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"False" foramina and fissures of the skull: a narrative review with clinical implications

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"False" foramina and fissures of the skull are described as openings formed between the adjacent edges of two or more bones and not conduits directly through a single bone. Trauma and metabolic disorders appear to affect these foramina and fissures differently when compared to the "true" foramina and fissures. Therefore, the aim of this paper is to provide a narrative review of the current literature about "false" foramina and fissures of the skull and skull base with a focus on their clinical significance. (Folia Morphol 2022; 81, 3: 551–558)

Key words: pseudo, foramina, fissures, skull base, calvaria

INTRODUCTION

The skull and skull base are the most complex bony structures in the human body. They must provide numerous passageways for arteries, veins, and nerves to enter and exit from the brain and surrounding intracranial structures. Many of the foramina that allow the passage of neurovascular structures are entirely contained within a single bone. The foramen rotundum and foramen spinosum located within the sphenoid bone are two examples [53]. However, additional "false" or pseudoforamina and fissures are openings formed between the adjacent edges of two or more bones. With similar logic, we have previously reviewed the so-called "false" ligaments that do not connect two bones together but rather begin and end on the same bone (e.g. suprascapular ligament) [50]. With similar logic, we here describe a "false" foramen as an opening or passage not within a single bone but

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rather created by the approximation of two nearby bones. An example of such a foramen is the jugular foramen, which is situated between the temporal and occipital bones. As most medical dictionaries define a foramen as an opening through "a bone", the present article identifies and provides a narrative review of the "false" foramina and fissures of the skull and skull base with a focus on their distinction from "true" foramina and their clinical significance. The term "false foramen" has been used by other authors [3, 7, 13]. However, the definition of false skull foramina has been loose, so that our review is the first attempt to establish a true definition and review the topic. Moreover, the notion of a false foramen does have significant clinical implications. Skull base fractures are more likely to injure structures traveling in a "true" skull base foramen than a "false" foramen as fractures are less likely to cross a foramen created between two adjacent bones. Therefore, the neurovascular structures contained within these passages are somewhat more protected. The present article identifies and provides a narrative review of the "false" foramina and fissures of the skull and skull base, focusing on their distinction from "true" foramina. The clinical significance of this distinction is also discussed.

REVIEW

Bony anatomy of the skull

The superior region of the skull, also known as the calvaria, is formed from parts of the frontal, occipital, and parietal bones [48]. The skull base or basicranium is formed from the inferior parts of those bones in addition to parts of the ethmoid, sphenoid, and temporal bones [44]. Inferior to the anterior region of the skull base, the facial skeleton or viscerocranium consists of 14 bones: the inferior nasal turbinate bones (2), lacrimal bones (2), mandible (1), maxillae (2), nasal conchae bones (2), palatine bones (2), vomer (1), and zygomatic bones (2) [30]. Most skull bones are joined together by fibrous sutures that normally close during the first through sixth decades of life [20, 32].

The skull base is typically divided into anterior, middle, and posterior parts for convenience of organization. The anterior skull base consists mainly of the frontal and ethmoid bones in addition to parts of the sphenoid bone, and its posterior borders are the lesser wings of the sphenoid [45]. The inferior depression known as the anterior cranial fossa houses the frontal lobes of the cerebral hemispheres and allows for the passage of cranial nerve (CN) I fibres through the cribriform plate foramina. The named foramina include the foramen cecum and the anterior and posterior ethmoidal foramina, which transmit the anterior and posterior ethmoidal arteries and nerves, and ethmoidal veins.

The middle cranial fossa is formed from the occipital, sphenoid, and temporal bones, the body of the sphenoid forming its anterior border and the basilar part of the occipital bone (anterior to the foramen magnum) its posterior border [29]. The middle cranial fossa contains many foramina and fissures: the optic foramen (canal), superior orbital fissure, foramen rotundum, foramen ovale, foramen spinosum, foramen lacerum, the hiatuses of the greater and lesser petrosal nerves, and the carotid canal. These constitute passages for CN II–VI or their major branches, the internal carotid artery (ICA), and numerous smaller arteries, nerves, and veins.

The posterior skull base is formed from the sphenoid, temporal, and occipital bones and houses the cerebellum, pons, and medulla oblongata. The anterior borders of the posterior cranial fossa are the dorsum sellae, parts of the body of the sphenoid, and parts of the basilar part of the occipital bone [44]. The foramen magnum is the most significant passageway in the posterior cranial fossa and transmits the medulla oblongata, which continues inferiorly as the spinal cord, and the spinal and vertebral arteries [49]. In addition, the spinal part of the accessory nerve traverses this foramen. Additional named passageways include the internal acoustic meatus, jugular foramen, and hypoglossal canal, which are passages for CN VII–XII and important vasculature, including the internal jugular vein [45]. Variant skull foramina include the orbitomeningeal, retromolar, and sphenoidal emissary foramina [27, 28, 31, 36].

False foramina and fissures of the anterior skull

"False" foramina and fissures can be found in all three fossae of the skull base. A complete list is given in Table 1.

The **foramen cecum** (Fig. 1) is a small notch formed at the junction between the frontal crest of the frontal bone and the ethmoid bone [45]. It normally closes via ossification during the first few years of life; however, foramen cecum patency has been reported in adults and can manifest as a nasoethmoidal encephalocele or dermoid cyst secondary

Table 1. Summary of calvarial and skull base false foramina and fissures

Skull location	Name	Surrounding bones	Contained structures
Anterior	Foramen cecum	Frontal, ethmoid	Emissary vein
	Sphenopalatine foramen	Sphenoid, palatine	Sphenopalatine artery and vein, posterior superior lateral nasal nerve, nasopalatine nerves
	Palatovaginal canal	Sphenoid, palatine	Pharyngeal branch of maxillary artery, pharyngeal nerve
	Vomerovaginal canal	Sphenoid, vomer	None
Middle	Foramen lacerum	Sphenoid, temporal, occipital	Meningeal branch of ascending pharyngeal artery, greater petrosal nerve, deep petrosal nerve, vidian nerve
	Superior orbital fissure	Sphenoid	Cranial nerve III–VI branches, ophthalmic veins, branches of middle meningeal and lacrimal arteries
	Inferior orbital fissure	Sphenoid, maxilla, zygomatic bone	Cranial nerve V branches, infraorbital artery
Posterior	Jugular foramen	Temporal, occipital	Inferior petrosal sinus, sigmoid sinus, cranial nerve IX–XI, meningeal branches of ascending pharyngeal artery and occipital arteries

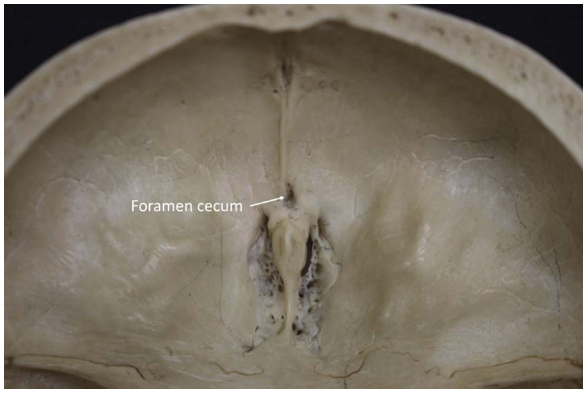

Figure 1. Superior view of the foramen cecum.

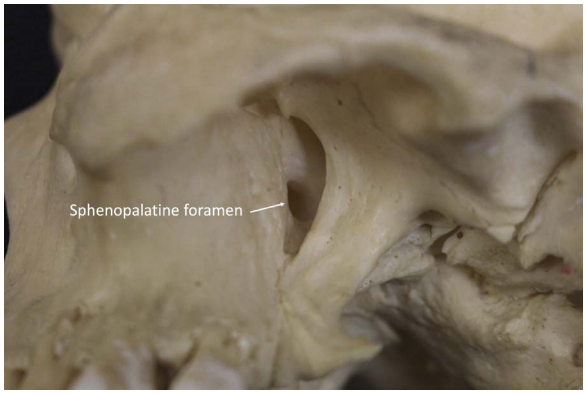

Figure 2. Inferolateral view of the left sphenopalatine foramen.

to dural extension of a dermoid sinus [2, 30]. It can transmit an emissary vein that runs from the nasal cavity to the superior sagittal sinus, thus paranasal sinus infections can spread to the intracranial cavity and cause meningitis and brain abscesses [45].

The **sphenopalatine foramen** (Fig. 2) is formed at the junction of the sphenopalatine notch of the palatine bone and the body of the sphenoid and leads from the pterygopalatine fossa to the nasal cavity [45]. While it is historically described as culminating in the posterior part of the middle nasal concha of the superior meatus, more recent anatomical studies have classified it on the basis of its openings to the superior and middle meatuses [52]. It transmits the sphenopalatine artery and vein, the posterior superior lateral nasal nerve, and the nasopalatine nerves [45]. The sphenopalatine foramen is clinically significant primarily in regard to epistaxis arising from the sphenopalatine artery, and anatomical variations can affect surgical planning and the outcome of procedures such as sphenopalatine artery ligation [41]. It is also used as a target for pterygopalatine ganglion aesthetic blockade [22].

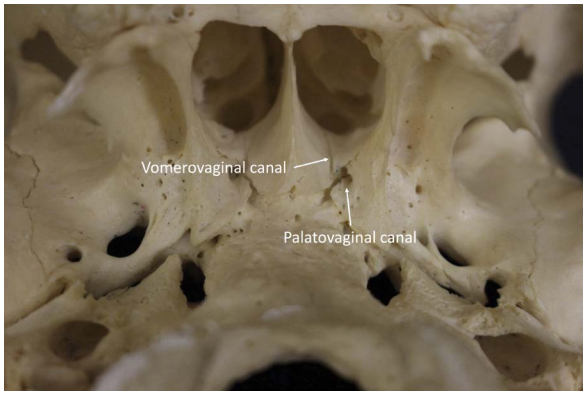

Figure 3. Inferior view of the left vomerovaginal and palatovaginal canals.

The **palatovaginal canal** (Fig. 3) is a passage from the pterygopalatine fossa to the roof of the pharynx

Figure 4. Superolateral view of the right foramen lacerum.

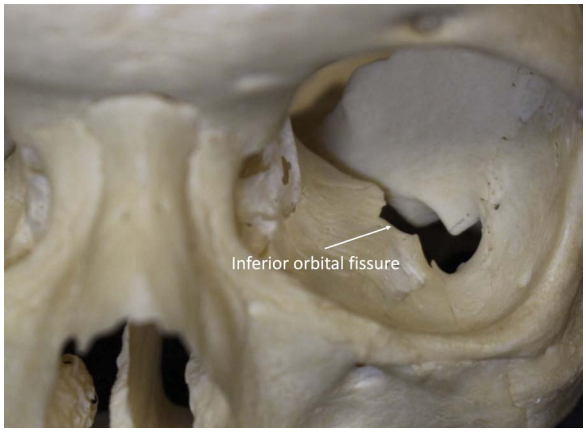

Figure 5. Anterior view of the left inferior orbital fissure.

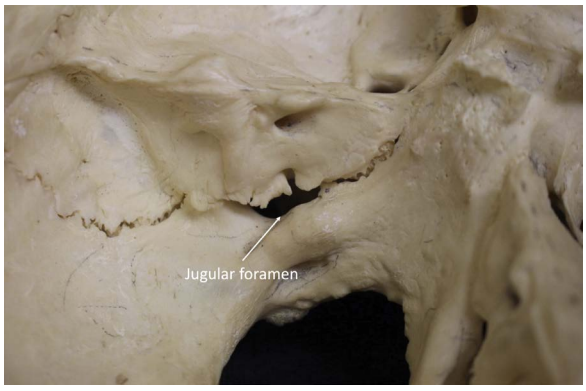

Figure 6. Superomedial view of the left jugular foramen.

located between the vaginal process from the medial plate of the pterygoid process of the sphenoid bone and the sphenoidal process of the palatine bone [45]. The major structures it contains are the pharyngeal branches of the maxillary artery and the maxillary division of CN V. Additionally, anatomical variants of the sphenopalatine vasculature can travel through the palatovaginal canal and complicate epistaxis treatment [40]. Neoplastic spread through the palatovaginal canal has also been reported in the literature [40].

Medially, the **vomerovaginal canal** (Fig. 3), if present, is located between the alae of the vomer and the vaginal processes of the sphenoid bone bilaterally. It leads into the anterior end of the palatovaginal canal [45]. It is an inconstant canal and no major structures pass through it [35].

False foramina and fissures of the middle skull

The triangular-shaped **foramen lacerum** (Fig. 4) is located posteromedial to the foramen ovale and is formed by the body and roots of the pterygoid process and the greater wing of the sphenoid bone anteriorly, the apex of the petrous part of the temporal bone posterolaterally, and the basilar part of the occipital bone medially [45]. A meningeal branch of the ascending pharyngeal artery, the vidian artery and nerve (formed within the foramen by the greater petrosal and deep petrosal nerves), and an emissary vein pass through it. The lacerum segment of the ICA traverses it superiorly but does not pass through it [32]. Normally, the inferior foramen lacerum fills with cartilage after birth. Partial occlusion has been reported and used to classify different foramen types, some of which necessitate alternative routes for structures that normally traverse the foramen such as the greater petrosal nerve [43]. The greater petrosal nerve, deep petrosal nerve, or vidian structures can also be compressed in cases of postnatal obliteration, leading to autonomic neuropathy. The foramen lacerum can provide a passageway for tumours to pass through the skull base, particularly if it is incompletely obliterated [38].

The **inferior orbital fissure** (Fig. 5) is located in the floor of the orbit and is formed from the greater wing of the sphenoid bone superiorly, the maxilla and orbital process of the palatine bone inferiorly, and the zygomatic bone laterally [45]. Branches of the maxillary division of CN V are the major structures that pass through it. It is an important landmark for endonasal endoscopic and orbital surgeries [10, 37]. The fissure ends at the infraorbital foramen on the face [42].

False foramina and fissures of the posterior cranial fossa

The irregularly-shaped **jugular foramen** (Fig. 6) is located at the posterior end of the petro-occipital suture and is formed anteriorly by the jugular fossa

of the petrous portion of the temporal bone and posteriorly by the jugular process of the occipital bone [15, 16, 45]. Historically, this foramen was divided into two compartments by the bony jugular spine: a pars nervosa that contained CN IX, the inferior petrosal sinus, and a meningeal branch of the ascending pharyngeal artery; and a pars venosa that contained the sigmoid sinus and CNs X and XI [24, 45]. Recent anatomical studies have yielded a three compartment classification: an anterior portion that transmits the inferior petrosal sinus, an intermediate portion that transmits CNs IX–XI, and a posterior portion that transmits the sigmoid sinus and meningeal branches of both the ascending pharyngeal and occipital arteries [24, 25]. These compartments are separated by dural septa that connect opposing intrajugular processes of the temporal and occipital bones [29]. The jugular foramen is a common site of skull base tumours including paragangliomas (globus jugulare tumours), schwannomas, and meningiomas [13].

Developmental differences and similarities

The skull is a developmentally complex structure to which both the neural crest and mesoderm contribute. It undergoes both intramembranous and endochondral ossification [31]. The palatine and maxillary bones are neural crest-derived intramembranous bones, and the occipital bone is a mesodermal endochondral bone [33]. Therefore, both bone ossification patterns form "false" foramina: the infraorbital fissure is formed from the maxillary bone and the jugular foramen from the occipital bone. The various ossification patterns of the foramen lacerum are possible owing to its formation within the endochondral bones [43]. Likewise, both ossification patterns form "true" foramina such as the palatine foramina (within the palatine bone) and hypoglossal canal (within the occipital bone).

Studies on chick embryo models examining the development of the jugular foramen and hypoglossal canal have shown that mesenchymal tissue first surrounds the contained blood vessel [33]. The mesenchyme is less dense than surrounding mesenchyme, which forms the cartilage of the non-foraminal bone. Next, the mesenchymal cells adjacent to the blood vessel and nerves change morphology to resemble perichondral cells. At this stage, the jugular "false" foramen appears different from the hypoglossal "true" foramen in that its shape conforms better to the contained blood vessel. However, no studies have examined the clinical significance of this difference. It is currently under debate whether blood vessels, nerves, or both are primarily responsible for directing the formation of foramina, and further studies could provide more information on developmental differences between "true" and "false" foramina. A better understanding of this embryology might lead to an improved understanding of patients who are prone to developing stenosis of various skull base foramina such as patients with achondroplasia and stenosis or narrowing of the jugular foramen.

Skull fractures involving foramina and fissures

Traumatic skull base injuries can pose significant challenges to surgeons because of their complex anatomy and symptomatology. The orbit is a common place for fractures owing to its relative weakness [25]. Fractures of the superior orbital fissure can result in superior orbital fissure syndrome [54]. Typical symptoms include diplopia, extraocular muscle weakness, exophthalmos, and ptosis [54]. Crush injury and fracture of the petrous part of the temporal bone can lead to avulsion of the petrous part of the temporal bone backwards with inward rotation of the apex and can cause severe distortion of the foramen lacerum [23]. This can damage nearby structures such as the ICA. Additionally, CN VI can be stretched, so patients with fractures in this region typically present with abducens nerve palsy [26].

Fractures of the posterior skull base are less common but identifiable when patients present with deficits in multiple cranial nerves [17]. Jugular foramen fractures presenting with lower cranial nerve deficits have been reported in the setting of occipital bone fracture with subsequent direct nerve injury or delayed ischaemia-related oedema [1, 47]. Rarely, severe trauma to the occiput can cause a fracture that courses from the jugular foramen, across the petrous part of the temporal bone, and through the foramina spinosum to the foramen lacerum and the foramen magnum [25]. However, skull base fractures are more likely to injure structures travelling in a "true" skull base foramen than a "false" foramen as fractures are unlikely to cross a foramen created between two adjacent bones unless there is significant blunt trauma to the region that affects multiple bones. Therefore, the neurovascular structures contained within these passages are somewhat more protected.

Severe basilar fractures can involve these "false" foramina because unions of multiple bones are inher-

ently weak points in the skull base [51]. Moreover, most cranial nerves are carried by these foramina, so fractures of the "true" foramina could be underreported because the resulting neurological deficits are inconspicuous. Hypothetically, the true foramina might offer additional protection to the nerves and vessels that traverse them and future studies might address this difference.

Disorders of bone and cartilage

Paget's disease (PD) is a chronic bone disorder characterised by increased cellular turnover that can involve bones of the skull [8]. PD of the skull frequently results in areas of localised or diffuse thickening and interspersed areas of osteoporotic lesions [39]. The most common neurological symptom is hearing loss, which is thought to be due to loss of bone mineral density in the cochlear capsule, not to internal acoustic meatus narrowing (stenosis) secondary to sclerosis [34]. To the best of our knowledge, there are no reported cases of "false" foramina narrowing in the setting of PD of the skull. However, optic neuropathy due to optic canal sclerosis has been reported [12].

Fibrous dysplasia is another chronic bone disorder that often affects craniofacial bones including the ethmoid, sphenoid, frontal, and temporal bones [19]. It is thought to involve abnormal growth and differentiation of marrow stromal cells resulting in abundant expansile fibrous tissue deposition in place of normal bone, causing structural defects [19]. In contrast to PD, fibrous dysplasia causes conductive hearing loss due to external acoustic meatus stenosis [6]. Narrowing of the optic canal and jugular foramen has also been reported [4, 46]. While there have been no studies directly examining the histopathology of PD or fibrous dysplasia in cranial foramina, differences in which foramina are affected could be due to pathophysiology instead of differences between "true" and "false" foramina. However, multiple bones provide multiple sites for the initiation of bony expansion; the jugular foramen can become stenotic from either temporal or occipital bone overgrowth, while the hypoglossal canal must become stenotic from occipital bone overgrowth.

As both intramembranous and endochondral ossification are involved in the formation of both "true" and "false" foramina, it is unlikely that cartilaginous disorders would preferentially affect one type of foramen. In one such disorder, achondroplasia, cartilage cannot be fully converted to bone and the foramen magnum is commonly stenotic at birth. The narrowing of the jugular foramen increases intracranial venous pressures and in turn alters the cerebrospinal fluid dynamics, frequently resulting in hydrocephalus in this particular patient population. Notably, studies suggest that this stenosis results partly from premature closure of the posterior intraoccipital synchondroses [18]. Abnormalities in other skull base synchondroses could in principle affect the "false" foramina formed from them [5]. For example, the spheno-occipital synchondrosis, which forms part of the foramen lacerum border, can close prematurely, preventing normal foramen expansion and causing constriction of neurovascular structures [11].

CONCLUSIONS

In this review we have explored all the cranial foramina and fissures that are formed from parts of more than one bone. These "false" foramina share similarities with the "true" neurovascular passageways formed within a single bone, but also differ from them. Future study on the developmental differences between the two foramen types is warranted in order to elucidate their clinical significance further. Additionally, how anatomical variations and various mechanisms for skull base fracture affect the true versus false skull base foramina should be studied in patient cohorts [9, 14, 41].

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