutter. My clinical experience has strengthened my belief in the usefulness of evidence-based approaches to the direct management of stuttering. I have seen that both reduction of stuttering and spontaneous communication are not mutually exclusive goals, especially with young children. When stuttering persists it is the clinician’s responsibility to consider the client’s objectives in choosing goals, which include exploration and acceptance of the experience of stuttering, as well as self-advocacy and education in the environment of the person who stutters.

**Mark:** The question that drove this conversation was “what does brain imaging research mean to clinicians?” and it seems clear that, for now at least, it does not mean that neuroscience treatments are available that clinicians can use. But sometime in the future, a critical mass of neuroscientists will likely produce such treatments. Our two neuroscientists seem to have diverse views about how that will occur, but they are keen to interact as they strive for that glittering prize. That is how it should be. Regardless, at present, neuroscience does have practical clinical application. The reality of neuroplastic change is motivational for clients and parents. Neuroscience conveys the physical nature of stuttering to them, and that they are to be free of blame for it. Also, a neuroscience explanation of stuttering can alleviate negative emotions about stuttering and associated self-stigma.
For junior clinicians and students who are seeking a contemporary bridge between neuroscience research and clinical practice, there it is for you. And there is no need to believe that a medically based neuroscience influence on clinical practices excludes a concurrent neurodiversity influence on clinical practices. Overall, it seems that the clinical practices of our clinicians are supported by basic research. And that also is how it should be.

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Declaration of interest

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