Deformational Plagiocephaly, Brachycephaly, and Scaphocephaly. Part I: Terminology, Diagnosis, and Etiopathogenesis

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Abstract: Cranial deformation is the most common cause of abnormal head shape. Intentional and unintentional alterations of cranial form are associated with the application of external pressure to the growing infant head, and such changes have been recorded throughout man's history. Recent changes in Western sleeping practices, instituted to reduce the incidence of sudden infant death syndrome, have led to a dramatic rise in the incidence of cranial deformation and renewed interest in this subject. This 2-part review presents a pragmatic clinical approach to this topic including a critical review of the literature as it applies to each aspect of this common diagnosis: historical perspective, terminology, differential diagnosis, etiopathogenesis and predisposing factors, and prevention and treatment.

Key Words: Deformational plagiocephaly, deformational brachycephaly, deformational scaphocephaly, congenital muscular torticollis, craniosynostosis, cranial deformation

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BACKGROUND

The American Academy of Pediatrics initiated the "Back to Sleep Campaign" in 1992 based on a mounting body of evidence that supine positioning of infants during sleep may reduce the incidence of sudden infant death syndrome.[1–3] This policy has been widely implemented in North America and resulted in a 40% reduction in the incidence of sudden infant death syndrome in the United States.[4] One of the unforeseen consequences of the campaign was an exponential rise in asymmetric (plagiocephaly) and symmetric (brachycephaly) occipital flattening.[5–8] Similar cranial shape changes (frontal plagiocephaly) had historically been observed in infants who slept prone,[9–14] but the prevalence and degree of flattening were considerably less. Recent studies estimate the prevalence of deformational posterior cranial flattening to be as high as 18% to 19.7% in healthy infants[15]; these calculations vary, depending on how this entity is defined.[16] For example, Hutchison and coworkers[15] followed up 200 normal infants from birth to 2 years of age. They defined deformational plagiocephaly (DP) as an oblique cranial length ratio of 106% or greater, and brachycephaly as a cephalic index (CI = cranial width/length) of 93% or greater. Using these definitions, the combined prevalence of flattening was as high as 19.7% at age 4 months and declined to 3.3% by 2 years of age. However, when flattening was defined as an oblique cranial length ratio of 105% or greater and CI of 91% or greater, the prevalence increased to 28% at 4 months and 12.7% at 2 years. There has been even more variation in the methods used to measure and report flattening, as discussed below.

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DIAGNOSIS AND TERMINOLOGY

Deformational Plagiocephaly

Deformational cranial flattening can take many forms, depending on the position of the infant's head during the first few months of life. Most clinicians incorrectly refer to any type of cranial flattening as "plagiocephaly." Plagiocephaly is derived from the Greek *plagios* meaning "oblique" or "slanted," and *kephalē*, meaning "head." Thus, the term *deformational plagiocephaly* is correctly applied to describe only flattening that is on one side of the head (Fig. 1). Deformational plagiocephaly occurs primarily in infants who consistently favor turning their head to one side, that is, those with congenital muscular torticollis (CMT).[17] The resultant cranial shape has been compared with a "parallelogram";[18] however, the frontal bossing is never equal to the degree of occipital flattening, and thus, the shape is really more trapezoidal. Asymmetric growth of the head often is accompanied by facial asymmetry, specifically an anterior shift of the ipsilateral forehead, ear, and cheek (Fig. 2). Asymmetric opening of the palpebral fissures can also be observed as a consequence of the sagittal displacement of the ipsilateral zygoma. As asymmetric occipital flattening progresses, forward movement of the zygoma and attached lateral canthus on the affected side effectively shortens the distance between the medial and lateral canthal tendons. As a result, tension is reduced on the tarsal plates, and the eye appears more open on the side of the flattening (Fig. 3). The vertical palpebral asymmetry can be easily confused with contralateral eyelid ptosis. Deformational plagiocephaly must be distinguished from 2 types of craniosynostosis that also can cause an asymmetric head shape: unilateral coronal synostosis (UCS) and lambdoidal synostosis. Both conditions are rare compared with deformational flattening. Unilateral coronal synostosis, or premature closure of 1 coronal suture, causes anterior plagiocephaly. Features of this entity include flattening of the forehead and superior orbital rim such that the anterior globe protrudes beyond these structures, nasal root and midfacial angulation, and anterior displacement of the ear ipsilateral (Figs. 4A, B). These features are not seen in DP. Asymmetry of the palpebral fissures in UCS can look similar to that seen in severe DP. However, the more-open–appearing eye in UCS is on the side of the flat forehead, whereas in DP it is on the side of increased forehead bossing. Lambdoidal synostosis, or synostotic posterior plagiocephaly, can be difficult to differentiate from DP. This condition is associated with asymmetric cranial height (shorter on the

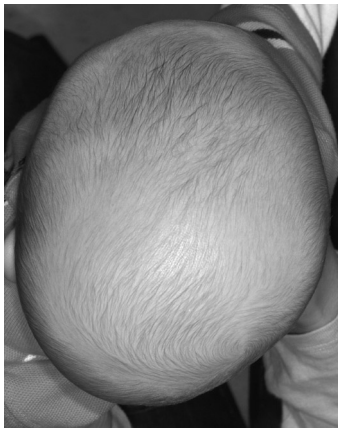

FIGURE 1. Right DP, vertex view. Note that shape is more trapezoidal and not a true parallelogram as often stated.

flattened side, the opposite of DP) that gives a "wind-swept" appearance to the head, and there is usually mastoid bossing on the affected side (Figs. 5 and 6).

Deformational Brachycephaly

Brachycephaly (Greek *brachy*, meaning "short") denotes symmetrical occipital flattening and compensatory parietal widening. Infants with deformational brachycephaly (DB) have little or no rounding on the back of the head and appear to have a disproportionately wide or "big" head viewed from the front (Fig. 7A). The posterior vertex may appear taller than the front (turricephaly), giving a sloped appearance to the head in profile (Fig. 7B). The ratio of cranial width to length, termed the cranial index or CI, is generally higher than normal (Fig. 7C)—this figure is historically 0.75 to 0.80 in North America, although some observers suggest that the normal CI has risen to 0.8 to 0.85 in response to back sleeping.[19] Most children with DB also have some element of asymmetry, or plagiocephaly. The combination effect, which I refer to as "asymmetric brachycephaly," is the most common type of deformational shape (Fig. 8). Brachycephaly can also be seen in infants with craniosynostosis when both coronal sutures are fused. Synostotic brachycephaly is relatively rare and has features not seen in DB: severe forehead retrusion such that the superior orbital rim is behind the anterior surface of the globe (eyes appear very prominent) and anterior turricephaly ("tall" head) (Figs. 9A, B).

Deformational Scaphocephaly

Deformational scaphocephaly (DS) ("boatlike head") is an uncommon variant of plagiocephaly. It is more commonly seen in

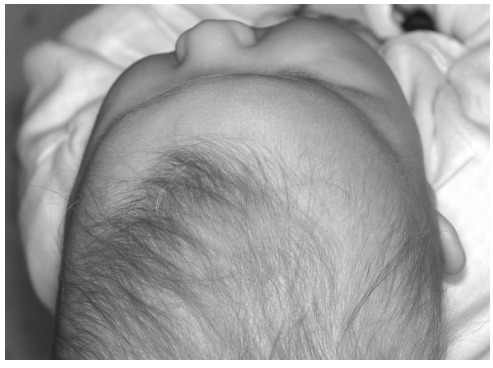

FIGURE 2. Right DP, vertex view. Anterior displacement of the right ear, cheek, and forehead.

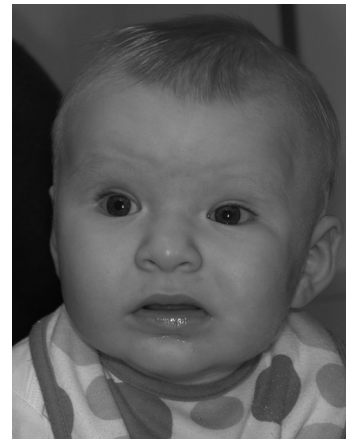

FIGURE 3. Right DP, frontal view. Forehead and cheek more prominent on the right than the left; right ear is anterior relative to the left in the sagittal plane; right eye appears more open than left; chin point rotated to left. Nose is straight.

infants who have extreme head rotation to one side or in premature infants who are positioned side-to-side in the intensive care units (Fig. 10). Flattening develops on the side(s) of the head, and compensatory expansion occurs in the anterior and posterior cranium. These infants tend to develop a long, slender head, colloquially referred to by some as a "toaster head." There is often relatively pronounced facial asymmetry. This presentation can be confused with scaphocephaly caused by premature fusion of the sagittal suture. Unlike DS, sagittal synostosis typically results in frontal bossing, bilateral occipital/parietal narrowing posterior to the anterior fontanelle, and decreased vertical height of the posterior cranium (Figs. 11A, B). Facial asymmetry is rare in sagittal synostosis. Additionally, most infants with this type of craniosynostosis have a head circumference in excess of the 90th percentile.

Radiographic Imaging

If the diagnosis is unclear, the child should be referred to a specialist before ordering radiographic studies. The accuracy of plain radiography to diagnose suture fusion is questionable. Computed tomography is costly, often requires sedation, and involves

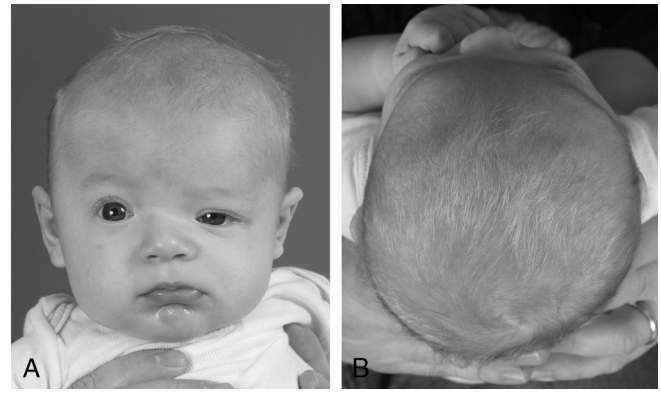

FIGURE 4. Right UCS. A, Frontal view. Similarities to DP include right eye appears more open than left eye, anterior displacement of the right ear, and chin point deviation to left; unlike DP, the nose is slanted. B, Vertex view. The forehead finding is the opposite of DP; that is, the forehead is severely flattened on the right.

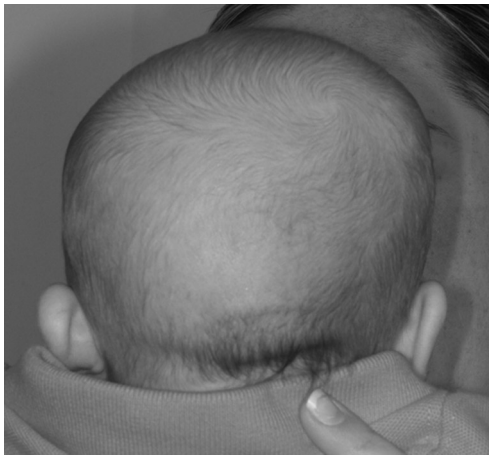

FIGURE 5. Right DP, posterior view. Compensatory increase in cranial height on side of flattening with level ears.

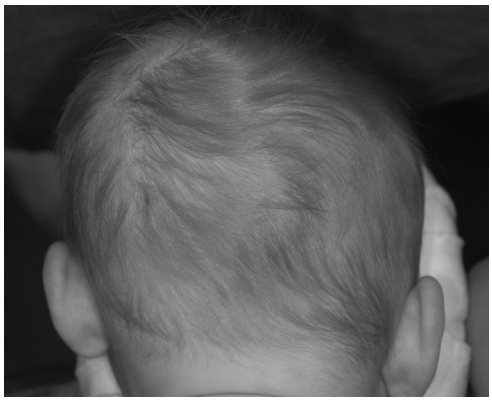

FIGURE 6. Right lambdoidal synostosis, posterior view. Decreased cranial height on affected right side, and the right ear is positioned inferiorly relative to the left.

low-dose ionizing radiation. The impact of such exposure is controversial; however, theoretical studies suggest that even a low-dose computed tomography in an infant can increase the risk of lethal brain cancer.[20,21] Because the overwhelming majority of infants with cranial asymmetry will have deformation and not synostosis, it is impractical to have every child with cranial flattening undergo imaging. A specialist can usually distinguish these processes by history and physical examination and should make the decision whether radiologic imaging is necessary.

FIGURE 8. Asymmetric brachycephaly.

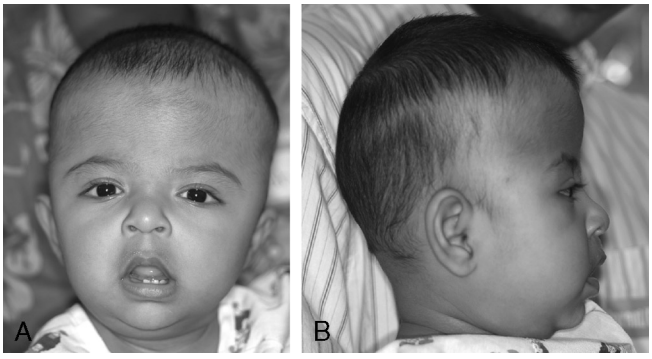

FIGURE 9. Synostotic brachycephaly (bilateral coronal synostosis). A, Frontal view. B, Lateral view. Unlike DB, the turricephaly involves both the anterior and posterior cranium.

THE MECHANISM OF CRANIAL DEFORMATION

Several hypotheses have been proposed to explain deformational calvarial flattening. It is often suggested that the infantile cranium is "soft" or "malleable" and that this predisposes the bony plates to deform when the head lies on a planar surface.[22,23] This mechanism is analogous to the type of shape distortion that occurs when a water balloon is placed on a table. This concept is easily dismissed by merely placing a newborn on a firm, flat surface—the

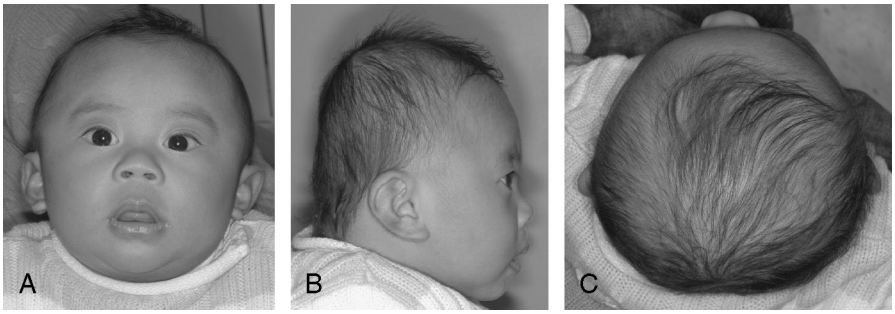

FIGURE 7. Deformational brachycephaly. A, Frontal view. B, Lateral view. C, Vertex view. The turricephaly (increased cranial height) is limited to posterior cranium with a severe loss of occipital projection.

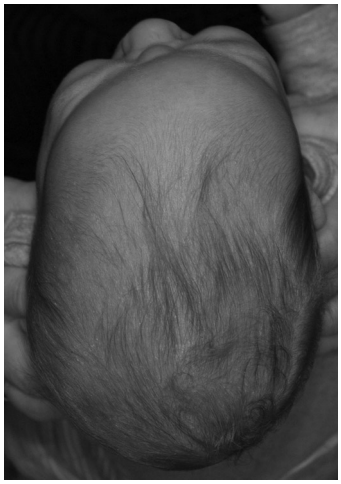

FIGURE 10. Deformational scaphocephaly. There is severe facial asymmetry; the cranium demonstrates a normal increase in width from anterior to posterior.

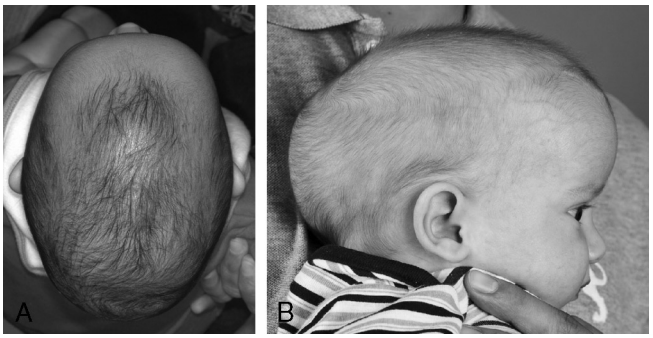

FIGURE 11. Synostotic scaphocephaly (sagittal synostosis). A, Vertex view. In contrast to DS, the posterior cranium narrows relative to the frontal regions, and there is no facial asymmetry. B, Lateral view. There are frontal bossing and depression of the posterior vertex, features not seen in DS.

head does not immediately deform. Furthermore, because all infants would conceptually have "soft heads," one would expect every newborn to undergo cranial flattening if this were true. In reality, less than a quarter of infants positioned on their back develop visible cranial flattening, and the severity peaks around 4 months of age.[15] In an attempt to reconcile these facts, some have proposed that susceptible infants may have an inherent problem with bone mineralization that makes them more susceptible.[24] There is no evidence to support this hypothesis either.

Another common, but generally incorrect, theme is that cranial flattening is hereditary. It is not uncommon for the parents to suggest that some member of the extended family has a similar cranial shape and that the baby "looks like Uncle Harry." This is particularly true in families whose cultural traditions include back sleeping. Although ethnic variations in the growth of the cranial base are largely genetic in origin, growth of the neurocranium (calvaria) occurs passively in response to expansion of the brain and intracranial contents.[25] Because the human cerebrum is not naturally flat or asymmetric, flattening can occur only when external force is exerted by a planar surface. It is interesting that many parents in my practice who make this argument are themselves not flat or asymmetric. Furthermore, they will often concede that the infant had a round and symmetric occiput at birth, but that the flattening developed after 2 to 3 months of age. I have observed several sets of monozygotic twins (genetically identical) in which each twin had a very different head shape from the other (Figs. 12A–D).

To better understand the mechanism of cranial deformation, it is instructive to look back in history. This is not a recent phenomenon. Intentional cranial deformation is the volitional alteration of normal head shape. This was practiced by many cultures including the ancient Peruvians, the North American Chinook Indians, and the French aristocracy.[26–29] In most instances, cranial deformation was accomplished by applying a constant external force, usually exerted by a board or cloth wrap, to the growing head. Over time, the natural shape of the head was permanently altered in a predictable and, presumably, culturally desirable way. Although intentional cranial deformation is no longer practiced, some culturally based rearing practices can lead to unintentional changes in cranial shape. For example, swaddle boards (hard, flat infant resting surface) are used in some Asian cultures. Swaddling diminishes infant mobility and, when coupled with supine positioning on a hard resting surface, results in a high rate of occipital flattening. Not surprisingly, cranial flattening is more common and culturally accepted in such regions. Comparisons between cultures that have historically positioned their infants supine during sleep (eg, Japan, Korea, India, and Pakistan) and those that have traditionally practiced prone positioning (eg, United States, Canada, Nigeria) demonstrate a higher CI in the former populations.[19]

Flattening can occur only when cranial expansion and growth are consistently resisted in a specific area by an external force.[30] The cranium grows passively in response to minor internal pressure exerted by the rapidly growing infant brain.[25] This process is fastest in early infancy and tapers dramatically even after the first year of life. When an infant is placed on a resting surface, there is a contact force generated between the head and the surface. The force applied by the head to the resting surface equals the weight of the infant's head multiplied by the force of gravity (F = mg). Newton's first law

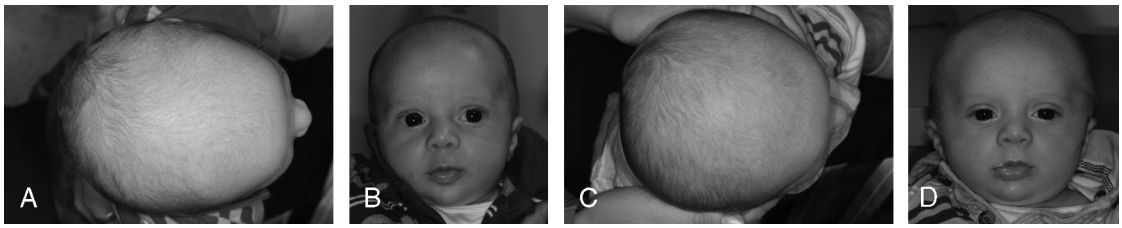

FIGURE 12. Monozygotic twins with different head shapes. A, Vertex view, twin with normal cranial shape. B, Frontal view, minimal cranial widening. C, Vertex view, twin with DB. Note occipital flattening and biparietal widening. D, Frontal view showing widened cranium.

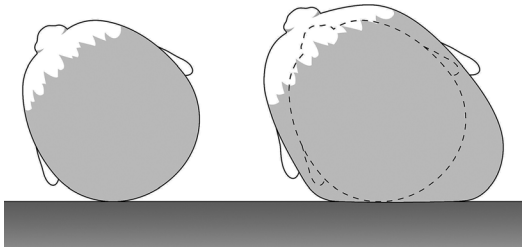

FIGURE 13. Mechanism of cranial deformation. Newborn with head rotational preference; the left occiput in constant contact with the resting surface (left image). Left DP develops after cranial expansion occurs around the fixed point of contact (right image).

predicts that for an object at rest, there will be an equal, but opposite, force from the bed to the infant's head. This counterforce will resist cranial growth in the area of contact, and consequently, volume increases will be displaced to areas where there is no resistance. Over time, this compensatory growth leads to cranial deformation and flattening (Fig. 13). Thus, the pathogenesis of DP, DB, and DS is analogous to how a pumpkin flattens as it grows in a field—it cannot expand into the ground and must grow along it (Figs. 14A, B). This explains why most parents begin to notice head flattening in their infants at an average of 6 to 8 weeks of age[10,31–37]—it takes this long for cranial flattening to occur. A larger pumpkin (ie, one growing faster) exerts a greater downward force on the ground (and the ground on it) than a smaller pumpkin, and consequently, the degree of flattening that occurs over a given time is proportionately greater (Fig. 15). This concept may explain the observation that flattening is more common in male infants,[8,10,32,34–40] as they have larger and faster-growing heads than females.

PREDISPOSING VARIABLES

Even if the mechanism of cranial deformation can be explained, it is not so easy to predict why this occurs in only some infants. Parents of an affected infant often ask why their child's head flattened, whereas other supine-positioned infants in their baby group or family did not. Many authors have struggled with this question. It is useful to discuss some of these well-intentioned, but misleading, ideas.

One commonly held belief is that flattening begins in utero and progresses after birth.[9,10,13,14,41,42] According to this logic, the "congenital" flat head would be the most comfortable and geometrically most likely spot on which the infant would lie. Over time, prolonged contact between the same area of the occiput and the sleeping surface leads to progressive flattening.[12] In support of this view, Petisch and coworkers[41] documented localized flattening in

FIGURE 15. The pumpkin analogy. The degree of deformation is proportionate to the rate of growth against a constant and fixed external force. This may explain why infants with larger average head sizes (eg, males and larger infants) and those with rapid rates of head growth (eg, premature infants) are more likely to develop DP and DB.

13% of otherwise healthy newborns. The measured asymmetry was in the order of several millimeters and may be in the realm of normal. Graham and colleagues[43] found an average 3 mm of asymmetry in normal 6-month-old infants. It is improbable that such a minor geometric disparity would have a prolonged impact on the infant's ability to change head position. This theory has been called into question by van Vlimmering and colleagues, who found no correlation between cranial asymmetry at birth and subsequent occipital flattening at 7 weeks.[37]

Back sleeping is another easy etiologic scapegoat. Given the acute rise in observed cases since the Back to Sleep Campaign, it has become an almost knee-jerk response to blame back sleeping exclusively for head flattening.[5,15,22,23,35,36,41,44,45] Although back sleeping has clearly increased the observed incidence of cranial deformation,[6,8] it cannot be the only etiologic factor. Deformational flattening also occurs in prone-positioned infants and was well described long before supine sleeping was commonly practiced in the West.[6,11–14] Additionally, most infants who are positioned supine do not develop clinically significant occipital flattening.[15] One factor is that brain growth and maturation (and consequently cranial growth) in the occipital/parietal region of the cranium are more pronounced than the frontal area during infancy.[46] Because deformation is a

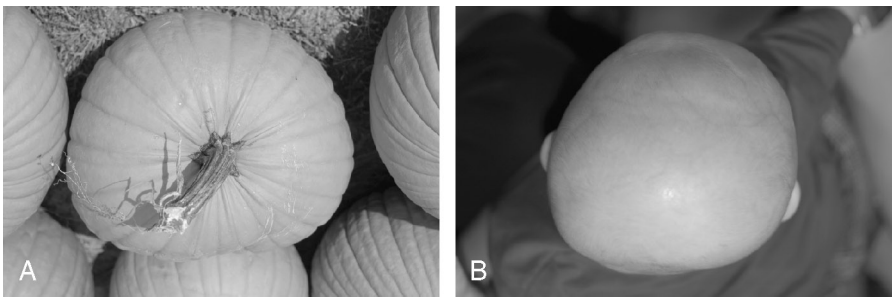

FIGURE 14. The pumpkin analogy. A, A stationary pumpkin growing against a firm planar surface will become flat over time. B, Similarly shaped infant cranium resulting from the same mechanism.

TABLE 1. Risk Factors of Plagiocephaly and Why They Are Associated

	References	Reason for Association
Prematurity	6,33,35,36,40,44,47,48	Neuromuscular immaturity results in delayed head mobility
Developmental delay	44,49–53	Slower neuromuscular development delays independent head mobility; increases time infant at risk for flattening
Torticollis	6,8,10–13,15,17,31,33,35,36,38,40,44,45, 48–50,54–61	Cervical contracture/imbalance restricts independent infant head mobility
(1) Multiple gestation pregnancy	6,17,33,39,40,48,49,59,61	Increased risk of CMT due to uterine crowding; increased likelihood of prematurity
(2) First born	15,35–38,50,54	Increased risk of CMT due to uterine crowding
(3) Male	6,8,15,17,35–40,49,50	Increased risk of CMT due to uterine crowding—on average, male infants have larger head and body than do female infants; faster head growth after birth also increases risk of deformation
(4) Assisted delivery	6,9,38,50	Increased risk of CMT due to abnormal fetal position in utero or large infant
(5) Breech	35,54	Increased risk of CMT due to abnormal fetal position in utero
(6) Positional preference	15,17,35,37,40,44	Earliest manifestation of torticollis

consequence of redirected growth, it is logical that faster-growing regions would be more affected than slower-growing ones. It is reasonable to hypothesize that back sleeping amplifies the effect of certain risk factors for DP,[39] but is not the cause per se. Because the rate of deformation is increased by back sleeping versus prone sleeping, the severity of flattening that develops in a given time is also greater in the former group than the latter group. Consequently, supine-positioned infants are more likely to be identified as flat.

Cranial flattening occurs in infants who have limited head mobility early in life. Most term infants develop sufficient strength and coordination to support their head against gravity by 3 months of age, and further flattening is unusual after this age.[15] Any intrinsic or extrinsic factors that limit the ability to change an infant's head position during the first few months of life greatly increase the likelihood of cranial deformation (Table 1). Therefore, it is understandable why premature or developmentally delayed infants have a higher risk of DP and DB,[47,49,50] as each of these risk factors leads to a delay in independent head mobility. Nevertheless, these infants comprise a minority of children with DP.

Torticollis/Cervical Imbalance

The most significant condition that limits head rotation is CMT. This is found in up to 70% to 95% of infants with DP.[6,8,10–13,15,17,31,33,35,40,44,45,48–50,54–60] Many of the known risk factors for plagiocephaly also increase the likelihood of in utero constraint and CMT (Table 1). This imbalance in the cervical muscles results from immobilization of the head in utero[10,12,17,45,50,55] and varies in severity, depending on the duration of immobilization. It has been observed in 16% of healthy newborn infants.[60] As with any muscle group, the cervical muscles require mobilization to maintain strength, flexibility, and tone. Immobilization of the fetal head in a rotated and tilted position can result in shortening and contracture of one sternocleidomastoid muscle and relative elongation and atrophy of the opposite sternocleidomastoid. The scalene muscles can also be affected to a lesser degree. The resulting cervical muscular imbalance causes ipsilateral head tilt, contralateral head rotation, and minor cervical extension. The position of the head during intrauterine immobilization impacts how the neonate will lie. Because most infants engage the pelvis in the left occiput anterior position (head turned to the right side), one would predict a tendency for affected infants to lay on their right occiput.[9,13] Indeed, in support of this concept, nearly every major study of DP has documented a higher incidence of flattening on the right occiput.[6,8,15,17,23,33,34,37–40,43–45,48,50]

The association between CMT and DP has been underreported.[17] This is principally attributable to differences in clinician

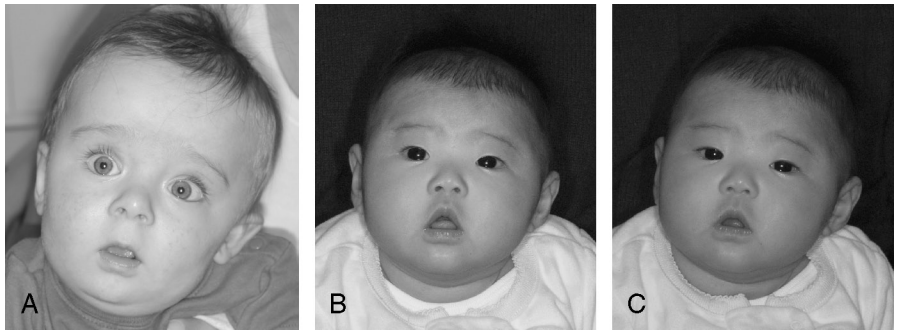

FIGURE 16. Head tilt associated with CMT. A, Severe tilt. Despite the striking appearance, this infant had excellent cervical range of motion (moderate overrotation to the right) and no neck tightness passively. B, Intermittent, minor head tilt is a common manifestation of resolving CMT. C, Tilt is usually more evident when the child is tired or preoccupied.

experience and diagnostic criteria. Pivar and Scheuerle[62] found that the published rate of CMT in infants with DP ranged from 5% to 67% in 18 treatment centers in Texas. These authors concluded that this inconsistency did not reflect true patient variability but was instead attributable to differences in the training and experience of the treating clinicians. Another factor that may cause underreported CMT in infants with DP is the strong tendency for the sternocleidomastoid imbalance to improve during the first year of life.[30,45,58,63–66] By the time many physicians see these patients, the findings are often minor or gone. Congenital muscular torticollis can transiently slow achievement of early motor milestones,[67] and this can further increase the risk of cranial deformation. The earliest manifestation of CMT is the tendency for the infant to maintain a particular head position despite attempts to reposition. We found that nearly all parents observed this in their plagiocephalic infants.[40] Both parents and clinicians often attribute this tendency to different environmental factors (eg, the side on which they feed the baby, the location of the bed in the room, etc). Nevertheless, attempts to alter the environment are almost invariably unsuccessful in altering the preference.

In my opinion, any infant with a "preferred head position" has a cervical imbalance, or torticollis, until proven otherwise. Failure to recognize head rotational preference in an infant as de facto torticollis has led some to incorrectly deduce that plagiocephaly can lead to torticollis.[68] In many instances, the classic head tilt associated with CMT manifests only when the infant attempts to balance the head weight against gravity at about 3 to 4 months of age. Before this, the neonatal head is almost constantly supported by a resting surface, and the cervical muscles provide only minimal head support. Until there is a true gravity "challenge" to the cervical muscles, head tilt may not occur. Accordingly, we have found that the presence of a head tilt is a less reliable and late physical finding compared with head rotational discrepancy for diagnosing CMT.[17] Interestingly, the head tilt seen in most 5- to 6-month-old infants with CMT is not related to muscle tightness on the side of the tilt, but weakness of the SCM on the opposite side (Fig. 16A). The tight SCM often stretches out much earlier (typically by 4–5 months) than the weak SCM muscle strengthens. The presence of unilateral SCM weakness will result in intermittent head tilt, especially when the child is tired or preoccupied (Figs. 16B and C). This type of tilt is not the sign of a contracted muscle, which would create a consistent head tilt and a major head rotational disparity. Failure to understand this difference often leads to fruitless attempts to treat the tight muscle with manual stretching or a cervical collar (eg, TOT, Symmetric Designs, Salt Spring Island, British Columbia, Canada) when, in fact, the correct management is to strengthen the weak contralateral SCM.

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