

BY EVAN GOLDBERG

Combating the Developmental Delay Defense for Children

When representing an infant alleged to have been brain damaged from fetal distress, lead poisoning, toxic inhalation or blunt head trauma, a developmental delay defense on causation is almost certain to be interposed. With cognitive and behavioral impairments of unknown causes experiencing a dramatic and epidemic increase in recent years, plaintiff's counsel representing a child at the time of trial is often placed in the unenviable position of disproving a laundry list of syndromes which are, by their very definitions, imprecise and overlapping, with great disparity in severity and prognosis.

For example, the Diagnostic and Statistical Manual of Mental Disorders IV (DSM IV) classifies autism as a subgroup of pervasive developmental disorder (PDD) and defines it as a "markedly abnormal or impaired development in social interaction and communication and a markedly restricted repertoire of activity and interests."

When a child has autistic characteristics but not to the extent that meet the DSM definition, the child may be diagnosed with asperger's syndrome or may be called a highly functioning autistic. While asperger's syndrome typically involves no delay in speech development, exceptions are common.

When normal development gives way to regression, a child may be diagnosed with child disintegrative disorder or Rett's syndrome, both of which bear substantial similarity to autism vis-a-vis social and communicative dysfunction.

Catch-All Diagnosis

When an infant's symptoms approach but do not meet the definitions set forth for the aforementioned syndromes, the DSM provides a catch-all "pervasive developmental disorder not otherwise specified (PDD NOS), which applies to atypical presentations. The mere listing and similarities of the aforementioned criteria demonstrate the difficulty inherent in diagnosing conditions that begin as developmental delays. Indeed, even if the disorders were limited in number and classification, specific cases would still be prone to considerable subjectivity.

Further, individualized symptoms associated with the aforementioned syndromes are syndromes in them-



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selves, such as sensory integration dysfunction (SID), attention deficit and hyperactivity disorder (ADHD), and a myriad of related learning, communicative, cognitive and behavioral disabilities.

Sadly, the state of scientific knowledge as to the diagnosis, treatment and prognosis of these disorders is, like millions of patients, in its infancy.

Alarming Increase

The number of afflicted children in the United States is staggering, having been called epidemic¹ and a national emergency.² California reported a 273 percent increase in school-age autism between 1987 and 1998;³ Rhode Island, a 1,115 percent increase between 1994 and 2002,⁴ and Ohio, a 6,822 percent increase between 1992 and 1999 for autism/PDD.⁵

Currently, a 10 percent to 17 percent increase per year on a national level has been reported,⁶ which does not even include milder forms of the disorder or those felt to be on the low end of the autistic spectrum. In terms of the population, recent reports suggest that as many as 1 in 150 of American children are autistic, compared with the 1 in 10,000 reported in 1990.⁷

The related disorders and syndromes discussed herein are also steadily on the rise. The reasons for the alarming increase, much like the existence of the disorders themselves, remain a mystery.

To compound the problem in the courts, children alleged to have been injured from negligence are most often treated the same way as those diagnosed with developmental delays of unknown causes. After assessment, children are provided with publicly funded speech, physical and occupational therapy, as well as special education. Regardless of etiology, the developmentally impaired learn side by side, and often with the same methodology.

While many hail the benefits of early therapeutic intervention, the medical community continues to be divided. Drug therapies, biofeedback, auditory therapy and special diets are also advanced, but are similarly controversial. To the governments and school boards charged with educating and providing appropriate services for the disabled pursuant to law,⁸ labels and diagnoses are needed to establish benefits, with causative determinations being unnecessary and most often irrelevant. However, when seeking compensation for the lifelong impairments occasioned by brain damage, practitioners are quite familiar with their obligations regarding proof in that regard.

The oft cited legal maxim *inclusio unius est exclusio alterius* (the inclusion of one is the exclusion of another)

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er) may be illustrative of both an approach toward substance and tactics. With regard to the latter, to prove that a matter is as proposed, it helps to prove that the matter is not as proposed by one's adversary. In the case of cognitive deficits, an attorney may attempt to prove that the plaintiff's presentation is atypical of the syndrome at issue, either because of temporal issues and/or the nature and extent of the symptoms and, thus, not diagnostic of the developmental delay being advanced by the defendant.

Or, the attorney may, in proving the existence of traumatic brain damage, disprove developmental delay in the process, using the same legal and medical principles employed for exclusion, which include, but are not limited to neuroimaging and related diagnostic studies.

However, when facing a developmental delay defense, counsel must remember that "[t]here may be more than one proximate cause of a plaintiff's injuries and a plaintiff need not exclude every other possible cause of injury to recover," the court wrote in *Spano v. Onondaga County*.⁹

The accepted standard is whether facts and circumstances are shown from which causation may be reasonably inferred, as ruled in *Derdarian v. Felix Contracting Corp.*¹⁰

Therefore, while a jury may consider alternative theories of causation advanced by a defendant, it may still base a verdict upon plaintiff's proof even when developmental delays have not been conclusively excluded.

'Ledogar'

In *Ledogar III v. Giordano*,¹¹ the Appellate Division, Second Department, reversed a trial court's finding that a liability verdict was against the weight of the evidence, and reinstated the verdict that was based on a finding of prenatal hypoxia, alleged to have caused autism. In analyzing the proof, which included the testimony of a neurologist who stated that the hypoxia would have been a substantial contributing factor in the etiology of the infant's autistic condition, the court noted:

Nor does the fact that this expert acknowledged that the plaintiff's autism could have had causes other than prenatal hypoxia warrant a finding, as a matter of law, that causation was not proved. A plaintiff is not obligated to eliminate all possibility that the injuries resulted from causes other than defendant's negligence.

When encountering a developmental delay defense, it is important to deal with terminology, and the diagnostic criteria contained in the DSM. Often, in order to receive requisite services, the infant plaintiff has already been diagnosed with a developmental disorder or, at the very least, with symptoms that are felt to be synonymous with a particular disorder.

Since educators and health providers are sometimes not privy to

a history of negligence and since it is easy to group students together when they are in the same class, have the same symptoms and are being given the same treatment, a diagnosis that fails to consider pertinent history is inherently suspect.

Similarly, a provider's diagnosis may be subject to scrutiny if the primary goal was to establish eligibility for services, with the consequential use of a diagnosis as a means to an end.

Three Options

Regardless, infant plaintiffs often find themselves in court with a developmental delay diagnosis throughout their academic and medical records. A lawyer who faces this situation may either: (i) disagree with the nature of the symptoms and diagnosis; (ii) agree with the symptoms but not the diagnosis; (iii) or agree with both the symptoms and diagnosis.

In choosing the first option, the tactics employed at trial will necessarily involve conflictive opinions from experts on every aspect of the claimed injury. When choosing the second option, the lawyers and experts will be battling over definitions and the overlapping of symptomatology as pertains to the various developmental syndromes.

With respect to the third option, this writer is unaware of any medical authority documenting an immunity from brain damage for children with developmental delays. Indeed, autism and related disorders do not make a child impervious to the toxic effects of lead, or to the dangers of oxygen deprivation to the brain or to the effects of blunt head trauma.

Conceding the diagnosis and/or label in the records can deflate the defense, but can also substantially limit damages. However, while it is often difficult to separate which cognitive and behavioral impairments are attributable to developmental delays and which are the result of the negligence, juries grapple with such issues all the time, since "a preexisting condition does not foreclose a finding that a plaintiff's injuries were causally related to the accident," the court said in *Matthews v. Cupie Transportation Corp.*¹²

If a child is experiencing cognitive and behavioral difficulties consistent with a reliable developmental diagnosis, and then sustains a brain injury that causes further problems in those areas, there is no reason a jury may not determine aspects of compensability.

In the lead poisoning case *Walton v. Albany Community Development Agency*,¹³ various theories of causation for the infant plaintiff's cognitive disabilities were advanced by defendant's experts. On defendant's motion for summary judgment, an expert affidavit was submitted in which the expert opined "with a reasonable degree of medical certainty, it is evident that the toxic effects of lead upon [plaintiff] are inconsequential in comparison with etiologies of her developmental impairments" and that "the developmental delays ... are more likely than not caused by her Fetal Alcohol Syndrome and not by lead."

In finding that this proof did not serve to establish movant's prima facie showing sufficient to shift the burden on the motion to plaintiff, the court said:

[T]he Court of Appeals has held that prima facie causation is established by evidence that the child's residence contained a hazardous level of lead-based paint and that the child was observed ingesting paint fragments, had elevated lead levels, was not out of the parent's custody for any substantial period of time and did not manifest the pertinent symptoms before moving into the residence (*see Juarez v. Wavecrest Mgt. Team*, 88 N.Y.2d 628) ... Nor does the opinion that exposure to lead was an inconsequential factor, or that another cause was more likely than not, preclude such exposure from being found, as a matter of fact, to be one of the proximate causes of the children's neuropsychological conditions.

Conclusion

Though hardly a new concept, practitioners must be up to the task of educating judges and juries that the old principles regarding proximate causation remain applicable and have not been overturned in cases of children with purported developmental delays and/or disorders.

As the nation experiences an unprecedented and emergent increase in the diagnosis of school-age children with developmental disabilities, it will become harder to successfully exclude such syndromes as a causative component when brain damage has been alleged.

When a convincing case of causation is presented, the attorney's best approach is to educate the jury and invoke common sense. When the proof is less clear, one should avoid spending the whole trial fighting over definitions doctors have yet to agree upon themselves.

Rather, the best course is to focus on symptoms. While no physician will say hypoxia or lead poisoning causes pervasive developmental disorders, since the cause(s) are still not known, there is no scientific impediment to testifying that those events can potentially cause and/or exacerbate the symptoms that are diagnostic of PDD.

(1) Hearing before 107th Congress' Committee on Government Reform, April 26, 2001.

(2) Hearing before Congress' Committee on Government Reform, April 18, 2002.

(3) Autism Prevalence, Center for Disease Control, July 2, 2002.

(4) Autism Autoimmunity Project, Rhode Island, 2002.

(5) 21st Report of the U.S. Dept. of Education to Congress (IDEA).

(6) Hearing before Congress' Committee on Government Reform, April 18, 2002.

(7) Center for Disease Control Report, April 2000.

(8) Individuals with Disabilities Education Act of 1997.

(9) 135 A.D.2d 1091, 523 N.Y.S.2d 310 (4th Dept. 1987).

(10) 51 N.Y.2d 308, 434 N.Y.S.2d 166.

(11) 122 A.D.2d 834, 505 N.Y.S.2d 899 (2d Dept. 1986).

(12) 302 A.D.2d 566, 758 N.Y.S.2d 66 (2d Dept. 2003).

(13) 279 A.D.2d 93, 718 N.Y.S.2d 456 (3rd Dept. 2001).