39. Groenink M, den Hartog AW, Franken R, Radonic T, de Waard V, Timmermans J, et al. Losartan reduces aortic dilatation rate in adults with Marfan syndrome: a randomized controlled trial. Eur Heart J 2013;34:3491-500.
40. Chiu H-H, Wu M-H, Wang J-K, Lu C-W, Chiu S-N, Chen C-A, et al. Losartan added to β-blockade therapy for aortic root dilation in Marfan syndrome: a randomized, open-label pilot study. Mayo Clin Proc 2013;88:271-6.
41. Milleron O, Arnoult F, Ropers J, Aegerter P, Detaint D, Delorme G, et al. Marfan Sartan: a randomized, double-blind, placebo-controlled trial. Eur Heart J 2015;36:2160-6.
42. Muiño-Mosquera L, De Nobele S, Devos D, Campens L, De Paepe A, De Backer J. Efficacy of losartan as add-on therapy to prevent aortic growth and ventricular dysfunction in patients with Marfan syndrome: a randomized, double-blind clinical trial. Acta Cardiol 2017;72:616-24.
43. Sandor GGS, Alghamdi MH, Raffin LA, Potts MT, Williams LD, Potts JE, et al. A randomized, double blind pilot study to assess the effects of losartan vs. atenolol on the biophysical properties of the aorta in patients with Marfan and Loeys-Dietz syndromes. Int J Cardiol 2015;179:470-5.
44. Mason DA, Moore JD, Green SA, Liggett SB. A gain-of-function polymorphism in a G-protein coupling domain of the human beta1-adrenergic receptor. J Biol Chem 1999;274:12670-4.
45. Metra M, Covolo L, Pezzali N, Zacà V, Bugatti S, Lombardi C, et al. Role of beta-adrenergic receptor gene polymorphisms in the long-term effects of beta-blockade with carvedilol in patients with chronic heart failure. Cardiovasc Drugs Ther 2010;24:49-60.
46. Filigheddu F, Argiolas G, Degortes S, Zaninello R, Frau F, Pitzoi S, et al. Haplotypes of the adrenergic system predict the blood pressure response to beta-blockers in women with essential hypertension. Pharmacogenomics 2010;11:319-25.
47. Baudhuin LM, Miller WL, Train L, Bryant S, Hartman KA, Phelps M, et al. Relation of ADRB1, CYP2D6, and UGT1A1 polymorphisms with dose of, and response to, carvedilol or metoprolol therapy in patients with chronic heart failure. Am J Cardiol 2010;106:402-8.
48. White HL, de Boer RA, Maqbool A, Greenwood D, van Veldhuisen DJ, Cuthbert R, et al. An evaluation of the beta-1 adrenergic receptor Arg389Gly polymorphism in individuals with heart failure: a MERIT-HF sub-study. Eur J Heart Fail 2003;5:463-8.
49. de Groote P, Helbecque N, Lamblin N, Hermant X, Mc Fadden E, Foucher-Hossein C, et al. Association between beta-1 and beta-2 adrenergic receptor gene polymorphisms and the response to beta-blockade in patients with stable congestive heart failure. Pharmacogenet Genomics 2005;15:137-42.

50 Years Ago in *The Journal of Pediatrics*

A Comprehensive Assessment of Gestational Age in the Newborn is Born

Dubowitz LM, Dubowitz V, Goldberg C. Clinical assessment of gestational age in the newborn infant. J Pediatr 1970;77:1-10.

In 1970, L. M. Dubowitz, V. Dubowitz, and C. Goldberg studied neurologic and external characteristics previously described in the clinical assessment of gestational age. They found a wide overlap in the gestational age at which an individual neurologic sign might be present or absent, resulting in difficulty predicting gestation objectively. A combination of neurologic signs and external characteristics identified in newborn infants for the clinical assessment of gestational age was described. Neurologic assessments were selected based on being easily definable and reproducible by multiple observers. These assessments were also the ones least influenced by the state of the newborn. A scoring system for all criteria, including both neurologic and external characteristics, was then developed. This Dubowitz scoring system resulted in a more objective and reliable method of assessing gestational age than that based on the presence or absence of individual criteria as described previously.

This high-impact study paved the way for a comprehensive and cohesive examination of newborns throughout pediatric medicine. Over the past 50 years, there have been further developments in newborn gestational age assessment tools, most notably the Ballard Maturational Assessment (BMA) described by Ballard et al in 1979.[1] In addition, there has been vast improvement in ultrasound dating of the fetus in developed countries. The BMA is a simplified version of the Dubowitz scoring system for clinical determination of fetal maturation of newborn infants in the range of 26-44 weeks.[1] This was expanded in 1991 to the New Ballard Score (NBS) to include extremely preterm infants born at <26 weeks gestational age.[2] The BMA is most reliable between 30 and 42 hours of life, whereas the NBS is most optimal at <12 hours of life.[1,2] At this time, the most accurate gestational age estimation is achieved by prenatal dating using the last menstrual period and early prenatal ultrasound, as well as postnatal physical examination and neurologic assessment.

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References

1. Ballard JL, Novak KK, Driver M. A simplified score for assessment of fetal maturation of newly born infants. J Pediatr 1979;95:769-74.
2. Ballard JL, Khoury JC, Wedig K, Wang L, Eilers-Walsman BL, Lipp R. New Ballard Score, expanded to include extremely premature infants. J Pediatr 1991;119:417-23.