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Small for Gestational Age: An Auxological Perspective

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ABSTRACT: *Depending upon timing and severity of insult/ nutritional deprivation during pregnancy, both symmetric and asymmetric SGA differ substantially in their etiology and thus also for implications of their postnatal physical growth and survival. Besides documenting etiological, classificatory and diagnostic aspects in this article, postnatal growth patterns encountered amongst symmetric and asymmetric SGA children representing different parts of the world have been reviewed. In majority of the instances, symmetric SGA children experienced substantial growth retardation and never caught up with their asymmetric counterparts. In some cases, as compared to symmetric SGA infants growth impairment was found to be more in asymmetric SGA children. Additionally, no difference in growth attainments of these two categories of SGA children was observed by some other researchers. Owing to aforementioned prevailing trilogy, no clear picture with regard to postnatal growth patterns of SGA children emerges hence, decision remains inconclusive. Obviously, to have insight into the auxological dynamics of both asymmetric and symmetric SGA children, there is a need to carry out well framed longitudinal studies on representative samples of children being reared world over under different ethnic, nutritional, economic and socio-cultural conditions.*

Keywords: Birth weight, Length, Brain-sparing, Symmetric SGA, Asymmetric SGA, Postnatal growth, Ponderal Index

INTRODUCTION:

Birth weight, is recognized as a significant measure of newborn infant's chances to survive and grow in health (WHO 1995, McCormick 1985). It usually predicts growth of a child over the subsequent years of life and is often considered as a risk factor for future medical conditions (Michels et al 1996). It is well known that relatively poor intrauterine growth raises risk of perinatal and infant morbidity, mortality (Kleigman et al 1990, Pollack and Divon 1992) as well as of compromised growth and development. Barker et al (1993) opined that adverse influence of intrauterine milieu not only remain restricted to fetal life but also continues during subsequent stages of postnatal life. Under normal circumstances, the birth of normal healthy newborn of appropriate size is a result of the neonate's, inherent growth potential (Peleg et al 1998). According to WHO (1961), birth of a preterm (i.e. gestational age <37 weeks) or low birth weight (i.e. LBW <2500 g) baby is because of adverse influence of intrauterine factors. The majority of LBW infants in the developed world are born preterm, whereas in the technologically under-developed world, most of the LBW babies are full-term. Incidence of LBW is highest in South Asia, with an estimated prevalence of 28% in India (UNICEF 2014).

Since the mid 17th century, prematurity was the presumed cause of small size at birth until the 1950s (Baker and Tower 2005). In 1917 the Finnish pediatrician Yllpö (Tanner 1979) considered babies having birth weight < 2500 grams at birth, as premature. During 1920s to the 1960s, clinicians used terms 'premature'and 'LBW' interchangeably. Around 1940s it came to the knowledge of physicians that low birth weight baby necessarily be not an infant born preterm, but might be result of intrauterine growth impairment. The first report of an 'under-nourished full-term infant' was published in 1947 (McBurnley 1947), leading to the recognition that some infants less than 2500 g were not premature, but were too small (Baker and Tower 2005). Thus, it became amply clear that not all small babies were premature, and similarly not all prematures were small in size. Accordingly, use of LBW as per recommendations of WHO (1961) was officially, excluded from scientific definition of prematurity.

Recognizing, LBW and preterm as separate entities, researchers found that full-term babies having birth weight less than 2500 g had higher risk of mortality. If not preterm delivery, then what accounts for this risk? The answer to this query was a newly named disease i.e. intrauterine growth retardation (IUGR), having same cut off value as that for small for gestational age (SGA). SGA being a statistical terminology (Yanney & Marlow 2004) bears significant relationship with birth weight and/or length with gestational age hence is a diagnosis at postpartum state (Lee et al 2003). By definition "Small for Gestational age" (SGA) defines a neonate who at birth weighs < 10th percentile of gestational age and sex of accepted reference standards (Bakketeig 1998, WHO 1950). The most frequently used intrauterine growth standards to diagnose SGA babies are those of Lubchenco et al (1963), Lubchenco et al (1966). These curves have frequently been used world over, to categorize newborns as small for gestational age (SGA), appropriate for gestational age (AGA) or large for gestational age (LGA) (McIntosh & Stenson 2008, WHO 1950). Accordingly, infants weighing above 90^{th} percentile are classified as LGA, infants between 90^{th} and 10^{th} percentile as AGA and infants weighing below 10^{th} percentile as SGA. Majority of LBW babies born at full-term in South Asia are SGA, the prevalence which is about two times that recorded for LBW. India had largest share of SGA births, estimated to be around 12.8 million in year 2010 (Black et al 2015). According to Arya (2006), 3-10% of the total babies worldwide are born SGA and its prevalence in India is extremely high i.e. 47% (Lee et al 2013, Black 2015).

DEFINITION & NOMENCLATURE:

According to Lee et al (2003), the term SGA does not denote extent of fetal growth but pertains to newborn's size at birth which may be a result of the reduced growth of the fetus. An interchangeable use of terminologies i.e. intrauterine growth restriction (IGR) and/or retardation (IUGR) with SGA in medical literature suggests presence of inhibiting pathophysiologic processes affecting growth of fetus during pregnancy. Unfortunately, this overlap and interchangeable use of terminology yielding confusion in the nomenclature was officially recognized by the National Institute of Child Health and Human Development (USA) which stated that "Both for clinical and research purposes, IUGR birth should be defined as that resulting in a birthweight less than the 10^{th} centile for gestational age, using criteria appropriate

to the population under study" (Mahadevan et al 1994). IUGR speaks of a reduced rate of growth in the fetus as often ascertained by at least two periodic ultrasonic intrauterine growth assessments (Hokken-Koelega 2001, Lee et al 2003). Thus, IUGR is a prenatal diagnosis and should not to be used as a replacement for SGA, which in fact remains a postpartum diagnosis (Lee et al 2003). According to "Pediatric Endocrine Societies and the Growth Hormone Research Society" the definition of SGA requires 1) precise estimation of gestational age (based on ultrasound examination during first trimester), 2) correct recording at birth, of body weight, crown-heel length and head circumference 3) a cut-off based on population specific reference data (Clayton et al 2007). Hence, anthropometric assessment of newborn infant populations for evaluation of nutritional status is considered as valid tool to evaluate influence of restricted (or excessive) growth of the fetus. Clayton et al (2007) recommended that postnatally, SGA children should be measured for length, weight and head circumference quarterly and thereafter, half-yearly. Serial anthropometry helps to determine alterations in the growth pattern of individual children known to be affected by genetic and environmental factors, of which latter are known to be of more pervasive in nature (Eveleth & Tanner 1990).

ETIOPATHOGENESIS AND CLASSIFICATION OF SGA:

Small for Gestational age infants envisage heterogeneous subgroups, with different etiologies. Depending upon magnitude of insult/ nutritional deprivation and its timing, SGA infants are usually categorized into symmetrical (proportionate (chronic), type I, stunted) and asymmetrical (non-proportionate (acute), type II, wasted) phenotypes (Gruenwald 1974, Villar & Belizan 1982). Inhibiting factors which occur during early pregnancy (1^{st} trimester) due to chemical exposure, viral infection and inherited abnormality of cellular development result in symmetrically growth impaired fetus. On the other hand, occurrence of insult during later phase of pregnancy yields asymmetrical growth retardation (Villar and Belizan 1982, Bakketeig 1998, Thureen et al 2001). As insult becomes progressively more chronic and severe, first weight, then length and finally brain mass are affected. Infants affected during first trimester of pregnancy, exhibit impaired growth of body weight, crown-heel length and head circumference i.e. symmetric growth retardation (Campbell 1974, Kelnar & Butler 2008). If insult during pregnancy occurs later, then brain remains unaffected i.e. spared (as head circumference measures normal)

but length will be decreased as well as weight, resulting into asymmetric growth retardation. The diagnosis of two etiologically different types of SGA i.e. symmetric versus asymmetric smallness (Gould 1986) is often made by using Rohrer's Ponderal Index (Miller & Hassanein 1971, Walther & Ramaekers 1982, Singh 1993, Bakketeig 1998, Akram & Arif 2005). Due to significant reduction in weight and a slight reduction in length, asymmetric SGA babies have a reduced Ponderal index (less than 2.0). When ponderal index is more than two, then the SGA baby is known to possess symmetrical growth retardation (Singh 1993, Bakketeig 1998).

The use of same single cut-off value for Ponderal Index (i.e. < or > 2) for categorizing both pre-term and full-term infants into two sub-types of SGA is in contradiction with recommendation of Miller and Hassanein (1971), who advocated the use of PI with value of 2.0 in case of pre-term and 2.2 as a cut-off level for categorizing babies born at full-term into two sub-types of SGA. It has further been observed that this cut-off suggested by Miller and Hassanein (1971) also matches with 10^{th} percentile values for Ponderal Index published by Lubchenco et al (1966) as well as by Mohan et al (1990) amongst full-term SGA infants of American and Indian origin, respectively.

CLINICAL SIGNIFICANCE OF TWO TYPES OF SGA:

The diagnosis of SGA baby becomes important as these infants remain at higher risk of mortality in the perinatal, neonatal and post-neonatal period (Bernstein et al 2000, Westby Wold et al 2009), morbidities like metabolic and neurodevelopmental disorders & short stature in life (Clayton et al 2007, Khadilkar et al 2016). Over the years a rise in incidence of cardiovascular and cerebrovascular diseases, hypertension and Type II diabetes mellitus amongst SGA infants in adult life has been reported by Barker et al (1989). Visceral adiposity amongst SGA children despite their not being overweight was also documented (Ibanez et al 2008). Studies documenting morbidity and mortality figures in infants with two different types of SGA are fewer. Two times higher mortality rates in symmetric and 2.9-5.7 times more in asymmetric growth retarded infants than normal infants have been recorded by Haas et al (1987). Lin et al (1991) stated that term infants with symmetric growth retardation not only demonstrate greater neonatal morbidity rates but also possess lesser mean birth weights than those with asymmetric growth retardation. The effect of this disadvantageous start tends to persist during postnatal

years. A failure to experience catch-up growth by around 10% of SGA infants by 2 years (Lee et al 2003), and short stature during childhood and adulthood (Carrascosa et al 2006) was found to be associated with psychosocial disadvantages and behavioral problems (Hoey 1993). Hediger et al (1998) found that despite catch-up growth, SGA infants of American origin remained shorter, lighter and possessed smaller head circumference throughout early childhood as compared to infants born AGA. Similar findings have also been reported on children of Indian origin (Bhargava et al 1984).

AUXOLOGICAL IMPORTANCE OF SYMMETRIC & ASYMMETRIC SGA:

The physical growth and developmental attainments of SGA babies during postnatal years depend on the cause of restriction, nutrition in infancy and the social environment (Kleigman 1997). The two subtypes of SGA infants differ substantially in their etiology and implication for physical growth, survival, morbidity and cognitive development. The patterns of catch-up growth tend to differ across SGA subtypes. In conditions of optimal early nutrition, babies with asymmetric growth retardation usually catch-up to within the normal centiles after birth whereas many symmetric growth retarded infants do not (McIntosh & Stenson 2008). It is therefore, expected that these babies may continue to behave differently as far as the course of their auxological attainments during different phases of postnatal growing years is concerned.

Physical growth amongst 61 full-term newborns with Low Ponderal Index (asymmetric) and High Ponderal Index (symmetric) born at the University of Kansas Medical Centre was studied during infancy (Holmes et al 1977). Infants with LPI accelerated their rate of weight gain which improved their malnourished state. However, slower postnatal growth in infants with HPI was considered a continuation of the pattern of their growth during fetal life. Similarly, 24 wasted (asymmetric) white, singleton term infants born in Leicester grew faster in weight, head circumference and crown-heel length than the 16 non-wasted (symmetric) infants (Davies et al 1979). Full-term SGA babies studied for intrauterine cephalometric growth by means of serial ultrasonography, were re-examined at 4 years of age by Fancourt et al (1976). Authors concluded that children with prolonged in utero slow growth, postnatally also experienced poor growth and development.

Crane & Kopta (1980) compared the influence of asymmetric and symmetric growth retardation on newborn anthropometric measurements and noted that the percentage decrease in head circumference was similar in both groups. Therefore, the concept of absolute "brain sparing" in asymmetric retardation was considered erroneous, and it was recommended that ultrasonic measurements of Bi-parietal diameter should be of value in detecting this form of intrauterine growth retardation. To verify the hypothesis that growth retardation in symmetric SGA infants starts in the 1st trimester, and amongst asymmetric ones it begins during third trimester of pregnancy and also to confirm existence of brain-sparing effect in asymmetric SGA infants, Vik et al (1997) conducted a study on 163 symmetric, 108 asymmetric SGA and 474 non-SGA infants of Caucasian origin inhabiting Norway and Sweden. Fetal abdominal diameter, femur length and bi-parietal diameter at week 17, 25, 33 and 37 of gestation were ultrasonographically measured to study intrauterine growth during prenatal life. Their findings revealed that study subjects representing both SGA groups experienced growth retardation during second trimester. The absence of significant difference in the prenatal growth pattern of two types of SGA refuted the hypothesis that retardation of growth in symmetric SGA infants occurs during first trimester. Also authors didn't observe 'brain sparing' in asymmetric infants when contrasted with symmetric SGA infants.

Villar et al (1984) studied 59 term infants of Guatemalan origin with two subtypes of intrauterine growth retardation and concluded that infants tend to follow growth pattern associated with their physical characteristics at birth as infants with asymmetric growth retardation measured heavier, lengthier and possessed larger head circumference than symmetric ones. Similar results on a group of 488 term SGA infants categorized as Type I (adequate ponderal index) and Type II (low ponderal index) studied in Finland upto first two years of life are also reported (Tenovuo et al 1987).

In 1989, Adair conducted a prospective survey of births in a community in Cebu region (Philippines) and reported that infants who possessed adequate Rohrer's Ponderal Index (symmetric) were smaller at first year of life than infants who had a low Rohrer's Ponderal Index (asymmetric). The author emphasized on the diagnostic value of Ponderal Index and the need for classifying SGA infants with intrauterine growth retardation on its basis.

As compared to non-growth retarded infants with same gestational age, the growth retarded infants (classified as mild, moderate and severe on the basis of fetal growth ratio) born at Montreal's Royal Victoria Hospital, Canada were found to possess substantially lower length, head circumference and proportionality ratios. The magnitude of the observed deficits increased significantly with increase in the severity of growth retardation (Kramer et al 1989).

Williams et al (1992) studied IUGR infants born at term at 7 years and 18 years respectively in New Zealand. Both the groups (symmetric and asymmetric) had similar height and weight measurements at both 7 and 18 years. However, these values in general remained lower than that of controls. Newman et al (1997) conducted an interesting prospective study in Australia amongst 65 SGA infants weighing < 3rd centile at birth and 71 control infants at 4 months of corrected age. They hypothesized that differences would exist in growth and development, and that these would relate to type (symmetric/asymmetric) of growth restriction present at birth. Neither type of SGA infants demonstrated any prognostic importance for catch up growth and neuromotor developmental scores.

Strauss and Dietz (1997) evaluated data on growth of 818 infants of IHDP (Infant Health & Development Program) cohort between birth to 36 months diagnosed as symmetrically and asymmetrically growth retarded. Infants born with symmetric growth retardation demonstrated increase in growth velocity until 8 months while, infants born with asymmetric growth retardation demonstrated deceleration for weight growth velocity as compared to that of AGA.

The catch-up growth in length was studied amongst 73 infants measuring below 10^{th} percentile for length of acceptable standards at birth, admitted to Neonatal Intensive Care Unit (NICU) by Seminara et al (2000) in Italy. Children were measured at birth, 3 and 6 months and half yearly till 2 years of age and thereafter, yearly until 6 years. 44% of children caught up with normal population standards by 3 months, 51% by 3 years and 66%, 73% by the age of 4 and 6 years, respectively. However, aforementioned trend remained independent of gender as well as type of SGA (symmetric vs. asymmetric).

Fewtrell et al (2001) documented that infants with lower ponderal index (asymmetric) at birth (recruited from five UK hospitals located in Cambridge, Nottingham and Leicester) demonstrated statistically significant increase in body

weight, crown-heel length and head circumference by the age of one and a half year than infants with high ponderal index (symmetric).

Growth pattern were studied among 74 LBW infants born in Woodbury Country, Iowa by May et al (2001). These infants were registered in the Special Supplemental Nutrition Program for Women, Infants and Children (WIC). Most of the subjects were of Hispanic or Caucasian origin. Postnatal growth was studied in terms of body proportions (i.e. symmetric and asymmetric) using ponderal index. It was observed that infants with LPI (asymmetric) were lengthier than proportionate PI (symmetric) infants at birth having similar weight z-scores. At 8 months of age, both types of infants showed improvement in their weight and length. Whereafter, asymmetric infants with respect to weight and length improved further, in contrast to symmetric infants who remained lighter and shorter. Harding and Mc Coman (2003) observed that SGA babies with relatively lesser growth restriction and its late onset had better chances for growth catch-up in 1^{st} half of infancy.

Ochiai et al (2008) longitudinally followed 56 Japanese SGA (34 asymmetric and 22 symmetric) infants for physical growth upto 6 years (measured at 6, 12 and 18 months and 2, 3, 4, 5 and 6 years of age). Growth was impaired in symmetrical SGA infants and in SGA infants having a head circumference < 10^{th} centile as compared with asymmetrical SGA infants and those having head circumference > 10^{th} centile, respectively. The authors concluded that growth catch-up in head circumference appears to be a good predictive tool for neurological development in SGA infants.

The sole longitudinal growth study carried out on symmetric SGA (n=100), Asymmetric SGA (n=100) and AGA (n=100) infants of Indian origin in the Child Growth and Anthropology Unit of the Advanced Pediatrics Centre (APC), Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh, like their aforementioned western peers also depicted impaired growth for weight, length (Kaur et al 2016) and head circumference (Kaur et al 2012) in symmetric SGA infants when contrasted with their asymmetric SGA counterparts.

Growth and neurodevelopment outcome were studied amongst 194 term SGA infants (38.7% Symmetric; 61.3% Asymmetric) in France (Maciejewski et al 2016). The asymmetric group showed better catch-up growth with larger head

circumference (p=0.03) than the symmetric group who were also found to be at a higher risk of delayed developmental outcome.

Attempts made world over by earlier workers, showed that postnatally babies having symmetric growth retardation exhibited poorer auxological prognosis, and also measured lighter (Lin et al 1991), shorter (May et al 2001, Kaur et al 2016) and also possessed smaller head circumference (Holmes et al 1977, Davies et al 1979, Villar et al 1984, Tenovuo et al 1987, Fewtrell et al 2001, Ochiai et al 2008, Kaur et al 2012, Maciejewski et al 2016) than their asymmetric counterparts. On the contrary, available information on the topic (Strauss and Dietz 1997) indicates that disproportionate (asymmetric) infants also experienced severe growth impairment than the babies with proportionate (symmetric) retardation. However, information presented by other researchers (Williams et al 1992, Seminara et al 2000) did not reveal any difference in postnatal growth attainments of asymmetric and symmetric SGA children. Existing evidence also suggests that proportionality among these infants represents a continuum, with progressive disproportionality (asymmetry) as severity of growth retardation increases (Kramer et al 1989, Wollmann 1998, Martinez & Simons 2005).

An inspection of the available information on the postnatal pattern of growth amongst two sub-types of SGA does not reveal any clear picture hence, situation remains inconclusive. However, studies revealing poorer growth attainments amongst symmetric SGA than their asymmetric counterparts outnumber those demonstrating impaired growth in asymmetric SGA as well as those exhibiting no difference amongst two SGA subtypes. Therefore, to have thorough understanding about growth kinetics of two sub-types of SGA children during their different phases of postnatal life, the need to carry out growth assessment periodically on adequate sample of children representing diverse population ecologies, environmental, nutritional, socio-economic conditions becomes obvious to arrive at scientifically meaningful auxological inferences.

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