Normal Variants and Congenital Anomalies in the Region of the Obelion

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A number of anatomic bony variants and several congenital anomalies are known to occur at the level of the obelion, that is, in the posterior interparietal region of the head, a short distance in front of the lambda. Among them are: (1) remnants of the embryonic parietal incisura, including parietal fissure, small and large parietal fontanelle, obeliac bones, persistent midline parietal foramen, and small and large parietal foramina; (2) encephalomeningoceles and related defects, including true encephaloceles, herniation of midline intracranial cysts (diencephalic and Dandy Walker cyst), epicranial arachnoid cysts, and scalp defects with ectopic glial tissue; and (3) congenital scalp and skull defects (cutis aplasia congenita). A review of these defects is presented with some illustrative examples not previously reported.

The obelion—probably so named from its resemblance to an obelus or obelisk (÷), a symbol used in ancient manuscripts to mark suspected spurious or superfluous passages or words [1, 2]—has been defined [3] as the region of the skull situated between the two parietal foramina, where the sagittal suture is normally of a more simple type and where its closure usually begins. This generally occurs about 2 cm in front of the lambda in the newborn and 2–5 cm in front of the lambda in adults [4] (fig. 1). The area is of interest since it is the site of a number of anatomic bony variants and several congenital anomalies either limited to the bone or involving the scalp and sometimes the central nervous system as well. These include (1) remnants of the embryonic parietal notch, (2) encephalomeningoceles and variants, and (3) congenital scalp and skull defects. A brief review of these conditions is presented with illustrative examples. (It is of interest to note that true midline dermoid cysts and sinuses, classically located in the midoccipital area, seldom if ever occur in this region.)

Parietal Notch and Derivatives

The parietal bone begins to ossify during the seventh or eighth week of fetal life from a single center according to some authors and from two or more centers which fuse during the fourth month according to others [4–7]. As fetal development progresses, the ossification of the parietal bones radiates in a uniform fashion except for a small area along the sagittal suture slightly in front of the lambda. There a slowing in the spread of ossification results in a notch in the posterosuperior aspect of each bone, causing a widening of the sagittal suture at that level. This notch normally disappears in the fifth fetal month, but vestiges may be observed postnatally in a number of bony defects including the parietal fissure, small and large parietal fontanelle, obeliac bones, persistent midline parietal foramen, and small and large parietal foramina.

Parietal Fissure or Incisura [4, 8, 9]

This is a thin cleft in one or both parietal bones at the obelion which extends outward from the sagittal suture for 1 cm or more. Le Double [8] found it in one of four newborn skulls. The defect is occasionally seen in routine skull roentgenograms where it may simulate a fracture (fig. 2). It probably disappears soon after birth.

Small Parietal Fontanelle [4, 5, 8, 10–14]

This is also referred to as the interparietal, sagittal, accessory, obeliac, or third fontanelle, or the fontanelle of Gerdy.

The defect presents as a round or diamond-shaped widening of the sagittal suture approximately 0.5 cm in size or slightly larger at the level of the obelion (fig. 3). It is usually midline but may indent only one parietal bone: the *unilateral sagittal fontanelle* [8]. It is observed in 5%–6% of newborns and possibly more frequently in infants with Down syndrome [11, 12, 14]. The defect usually disappears during the first 2–3 months of life but sometimes more slowly. How often it leads to small parietal foramina is not known.

Large Parietal Fontanelle

An occasional infant is born with a midline bony defect in the posterior interparietal region which is larger than the usual parietal fontanelle; sometimes it is huge. The larger defects are confluent with the posterior fontanelle and may be accompanied by bulging of the local soft tissues during crying. The anomaly, called here for convenience large parietal fontanelle, probably represents a true anomaly in the ossification of the parietal bone. As in the case of parietal foramina (see below), the differentiation between small and large parietal fontanelle is not clear-cut.

The fate of the defect varies. In most cases, the defect is the precursor of parietal foramina (see below), of varying size, possibly depending on the size of the original bony defect (figs. 4–6). In rare cases the defect may persist as a midline parietal or sagittal foramen (see below). Occasionally it becomes obliterated by a fontanelle bone, also referred to as an *obeliac bone* (fig. 7*A*), which is probably a type of Wormian bone similar to the "bregmatic bone" sometimes observed in the anterior fontanelle [15]. According to Le Double [8], an obeliac bone is found in about 0.8% of skulls. In some instances, two or more *Wormian bones* appear within the defect (fig. 7*B*).

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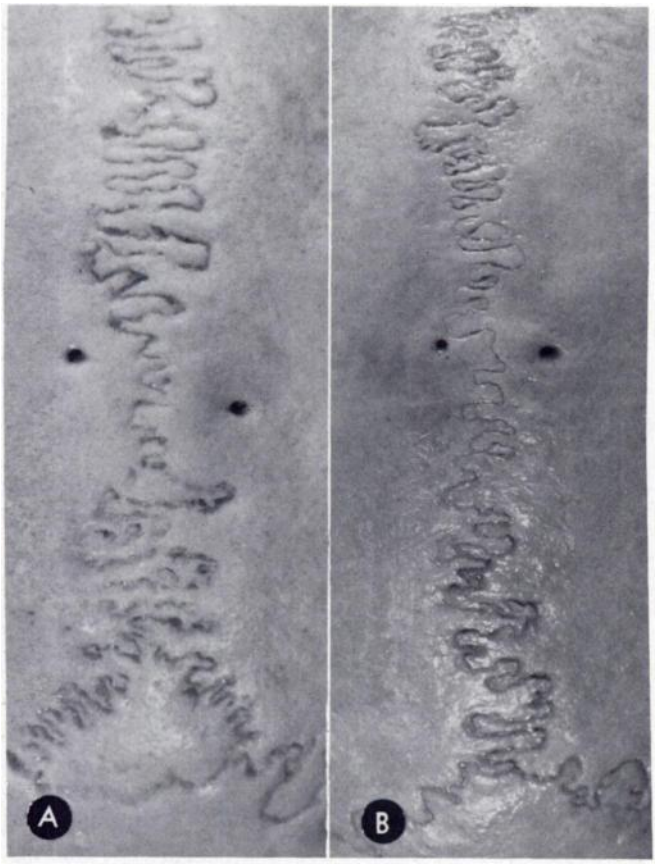

Fig. 1.—Photographs of two adult skulls with small parietal foramina. Metopic suture at level of foramina is of a more simple type than in front or more posteriorly.

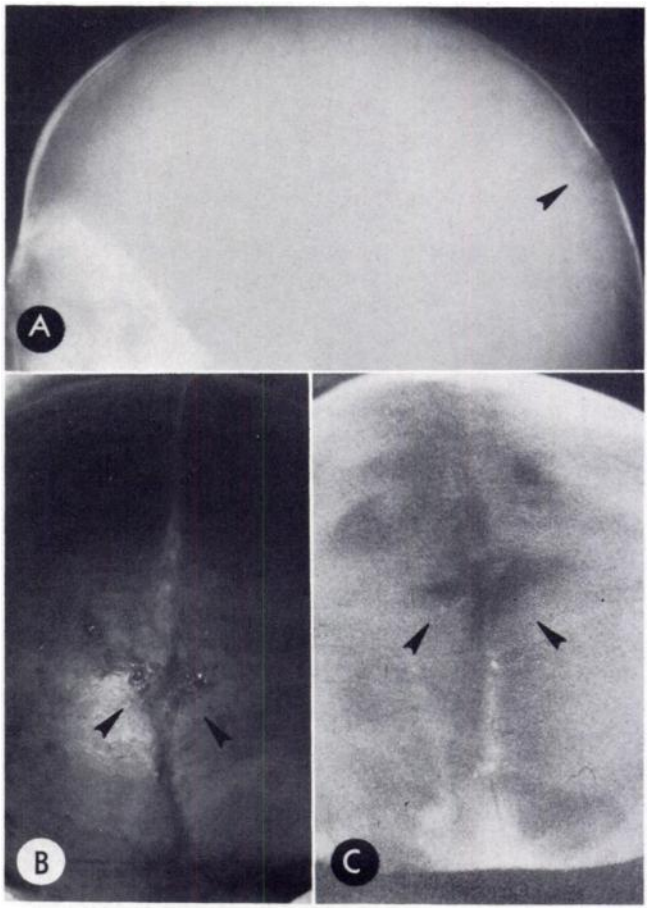

Fig. 3.—Small parietal fontanelle in newborn infant (*arrows*). *A*, lateral projection during life. *B*, Postmortem photograph of vertex of head with scalp and pericranium removed. Considerable amount of blood oozed from lateral aspects of posterior fontanelle at time of dissection. *C*, Postmortem roentgenogram of cranial vault.

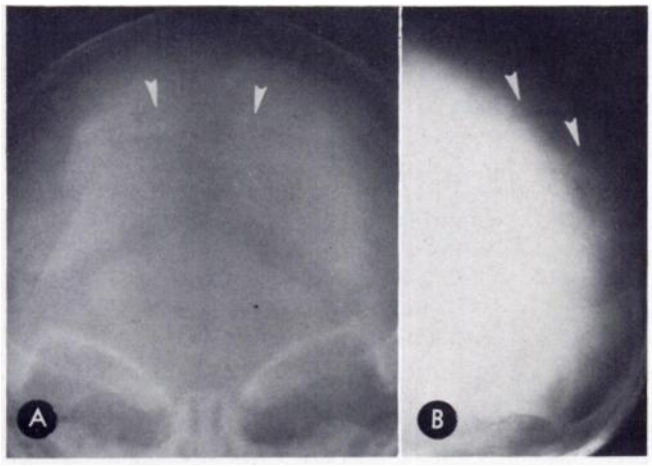

Fig. 2.—Frontal (*A*) and lateral (*B*) roentgenograms of skull of newborn infant with linear defect in posterosuperior aspect of each parietal bone presumably caused by parietal fissure or incisura (*arrows*).

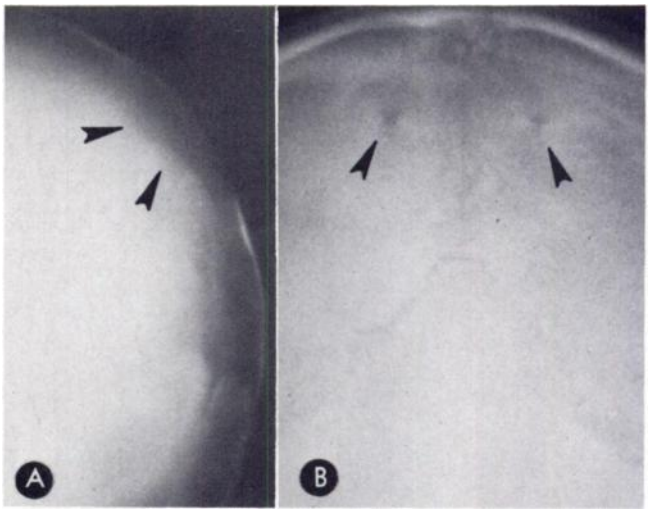

Fig. 4.—*A*, Lateral skull roentgenogram of 2-week-old infant with large parietal fontanelle (*arrows*). *B*, Frontal view of same patient 9 years later showing only small parietal foramina (*arrows*).

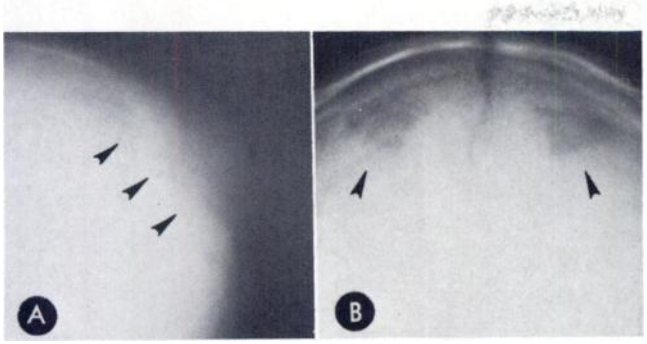

Fig. 5.—*A*, Lateral skull roentgenogram of 4-week-old infant with large posterior fontanelle and bulging of local soft tissues (*arrows*). *B*, Frontal view of same patient 4½ years later showing large parietal foramina (*arrows*)

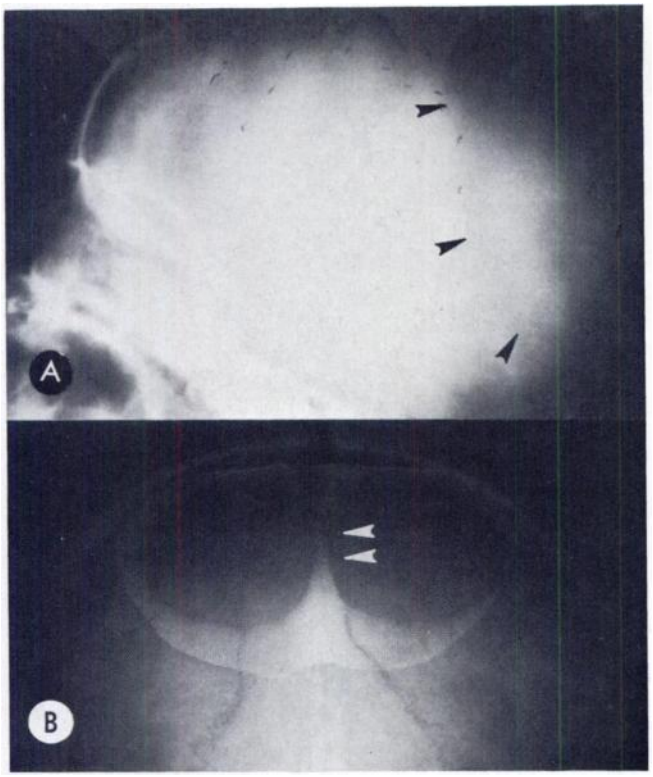

Fig. 6.—*A*, Lateral skull roentgenogram of newborn infant with large bony defect in posterior interparietal region and bulging of local soft tissues (*arrows*). At surgery 5 years later to cover defect with stainless steel mesh, galea aponeurotica found to be strongly adherent to defect with many arterial and venous bridging vessels. *B*, Frontal view of same patient at age 12 years showing very large parietal foramina. No appreciable change in bony defect since surgery except for appearance of strip of bone along sagittal suture (*arrows*).

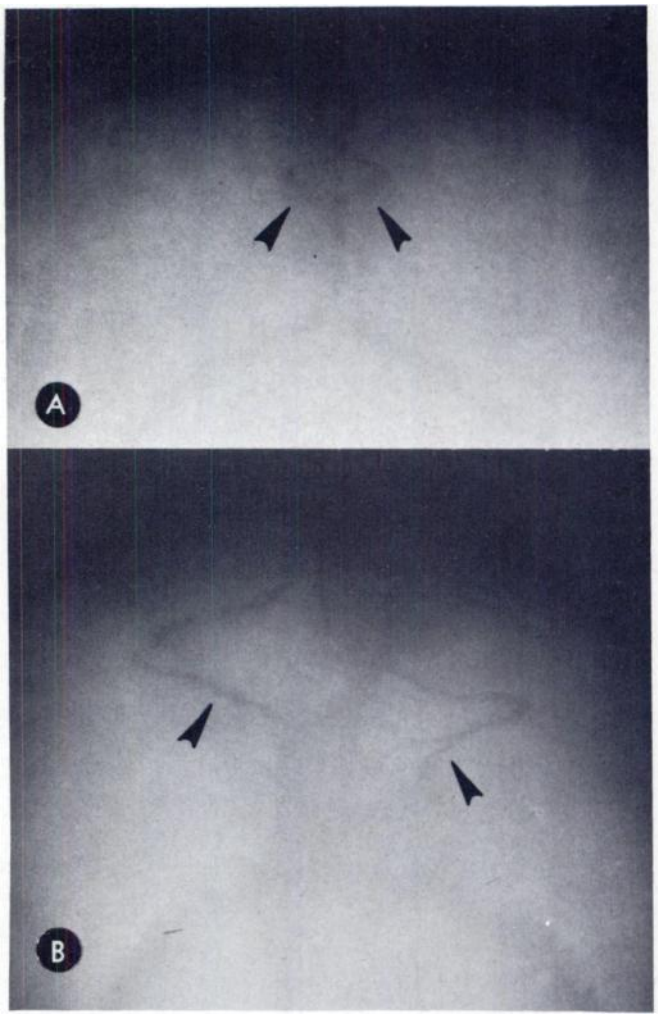

Fig. 7.—*A*, 7-month-old infant with obeliac or posterior fontanelle bone (*arrows*). *B*, 8-month-old infant with two large obeliac bones (*arrows*).

Persistent Midline Parietal Foramen

As already noted, a large parietal fontanelle may persist indefinitely as a large midline parietal or sagittal foramen (fig. 8). The anomaly is probably quite rare. A likely instance was reported by Caffey [16] and another by Zarfl [17].

Small Parietal Foramina [*4, 6, 8, 18–20*]

Also called emissaria parietalia or foramina Santorini, these are two minute round defects about 1 mm in size located at the level of the obelion. There is one on each parietal bone, usually within 1–2 cm of the sagittal suture but sometimes very close to it (fig. 9). Each foramen transmits an emissary vein, the vein of Santorini, which connects epicranial occipital veins with the superior sagittal sinus and sometimes also a very small artery anastomosing a branch of the occipital with a branch of the middle meningeal artery. These foramina probably represent a persistence of the most lateral aspects of the primitive parietal notch. They are said to occur in 60%–70% of adults and to be unilateral in almost half of the cases.

Small parietal foramina are not easily distinguishable in the newborn and are not always seen in routine skull roentgenograms of children and adults probably because of their small size and the x-ray projection. In frontal skull roentgenograms they sometimes appear like small areas of density of different shapes probably due to the projection of their bony walls rather than to an obliteration of the foramina (fig. 9*D*). It is not proven that once present they ever close spontaneously.

Large Parietal Foramina [*6, 17, 20–32*]

Other names include giant parietal foramina, foramina

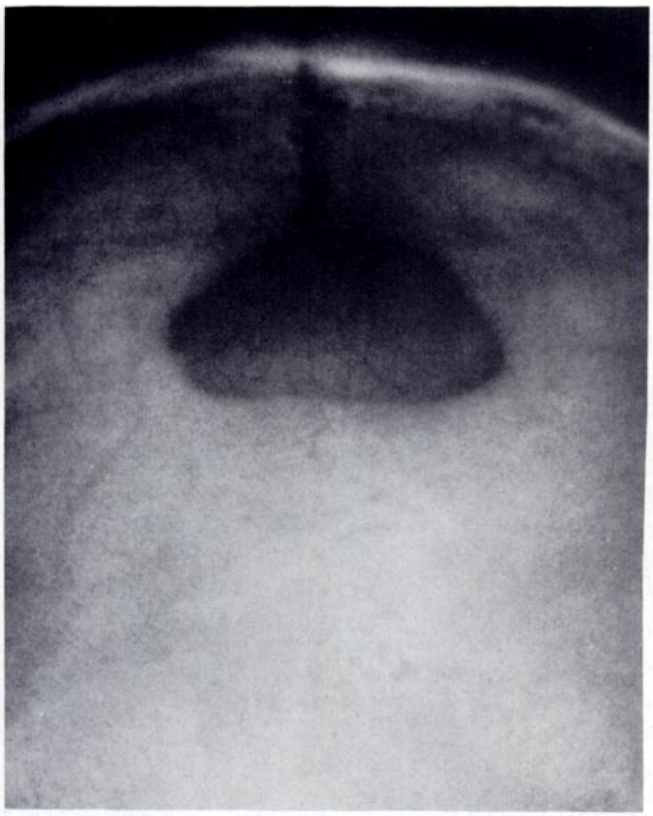

Fig. 8.—10½-year-old child with large interparietal foramen. Patient reportedly had large "soft spot" at vertex of head in infancy.

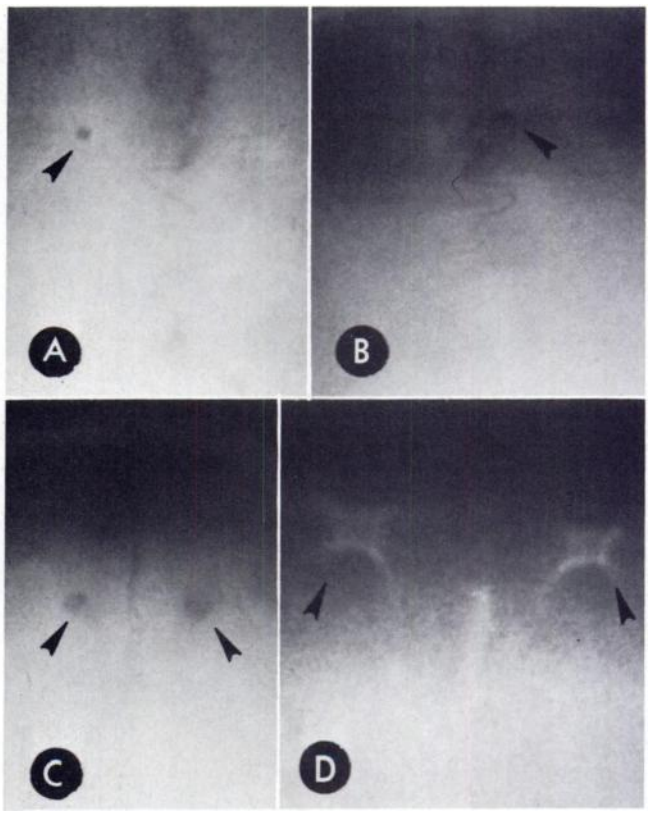

Fig. 9.—Frontal skull roentgenograms of four children aged 2, 5, 2, and 13 years respectively, with small parietal foramina (*arrows*). *A and B*, Unilateral; *C*, bilateral; *D*, bilateral parietal foramina causing starlike densities. Foramina appeared patent in lateral projection.

parietalia permagna, symmetrical parietal foramina, fenestrae parietales symmetricae, and "Catlin" mark from the name of the family with the anomaly reported by Goldsmith [33]. These are two symmetrical oval or rounded defects located one on each parietal bone, slightly anteriorly to the lambda and near the sagittal suture from which they are separated by a longitudinal strip of bone (figs. 5, 6, and 10). They correspond in location to the small parietal foramina but are much larger, varying from several millimeters to several centimeters in diameter. They are closed by a fibrous membrane which belongs both to the dura and the pericranium [18, 20]. The overlying scalp is normal, but rare cases have been reported in which it was the site of a congenital hairless defect [25, 34].

It seems well established that most, if not all, large parietal foramina are formed postnatally in the following manner. At birth, there is only one midline ossification defect involving both parietal bones (figs. 5 and 6). At times the defect is enormous and involves the entire vertex, simulating that seen in cleidocranial dysostosis (fig. 6*A*). Between the first and second year of life, small islands of ossification appear within the defect parasagittally. It is sometimes during this phase of development that one or both foramina are still united to the sagittal suture by a cleft or fissure (fig. 10*A*). Usually in the second or third year this parasagittal ossification becomes complete and the parietal defect becomes clearly double, probably without any further decrease in the size of the defects thereafter.

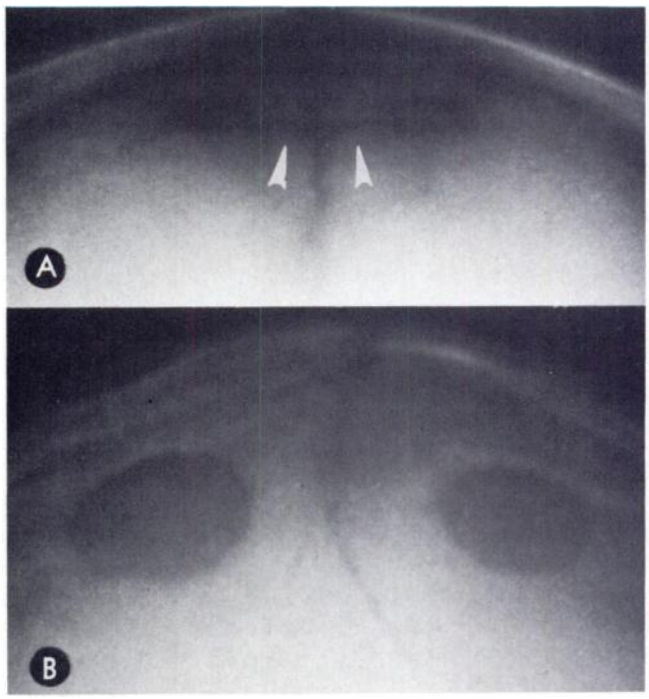

Fig. 10.—Frontal roentgenograms in two children aged 8 months (*A*) and 2½ years (*B*) with large parietal foramina. Foramina still bridged by a non-osseous stripe to sagittal suture in 8-month-old (*arrows*).

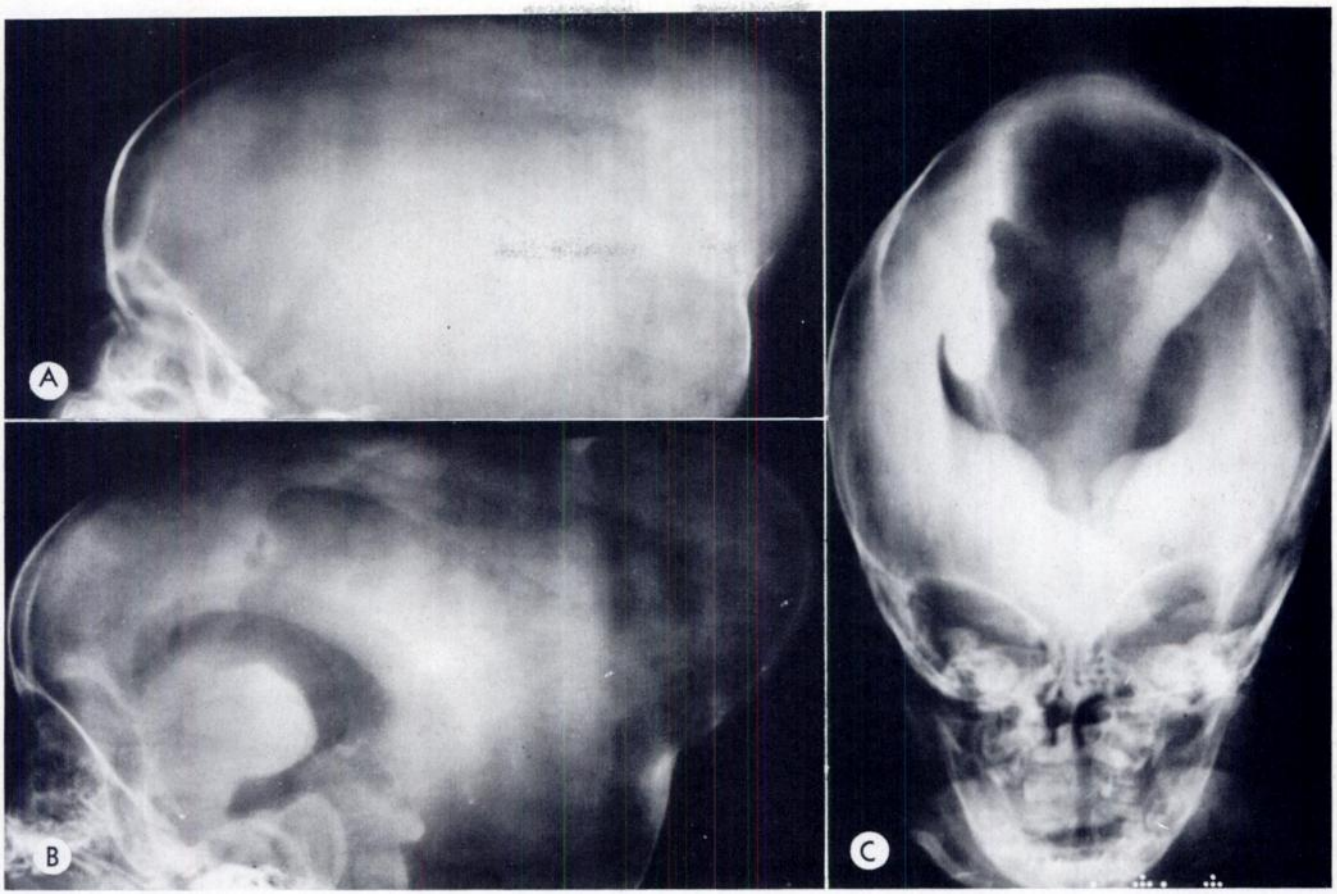

Fig. 11.—Large soft tissue mass in posterior interparietal region due to herniation of third ventricle (dorsal or diencephalic cyst) in newborn with absence of corpus callosum. *A*, Lateral skull roentgenogram. *B and C*, Ventriculogram, anterior and frontal projections. (Courtesy of F. N. Silverman)

Large parietal foramina are said to represent an anomaly of ossification of the parietal bone unrelated to small parietal foramina. Their cause is unknown. A familial incidence has been observed in a number of cases with a suggested dominant mode of inheritance. The defect may occur as an isolated lesion or, not uncommonly, in association with other malformations such as turricephaly [25, 35], craniofacial dysostosis [36] and other types of premature craniosynostosis, with asymmetry of the skull, microcephaly, eye and ear defects, mental retardation, convulsions, chest deformities, congenital vertebral anomalies, syndactyly, polydactyly, and many other anomalies [31]. It has also been observed with some frequency in the broad thumb syndrome of Rubenstein and Taybi [37]. Eckstein and Hoare [21] reported the anomaly in association with shortening of the lateral end of the clavicles in a mother and son, without other stigmata of cleidocranial dysostosis.

Encephaloceles and Related Anomalies

The region of the obelion is also the site of a number of uncommon neurocutaneous anomalies which are believed to be closely related developmentally. Some of these are true encephaloceles while others are probably remnants of encephaloceles or meningoceles which became pinched off at the level of the cranial vault during early intrauterine life. The presence of an underlying skull defect similar to a parietal fontanelle is the rule. These lesions include (1) true encephaloceles and herniations of intracranial midline cysts, (2) epicranial arachnoid cysts, and (3) heterotopic glial tissue.

True Encephaloceles

These occur most often in the occipital and nasofrontal regions and only occasionally in the nasopharynx or at the vertex of the head. Vertex encephaloceles are protrusions through a congenital skull defect of one or both hemispheres, with or without herniations of the ventricular system. As in other types of encephaloceles, the overlying scalp may be defective, meninges may or may not cover the hernia, and microcephaly and other brain deformities often coexist. Vertex encephaloceles are usually located near the anterior fontanelle and only rarely at the obelion. Two possible examples occurring at the obelion have been reported by Müller et al. [38].

Herniation of Midline Intracranial Cysts

A few cases of posterior interparietal "encephaloceles" have been described in which the anomaly was due to herniation of a diencephalic cyst or a Dandy Walker cyst. A *diencephalic cyst*, also called dorsal cyst, is a complicating feature of absence of the corpus callosum. It consists of a gross dilatation and marked upward extension of the third

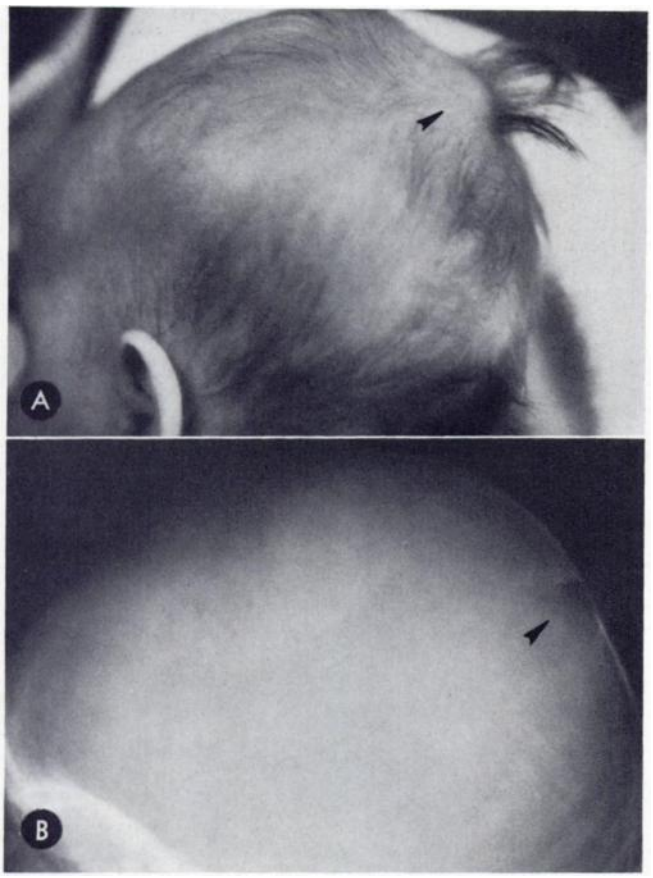

Fig. 12.—*A*, 2-month-old infant with congenital soft tissue mass in posterior interparietal region, with excessive growth of hair on mass (*arrow*). *B*, Lateral roentgenogram of same patient demonstrating underlying skull defect (*arrow*). A ventriculogram showed absence of the corpus callosum, without hydrocephalus or communication with the epicranial lesion. Diagnosis: epicranial arachnoid cyst.

ventricle between the two lateral ventricles. Brocklehurst [39] reported four cases. In two the cyst herniated through a skull defect at the obelion; in at least one it communicated freely with the surface lesion. A similar case observed by F. N. Silverman is illustrated in figure 11.

A *Dandy Walker cyst* represents a huge dilatation of the fourth ventricle associated with and possibly secondary to a congenital obliteration of the foramina of Luschka and Magendie. The cerebellum is reduced to two nubbings of brain tissue located one on each side of the cyst. The bifurcation of the superior sagittal sinus lies above the lambda, having failed to descend to its normal position in the midoccipital area during fetal life. The other ventricles are dilated. The corpus callosum is sometimes absent. In a case reported by McLaurin [40], the cystic fourth ventricle communicated with a cystic lesion at the vertex of the head just in front of the lambda.

Epicranial Arachnoid Cyst

This is probably the most common congenital mass lesion found at the obelion, a location which appears to be characteristic for the disorder. Typical cases have been reported by McLaurin [40] and by Müller et al. [38]. I have observed

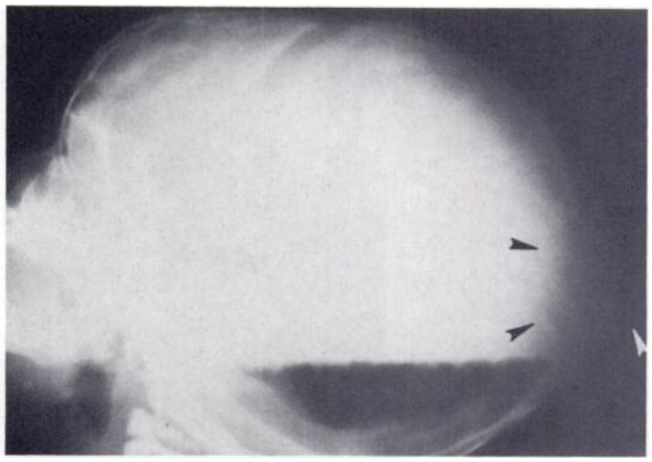

Fig. 13.—Ventriculogram (lateral projection with patient upside down) in newborn with craterlike mass at obelion associated with underlying skull defect (*arrows*). Nodule composed of glial tissue present in center of crater. Ventriculogram showed massive hydrocephalus but no communication with epicranial lesion.

two additional cases, one which is illustrated in figure 12. The following description is based on these cases plus the histologic observations by B. Landing (personal communication) on the patients reported by McLaurin [40].

The affected infant is born with a firm mass 1 cm or more in diameter located in the posterior interparietal region slightly in front of the lambda. Locally the scalp is intact but hair is usually excessive. A hemangioma on or around the mass may be observed. An underlying skull defect is present probably in all cases. The lesion is composed of arachnoid tissue cells surrounded by a fibrous wall. From this fibrous wall originates a solid or patent fibrous stalk which courses through the skull defect to blend with the dura, without a communication with deeper structures in most, if not all, cases. Epidermoid elements may be present histologically in rare cases. The central nervous system is usually normal, but absence of the corpus callosum has been observed. In a case referred to by McLaurin [40], this was complicated by a dorsal cyst.

Scalp Defect with Ectopic Glial Tissue

Lee and McLaurin [41] reported the case of an otherwise normal 1-year-old child who was born with a circular, hairless scalp lesion about 3 cm in diameter located in the midline of the head slightly anterior to the lambda. The lesion had a dry, flat epitheliumlike surface except for a nodular elevation near its center composed of glial tissue. At surgery this nodule was found to be connected by a stalk to the pericranium, apparently without an underlying skull defect.

I observed a similar case of a newborn with severe hydrocephalus probably caused by aqueductal atresia (fig. 13). Both lateral ventricles appeared to be in contact with the skull at the vertex of the head but did not communicate with the surface lesion. A midline bony defect was present at that level. The surface lesion consisted of a round area of hairless scalp 2 cm in diameter. Its periphery was elevated giving the lesion a craterlike appearance. In the center of the lesion was a nodular elevation composed of glial tissue.

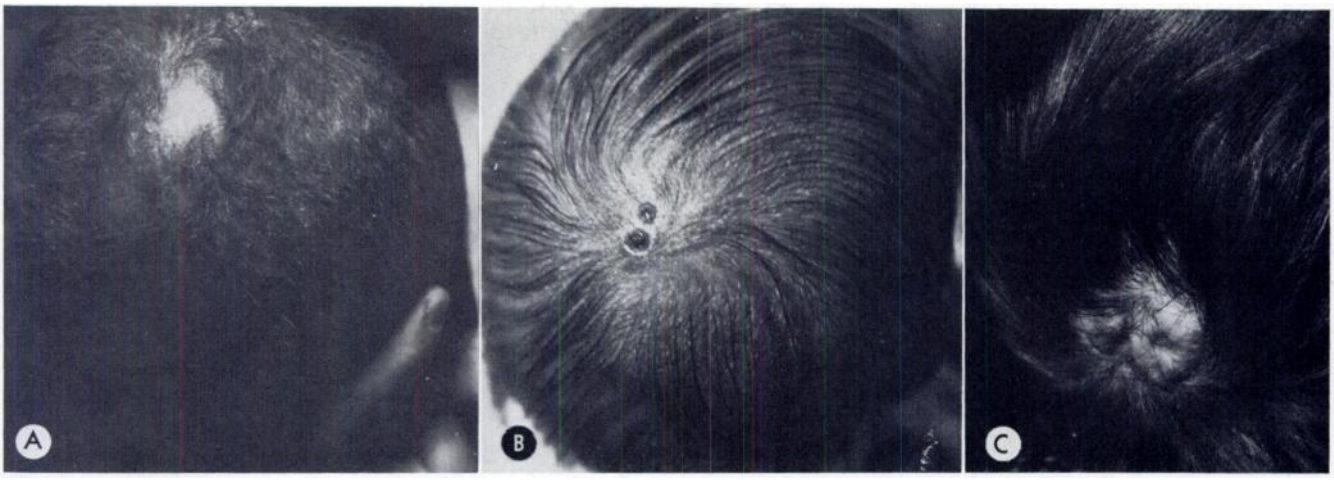

Fig. 14.—Congenital scalp defects at obelion. *A*, Flat, cicatricial lesion in 1-year-old child. *B*, Lesion with blister formations in newborn. *C*, Keloid-type lesion in 1-year-old child.

At surgery the lesion was found to be connected to the dura by a solid fibrous stalk.

Congenital Scalp and Skull Defects

The infant affected by this uncommon anomaly [42–48], also referred to as *cutis aplasia congenita*, is born with a sharply circumscribed area of hairless scalp 1 cm or more in diameter. In the vast majority of cases the defect is located at the vertex of the head just in front of the lambda (fig. 14). Its surface is most often shiny, cicatricial, and flat, but occasionally it is keloid in appearance [49]. In some cases one or more blisters are present, whereas in others there is a superficial ulceration with a serosanguinous exudate or fresh granulation tissue. These fresh lesions heal in a few weeks in a hairless depressed scar. Blister formation, rupture of the blisters, ulceration, and finally scar formation in utero is probably the mode of development of the most common atrophic form observed at birth. The main histologic features include absence of hair follicles, decreased elastic fibers, and an absence of smooth muscles, adipose tissue, subcutaneous glands, and other cutaneous adnexal structures.

According to O'Brien and Drake [46], in 18% of cases there is an underlying defect similar to a sagittal fontanelle without intracranial extension of the lesion. Underlying enlarged parietal foramina have been observed in two patients [25, 34]. The cranial defect is said to heal in a few months. In 5%–10% of cases, there is an area of absent skin in other parts of the body.

Associated malformations have included hydranencephaly, arrhyncephaly, hydrocephalus, tracheoesophageal fistula, polycystic kidney, cleft palate, microophthalmia, meningocele, and limb defects. The relatively high frequency of these additional defects may be due to the fact that the lesion occurs in a significant number of patients with trisomy 13–15 [50]. The lesion has also been observed in patients with a deleted short arm of chromosome 4 (46, XX, 4p−) [51] and in Noonan syndrome [52]. Familial cases are on record, and cases occurring in more than 1 generation or in identical twins have been reported [53–57]. The type of inheritance has been interpreted as dominant in some families and as autosomal recessive in others. Only histologic study can distinguish the lesion from the scalp defect with ectopic glial tissue.

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