

Dr. Money delivered a very thorough and appropriately lengthy personal and professional history of Dr. Wilkins. Dr. Money's closing paragraph is particularly pertinent as it is conceptually flattering and truthfully accurate: "Lawson Wilkins achieved fame, but as a by-product of accomplishment. His life's goal had been to achieve, not to become famous."

8. Fisher DA. A Short History of Pediatric Endocrinology in North America. *J Pediatr* 2003 (In preparation).

The purpose of this article is to record for posterity a historical perspective of the founding and development of pediatric endocrinology as a subspecialty, of the Lawson Wilkins Pediatric Endocrine Society, of pediatric

endocrine training programs, of pediatric diabetes as a discipline, and of advances in understanding, diagnosing, and treating pediatric endocrinopathies since 1950. A very excellent and complete presentation of the topic has been written by Dr. Fisher. As part of this, Lawson Wilkins' major roles as pediatrician, founder of the subspecialty, clinical investigator, and academician are evident.

9. Migeon CJ. *The Origins and Establishment of the LWPEs*. <http://www.lpwes.org/history.html>. The concept and history created by Lawson Wilkins invitation in 1963 of a scientific gathering to the formal creation in 1972 of a Society is interestingly detailed.

Abstracts from the Literature

Body Mass Index and Segmental Proportion in Children with Different Subtypes of Psychosocial Short Stature

Psychosocial short stature (PSS) has been classified by the authors into 3 categories: (1) Type IIA are hyperphagic children, in whom there is reversible growth hormone (GH) insufficiency with rapid catch-up growth with a change in their living environment but with minimal response to exogenous GH; (2) Type IIB is a heterogeneous sub group of non-hyperphagic children who have normal GH secretory dynamics and minimal or absent increase in growth rate with change in their environment and variable response to GH; and (3) Type III are children with anorexic eating habits, with an onset as early as infancy, with failure to thrive, depression, normal GH secretory dynamics, and significant growth response to exogenous growth hormone. Gohlke et al

report anthropometric evaluations of 46 children with PSS, before and after change in their environment (Table 1).

Significant improvement in height velocity SDS after intervention was observed in all groups. ANOVA failed to show any significant differences in growth velocity between groups. There was no significant change with treatment in body proportion in type IIA (hyperphagic) or in type IIB (heterogenous) children. In type III (anorexic) children, the body proportions decreased significantly after intervention indicating relatively shorter upper segments after treatment. In those who received GH treatment (n = 21), there was no significant change in body proportion after GH therapy. Body Mass Index

Table 1

Clinical Data of 46 Children with PSS

Classification	Type IIA (n = 20)	Type IIB (n = 16)	Type III (n = 10)
Mean age at presentation (years)	8.6	7.6	10.2
Age range (years)	4.9-15	3.8-14.9	5.4-14.9
Sex	9F. 11M	4F. 12M	6F. 4M
IUGR	2F. 2M	0F. 5M	2F. 2M
Mean bone age delay at presentation (years) (SD)	1.69 (1.0)	1.69 (1.3)	2.0 (1.5)
Prepubertal at presentation	18	15	9
Type of intervention			
Social services only	17	5	3
Social services and GH therapy	3	11	7

Adapted from: Gohlke BC, et al. *Eur J Pediatr* (220) 161: 250-254.

(BMI) did not increase in any of the groups after intervention and there were no significant changes in bone age. Multiple regression analysis showed that the type of PSS was a predictor for height velocity after intervention. The greatest effect in removal from adverse home events were in the type IIA (hyperphagic) subjects. The authors state that their findings should be helpful to clinicians managing children with PSS because of the emphasis on appetite disturbance and the variable treatment responses.

Gohlke BC, et al. *Eur J Pediatr* 2002;161:250-254.

First Editor's Comment: PSS, first described in 1947 by Talbot et al,¹ is often difficult to diagnose. Variable GH secretory dynamics, and responses to exogenous GH therapy make it important to attempt to better understand the etiologies involved and their potential response to psychosocial changes. The current manuscript report data on a large number of subjects with PSS and suggested that BMI is not useful in predicting response to treatment, but that categorization based on appetite may be of use in predicting growth changes. It is unfortunate that their data were not analyzed separately for those with intrauterine growth retardation (IUGR) and for those with and without GH insufficiency. However, the heterogeneous composition and variable treatment of these children strengthen the conclusions based on categorization of subjects by their eating behavior.

Reference

1. Talbot NB, et al. *N Engl J Med* 1947;236:783-789.

William L. Clarke, MD

Second Editor's Comment: PSS should be considered by pediatric endocrinologists or pediatricians in the differential diagnosis of short stature when a short child is seen in the clinic. If the possibility of this diagnosis is not considered and explored in the history, the diagnosis will be missed. PSS occurs much more frequently than is realized. Many parents of children with PSS (particularly Type II A of the English classification) are not concerned about their child's stature because the parents are psychologically rejecting the child.

PSS is a spectrum of entities as Gohlke, Frazer, and Stanhope state. The classification is muddy for this reason. In the patients reported by Gohlke et al there was no child less than 3.8 years of age. In the classification listed in Lifshitz's *Pediatric Endocrine Text*¹ (3rd Edition, 1996), infants with PSS comprise a broad clinical spectrum. This should be kept in mind so that the diagnosis of PSS is made and treatment properly

instituted, which in my opinion is not GH, but removal of the child from the adverse environment, particularly for type II.

Types of PSS as Described in the 3rd Edition of *Pediatric Endocrinology*¹

At least three subtypes of psychosocial short stature have been recognized (Table 2). The first (type I) occurs in infants and children 2 years of age or younger. These infants usually have failure to thrive (nutritional deficiency), as well as short stature, and have been very adequately described by Krieger, Whitten, and colleagues.²⁻⁶ There is no evidence that these children have a hormonal disturbance, such as growth hormone deficiency, and they usually recover when sufficient calories are ingested. Their parents do not usually blatantly reject the child. The mothers characteristically have multiple children or responsibilities. They are usually disorganized, and the children do not receive the food or the attention they need, but the attention they receive is usually adequate for infants to again grow, if they are given adequate nourishment. Nevertheless, growth in some may be inadequate without further psychosocial interventions, as reported by Bithoney et al.⁷⁻⁹

Type II PSS has been called transient hypopituitarism, reversible hyposomatotropism, emotional deprivation, maternal deprivation, psychosomatic dwarfism, abuse dwarfism, and the "garbage can" syndrome. The term PSS is preferable to definitions that include the presence or absence of GH, the presence or absence of overt psychologic abuse, or emotional deprivation. This type occurs characteristically in children 3 years of age and older.

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Table 2

Characteristics of Various PSS Syndromes¹

Type	Age of Onset	Failure to Thrive	Bizarre Behavior	Depression	GH Secretion	Parental Rejection	GH Responsiveness
I	Infancy	Usually	No	Often	Normal	No (see text)	?
II	≥3 years	Some & some overweight	Usual	Very often	Decreased or absent often	Usual	Minimal at doses used
III	Infancy or later	Not usual	Not usual	Yes	Normal	Concern, not rejection	Significant at dose used

Adapted from: Blizzard RM, Bulatovic A. In: Lifshitz F, ed. *Pediatric Endocrinology 3rd Edition*. New York: Marcel Dekker, 1996:83-93.

There is a greater psychologic component, and GH response may be inadequate after stimulation with pharmacologic agents, such as arginine or insulin. Other abnormalities indicating adrenocorticotrophic hormone (ACTH), thyroid stimulating hormone, and gonadotropin deficiency may be noted; however, GH deficiency is the most common endocrine aberrancy. The parents in this group usually reject their children and abuse them psychologically. The fathers and/or mothers are frequently chronic alcoholics. Occasionally type I patients are observed to advance into type II, which is not surprising.

Type III of PSS was described by Boulton et al,¹⁰ who studied seven children aged 3.6 – 11.6 years who did not have the bizarre signs and symptoms of type II patients. They were significantly depressed and/or had a disorder of attachment often dating from infancy. In contrast to previously reported patients they secreted GH when tested and had a significant increase in growth when given growth hormone treatment. A lesser response was obtained with a placebo. The authors emphasized that type III PSS patients did not show lack of discrimination in relationships, nor did they display the self-destructive behavior, pain agnosia, or bizarre eating and sleeping disorders seen in many type II patients. In addition, the parents were not indifferent and rejecting, as are those with PSS type II. The parents also had insight into the problem, which was not characteristic of the parents of other patients with PSS and several felt guilty and/or had depression.

The classifications discussed here by the English group and that presented in Lifshitz's *Endocrine Text* are compatible. Type I, as described above, should remain as type I and be applied to infants and very young children. Type II pertains to children with severe PSS of the hyperphagia type. In my opinion, type II should be limited to this group. Type III is where further subclassifications should be placed. For example, type IIIA could (should) be the group described by Boulton¹⁰ and type IIIB of the type referred to by Gohlke et al. With this classification type IIIA & B can be subdivided or a type IV added as further subgroups are recognized. I wonder if Drs. Gohlke et al or others agree with my thinking? A letter to the Editors of GGH will be most welcome.

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Robert M. Blizzard, MD

Leanness, Extended Lifespan & IGF-1 Receptor Mutations in Mice: Fascinating Observations

In flies and worms, loss-of-function mutations in insulin and insulin-related cell signaling pathways have led to *increase* in life span of the species studied. In order to evaluate these pathways in a mammalian specie, the

present investigators developed mice with hemizygous loss of one insulin-like growth factor-1 receptor (IGF-1R) allele and studied their longevity. The hemizygous IGF-1^{+/-} mice were generated by deletion of exon 3 of