17. Gay JC, Hain PD, Grantham JA, Saville BR. Epidemiology of 15-day readmissions to a children's hospital. Pediatrics 2011;127:e1505-12.
18. Brittan M, Martin SE, Shmerling J. Hospital readmission rates. Health Aff 2014;33:2266.
19. Auger KA, Simon TD, Cooperberg D, Gay J, Kuo DZ, Saysana M, et al. Summary of STARNet: Seamless Transitions and (Re)admissions Network. Pediatrics 2015;135:164-75.
20. Berry JG, Toomey SL, Zaslavsky AM, Jha AK, Nakamura MM, Klein DJ, et al. Pediatric readmission prevalence and variability across hospitals. JAMA 2013;309:372-80.
21. The American College of Obstetricians and Gynecologists. Frequently asked questions labor, delivery, and postpartum care. http://www.acog.org/Patients/FAQs/Cesarean-Birth-C-Section. Accessed April 30, 2013.
22. HCUP: Healthcare Cost and Utilization Project. Clinical Classifications Software (CCS) for ICD-9-CM. http://www.hcup-us.ahrq.gov/toolssoftware/ccs/ccs.jsp. Accessed April 14, 2013.
23. Horwitz LI, Grady JN, Cohen DB, Lin Z, Volpe M, Ngo CK, et al. Development and validation of an algorithm to identify planned readmissions from claims data. J Hosp Med 2015;10:670-7.
24. Basu SK, Fernandez ID, Fisher SG, Asselin BL, Lyman GH. Length of stay and mortality associated with febrile neutropenia among children with cancer. J Clin Oncol 2005;23:7958-66.
25. Klaassen RJ, Goodman TR, Pham B, Doyle JJ. "Low-risk" prediction rule for pediatric oncology patients presenting with fever and neutropenia. J Clin Oncol 2000;18:1012-9.
26. Bodey GP, Buckley M, Sathe YS, Freireich EJ. Quantitative relationships between circulating leukocytes and infection in patients with acute leukemia. Ann Intern Med 1966;64:328-40.
27. Mullen CA. Which children with fever and neutropenia can be safely treated as outpatients? Br J Haematol 2001;112:832-7.
28. Goldfield NI, McCullough EC, Hughes JS, Tang AM, Eastman B, Rawlins LK, et al. Identifying potentially preventable readmissions. Health Care Financ Rev 2008;30:75-91.

50 Years Ago in *THE JOURNAL OF PEDIATRICS*

Myotonic Dystrophy: A Neglected Form of Mental Retardation

Calderon R. J Pediatr 1966;68:423-31

Calderon described 6 cases of congenital myotonic dystrophy and compiled 55 infants reported, 53 of whom had associated global developmental delay. The author believed that patients with congenital myotonic dystrophy were unrecognized. In the end, his conclusion remains equally valid today—that when evaluating a child with developmental delay, myotonic dystrophy type 1 (DM1) should be considered among the differential diagnoses, particularly when features associated with neuromuscular disease were present.

As well-characterized by Calderon, diagnosis for DM1 is considered when a newborn or young infant is presented with facial and generalized weakness, global hypotonia, feeding difficulties, and respiratory failure commonly associated with myotonic dystrophy. As Calderon illustrated, family history and clinical exam of the patient and parents could lead to the diagnosis. As opposed to 50 years ago, diagnosis of DM1 is now confirmed by molecular genetic testing of the CTG repeat expansion in the 3′-untranslated region of the *DMPK* gene. Calderon was well aware that myotonic dystrophy was inherited in an autosomal dominant pattern; however; we now understand that disease severity and age of onset are not determined by the isoallele he suspected was provided by the other parent, but rather by the number of CTG repeats in the *DMPK* gene.

Use of ancillary testing such as electromyography and muscle biopsy can still be useful in questionable cases, but electrical myotonia is not present in affected infants and muscle biopsies can be indistinguishable from those of other congenital myopathies. Brain magnetic resonance imaging has now replaced the skull radiographs, providing better demonstration of cortical atrophy, ventriculomegaly, and white matter abnormalities.

The manuscript also posed the question of whether myotonic dystrophy, myotonia congenita, and paramyotonia were allelic disorders. We now know they are separate entities secondary to well-defined gene defects.

Survival of even the most severely affected infants has improved notably. Management remains supportive. Treatment of myotonia has been replaced by mexiletine, which can be moderately helpful.

Even though DM1 is more easily recognized today, greater awareness is still needed. The first clinical trials of antisense knockdown of the genetic defect are emerging. One can only imagine what the next 50 years will bring for patients with myotonic dystrophy.

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