Current Concepts in Pediatric Temporomandibular Joint Disorders: Part 1. Etiology, Epidemiology, and Classification

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Background: Pediatric temporomandibular joint dysfunction, resulting from either soft-tissue or skeletal disorders, may be congenital or acquired. Congenital temporomandibular joint disorders are uncommon. The authors review their experience with pediatric temporomandibular joint disorders and propose a new classification system.

Methods: Clinical records, cephalograms, computed tomographic scans, magnetic resonance images, and pathologic specimens of all pediatric patients (younger than 18 years) with trismus or restricted mandibular excursion from 1976 to 2008 were reviewed. Cases were stratified according to soft-tissue or skeletal pathologic findings; skeletal abnormalities were further characterized as intracapsular or extracapsular.

Results: Thirty-eight patients, ranging in age from 1 day to 18 years at diagnosis, were identified with temporomandibular joint disorders. Ten cases (26.3 percent) were attributable to soft-tissue abnormality. The remaining 28 cases (73.7 percent) were attributable to skeletal abnormality, consisting of 14 congenital and 14 acquired cases (50 percent each). Acquired skeletal deformities included 12 intracapsular ankyloses (85.7 percent) and two extracapsular ankylosis (14.3 percent) (extraarticular bone blocks). Congenital skeletal deformities accounted for five intracapsular ankyloses (35.7 percent) and nine extracapsular ankyloses (64.3 percent).

Conclusions: On initial survey, the data are consistent with published reports that attribute temporomandibular joint dysfunction to acquired abnormality (i.e., trauma and infection). However, the authors observed a significantly higher percentage (50 percent) of congenital temporomandibular joint skeletal disorders than previously reported. Most congenital cases involved extracapsular abnormality (i.e., coronoid hypertrophy); only a minority of cases had glenoid-condylar fibro-osseous fusion (i.e., intracapsular ankyloses). Because the diagnosis and classification of temporomandibular joint disorders determine treatment options, the authors provide a new classification that characterizes the extent of capsular involvement. (*Plast. Reconstr. Surg.* 126: 1263, 2010.)

Pediatric temporomandibular joint dysfunction may result from either soft-tissue or skeletal disorders and may be congenital or acquired. Symptoms and signs vary according to cause and severity, but typically include trismus, reduced interincisal opening, and pain with forced excursion. Temporomandibular joint ankylosis—bony or fibrous union between the articular surfaces of the joint—is relatively rare, but can be extremely morbid and disabling. Although it can occur in any age group, children are at higher

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risk because of anatomical and physiologic differences at the joint (Fig. 1). Functional sequelae include limited mandibular range of motion, inability to form an oral seal, interference with mastication, and difficulty with nutrition and oral hygiene. Furthermore, ankylosis in children may result in underdevelopment of the mandible and aberrant facial growth.

Historic studies attribute adult and pediatric temporomandibular joint disorders mainly to acquired abnormality, specifically, trauma and infection. In Part 1 of this series, we present our experience at the Institute of Reconstructive Plastic Surgery. In contradistinction to prior published reports, we observed a significantly higher percentage of congenital cases of pediatric temporomandibular joint skeletal disorders. We provide a review of the relevant literature and present a detailed discussion of the anatomical basis, epidemiology, cause, and sequelae of temporomandibular joint dysfunction. Because precise diagnosis and classification direct the choice of therapy, we propose a classification to differentiate between soft-tissue and skeletal pathologic findings and to better define the extent of capsular involvement. Part 2 of this series continues with detailed discussion of the available treatment modalities.

PATIENTS AND METHODS

An institutional review board–exempt (no. 06-549) retrospective review was conducted of all pediatric patients (younger than 18 years) with trismus or restricted mandibular excursion treated at the Institute of Reconstructive Plastic Surgery between 1976 and 2008. Clinical records, cephalograms, computed tomographic scans, magnetic resonance images, and pathologic specimen reports were reviewed. Cases were stratified according to soft-tissue and/or skeletal pathologic findings. Skeletal abnormalities were further characterized as intracapsular or extracapsular based on radiographic findings or intraoperative observations.

RESULTS

Thirty-eight patients (ranging in age from 1 day to 18 years at diagnosis, with a mean age of 5 years) were identified with temporomandibular joint dysfunction (Table 1). Ten cases (26.3 percent) were caused by soft-tissue disease and 28 cases (73.7 percent) were skeletal in origin (Fig. 2). Irrespective of anatomical classification, the acquired cases accounted for 24 of the total cases (63.2 percent) and were predominantly caused by trauma [10 of the 38 total cases (26.3 percent)] and infection [seven cases (18.4 percent)]. Con-

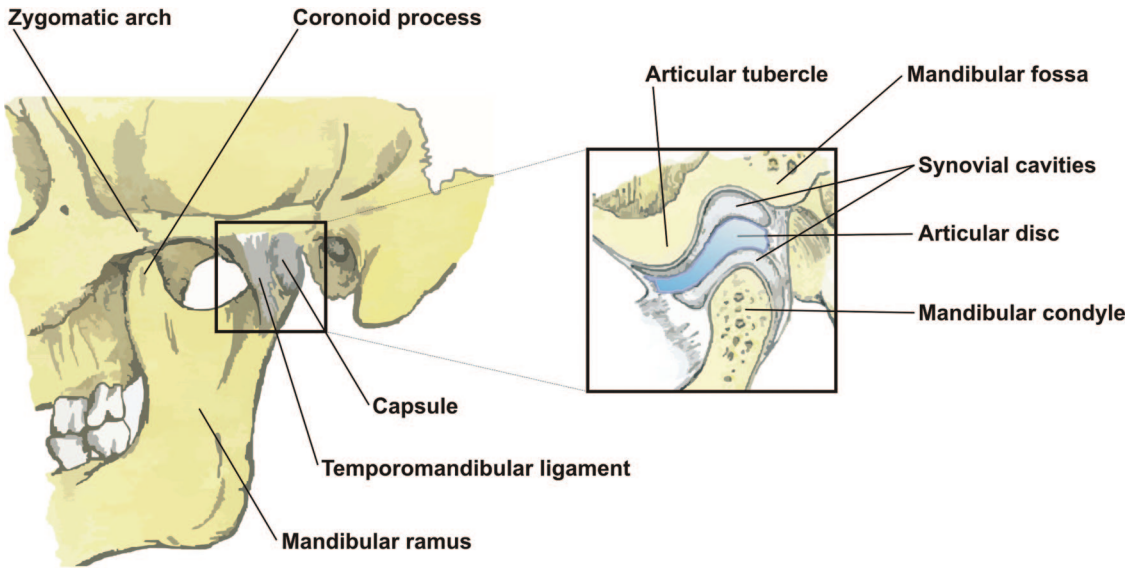

Fig. 1. The temporomandibular joint has two synovial cavities divided by an articular disk, allowing for both rotation and gliding motions between the mandibular condyle and the cranial base. The pediatric mandible is characterized by a broad condyle and thinner cortical bone, anatomical features that predispose to intracapsular comminuted fractures; the elongated condylar neck in adults limits fractures to the extracapsular space.

Table 1. Patient Demographics and Case Summaries

Patient	Sex	Age*	Laterality	Cause	Resulting Pathologic Findings	Classification	
						Region	Subtype
Congenital							
1	M	1 yr	Bilateral	Congenital ankylosis	Ankylosis of TMJ	Skeletal	Intracapsular
2	F	1 yr	Unilateral	Congenital ankylosis	Ankylosis of TMJ	Skeletal	Intracapsular
3	F	2 days	Bilateral	Congenital ankylosis	Ankylosis of TMJ	Skeletal	Intracapsular
4	M	4 yr	Bilateral	Congenital ankylosis	Ankylosis of TMJ	Skeletal	Intracapsular
5	F	1 day	Bilateral	Congenital ankylosis	Ankylosis of TMJ	Skeletal	Intracapsular
6	F	5 yr	Bilateral	Coronoid hypertrophy	Ankylosis of coronoid to zygoma	Skeletal	Extracapsular
7	M	2 yr	Unilateral	Coronoid hypertrophy	Ankylosis of coronoid to zygoma and skull base	Skeletal	Extracapsular
8	F	3 mo	Bilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
9	F	1 mo	Unilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
10	F	4 yr	Bilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
11	M	2 yr	Bilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
12	M	1 yr	Bilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
13	M	3 yr	Bilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
14	M	6 yr	Bilateral	Coronoid hypertrophy	Mechanical interference	Skeletal	Extracapsular
Acquired							
15	M	2 days	Bilateral	Neonatal infection	Ankylosis of TMJ	Skeletal	Intracapsular
16	F	10 yr	Unilateral	Chronic otitis media	Ankylosis of TMJ	Skeletal	Intracapsular
17	F	4 yr	Bilateral	Chronic otitis media	Ankylosis of TMJ	Skeletal	Intracapsular
18	F	9 mo	Unilateral	Parotid gland infection	Ankylosis of TMJ	Skeletal	Intracapsular
19	M	10 yr	Unilateral	Postsurgical; infected hardware	Ankylosis of TMJ	Skeletal	Intracapsular
20	F	6 yr	Unilateral	Postsurgical	Ankylosis of TMJ	Skeletal	Intracapsular
21	F	3 yr	Unilateral	Postsurgical	Ankylosis of TMJ	Skeletal	Intracapsular
22	F	8 yr	Unilateral	Postsurgical	Ankylosis of TMJ	Skeletal	Intracapsular
23	F	4 yr	Unilateral	Postsurgical relative coronoid hypertrophy (acquired short ramus)	Mechanical interference	Skeletal	Extracapsular
24	M	3 yr	Unilateral	Trauma	Ankylosis of TMJ	Skeletal	Intracapsular
25	F	10 yr	Bilateral	Recurrent dislocation	Joint deformity	Skeletal	Intracapsular
26	F	18 yr	Bilateral	Rheumatoid arthritis	Severely deformed temporomandibular joints	Skeletal	Intracapsular
27	F	3 yr	Bilateral	Secondary to other defect	Periarticular bone block	Skeletal	Extracapsular
28	F	7 yr	Unilateral	Mandibular teratoma	Invasion of jaw musculature; joint dislocation	Soft tissue	—
29	M	3 yr	Bilateral	Chronic otitis media	Pericapsular fibrosis	Soft tissue	—
30	F	1 yr	Bilateral	Neonatal infection	Pericapsular fibrosis	Soft tissue	—
31	M	5 mo	Unilateral	*Staphylococcus* infection	Pericapsular fibrosis	Soft tissue	—
32	F	11 yr	Unilateral	Embolization of vascular malformation	Pericapsular fibrosis	Soft tissue	—
33	F	13 yr	Unilateral	Radiation therapy	Pericapsular fibrosis	Soft tissue	—
34	F	10 yr	Unilateral	Oral teratoma	Contracture	Soft tissue	—
35	M	10 yr	Bilateral	Postsurgical	Contracture	Soft tissue	—
36	F	18 yr	Bilateral	Postsurgical	Contracture	Soft tissue	—
37	M	2 yr	Bilateral	Postsurgical	Contracture	Soft tissue	—
38	M	3 yr	Bilateral	Disuse atrophy	Contracture	Soft tissue	—

TMJ, temporomandibular joint.
*Age at the time of onset of TMJ disorder.

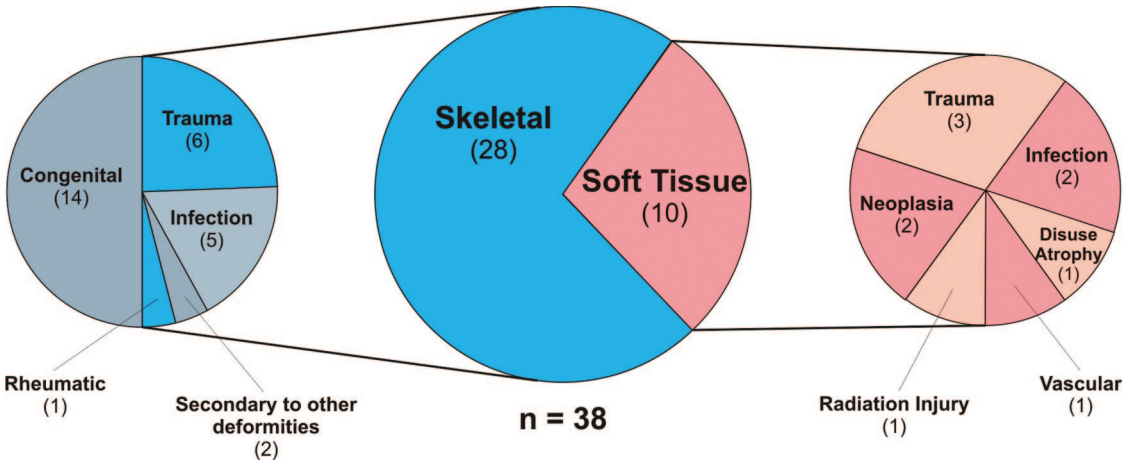

Fig. 2. Stratification of all cases of temporomandibular joint disorders according to location of disease (skeletal versus soft tissue), with substratification according to specific cause.

genital cases accounted for temporomandibular joint disorders in 14 cases (36.8 percent).

Soft-tissue temporomandibular joint dysfunction was attributable exclusively to acquired abnormality. Causes included scarring secondary to trauma [three cases (30 percent)], soft-tissue infection [two cases (20 percent)], tumor [two cases (20 percent)], radiation injury [one case (10 percent)], embolization therapy causing ischemic injury [one case (10 percent)], and disuse atrophy [one case (10 percent)].

Patients with skeletal temporomandibular joint dysfunction consisted of 14 congenital cases (50 percent) and 14 acquired cases (50 percent). With regard to anatomical classification, 11 skeletal cases (39.3 percent) were extracapsular and 17 (60.7 percent) were intracapsular (Fig. 3).

Acquired skeletal deformities included 12 intracapsular ankyloses (85.7 percent of acquired skeletal cases). These were secondary to trauma [six cases (50 percent)], infection [five cases (41.7 percent)], and degenerative rheumatic disorders [one case (8.3 percent)]. Two acquired skeletal cases (14.3 percent) were extracapsular ankyloses caused by coronoid process–zygoma fusion and extracapsular bone blocks. Congenital skeletal deformities included five intracapsular ankyloses (35.7 percent) and nine extracapsular ankyloses (64.3 percent) or mechanically restrictive skeletal disorders (e.g., coronoid hypertrophy without ankylosis).

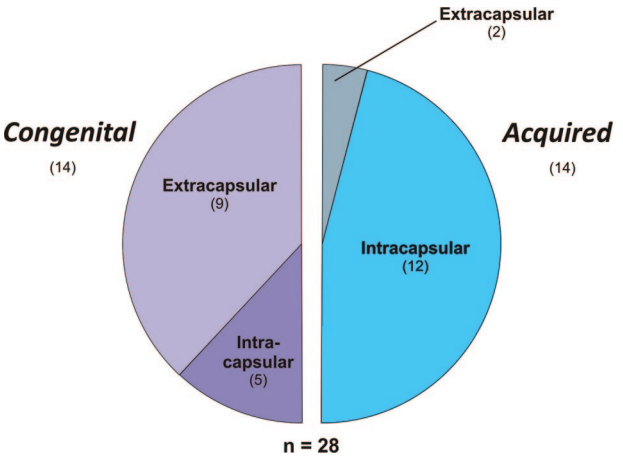

Fig. 3. Congenital and acquired skeletal cases of temporomandibular joint disorders, categorized anatomically as involving intracapsular or extracapsular abnormality. Most congenital disorders involve extracapsular abnormality (e.g., coronoid hypertrophy or zygomatic hypoplasia), whereas most acquired disorders directly involve the joint itself.

CASE REPORTS

Case 1: Acquired (Trauma)/Intracapsular

A healthy 3-year-old boy (patient 24) tripped while playing and sustained a comminuted fracture of the left mandibular condyle; the fracture was not treated. At 4 years of age, he complained of increasing trismus and difficulty in eating and speaking. On presentation, the patient had an interincisal opening of only 4 mm, with chin deviation toward the left (i.e., affected) side. Computed tomographic scan confirmed bony ankylosis of the left temporomandibular joint (Fig. 4). Furthermore, the traumatic comminution of the joint had reduced the height of the mandibular condyle such that the coronoid process was relatively hypertrophic. The patient was treated with gap arthroplasty with coronoidectomy and aggressive physiotherapy. Five months after surgery, the patient's interincisal opening was 32 mm.

Case 2: Acquired (Infection)/Intracapsular

A 22-month-old Portuguese infant (patient 18) was referred because of limited interincisal opening (Fig. 5). At 10 months of age, the patient had evidence of abnormal facial growth, chin deviation to the left, low-set left ear, limited mandibular excursion (6 mm), and snoring. Hospital records revealed a neo-

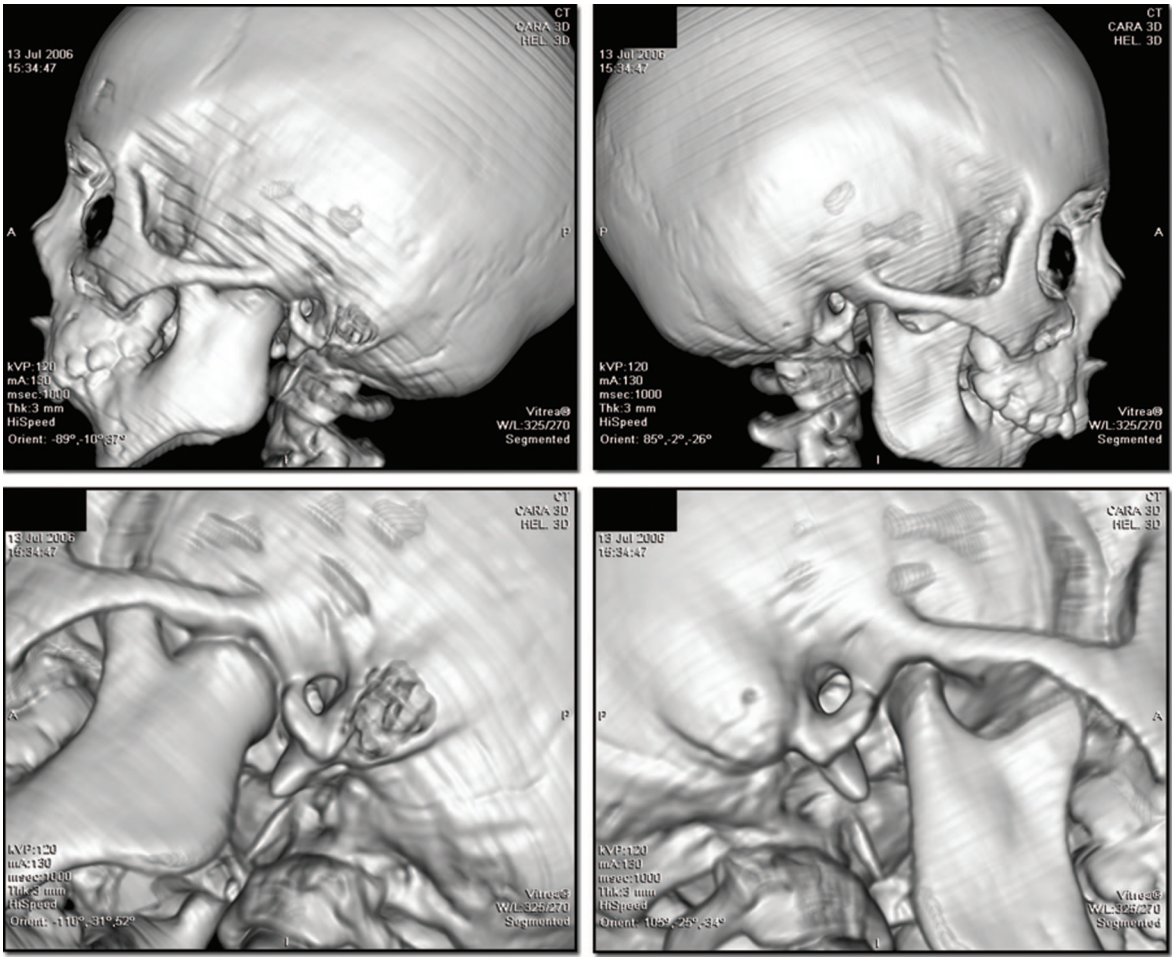

Fig. 4. Case 1. Three-dimensional reconstructed computed tomographic scan of a boy (patient 24) who developed left temporomandibular joint ankylosis following trauma at 3 years of age. Findings include a shortened, widened condyle that is fused to the cranial base. The left coronoid process is hypertrophic. In contrast, the right temporomandibular joint is normal.

natal history of acute, localized left parotid infection during the first month of life, with an elevated white blood cell count of 25,000 (with 70 percent neutrophils) and C-reactive protein, although blood cultures remained negative. She was treated empirically with ampicillin and gentamicin for 2 weeks until normalization of her examination findings and laboratory values. However, the parotid gland infection caused a left septic arthritis that resulted in craniomandibular dysplasia and micrognathia. Computed tomography demonstrated left temporomandibular joint ankylosis, with complete bony fusion of the mandibular condyle to the cranial base. The patient was treated with ipsilateral condylectomy (gap arthroplasty), reverse-L mandibular osteotomy, transport distraction osteogenesis, and coronoidectomy, followed by physiotherapy. At 2-year follow-up, she had an interincisal opening of 24 mm.

Case 3: Congenital/Intracapsular

A newborn girl (patient 5) was delivered prematurely at 30 weeks' gestation by means of caesarean section. She weighed 1300 g and was atonic at birth, with Apgar scores of 4 and 6. Because of limited mandibular excursion, she was not able to be intubated and required emergent tracheostomy. The first few months of life were complicated by the sequelae of prematurity: retinopathy, intraventricular hemorrhage, bronchopulmonary dysplasia, and respiratory syncytial virus pneumonia.

At 9 months of age, she was referred for evaluation. She was noted to be at the 25th percentile for weight and the fifth percentile for head circumference. The orbits were asymmetrical, and although midface projection was normal, the chin was underdeveloped. Mandibular excursion was limited to an interincisal opening of only 4 mm. Craniofacial computed tomography confirmed bilateral bony ankylosis without coronoid hypertrophy (Fig. 6). Surgical treatment is planned for age 3.

Case 4: Congenital/Extracapsular

A 2-week-old girl (patient 9) was born with Hecht syndrome and maxillomandibular fusion from the left mandibular coronoid process to the area of the left first bicuspid (Fig. 7). The patient had no interincisal opening. She underwent G-tube placement shortly after birth. At 1 month of age, the patient underwent partial excision of the maxillomandibular fusion and the coronoid process. Postoperatively, the interincisal opening was 5 mm, but physical therapy was hindered by patient compliance and the interincisal opening did not improve. At 5 years of age, the patient underwent a type I maxillectomy to

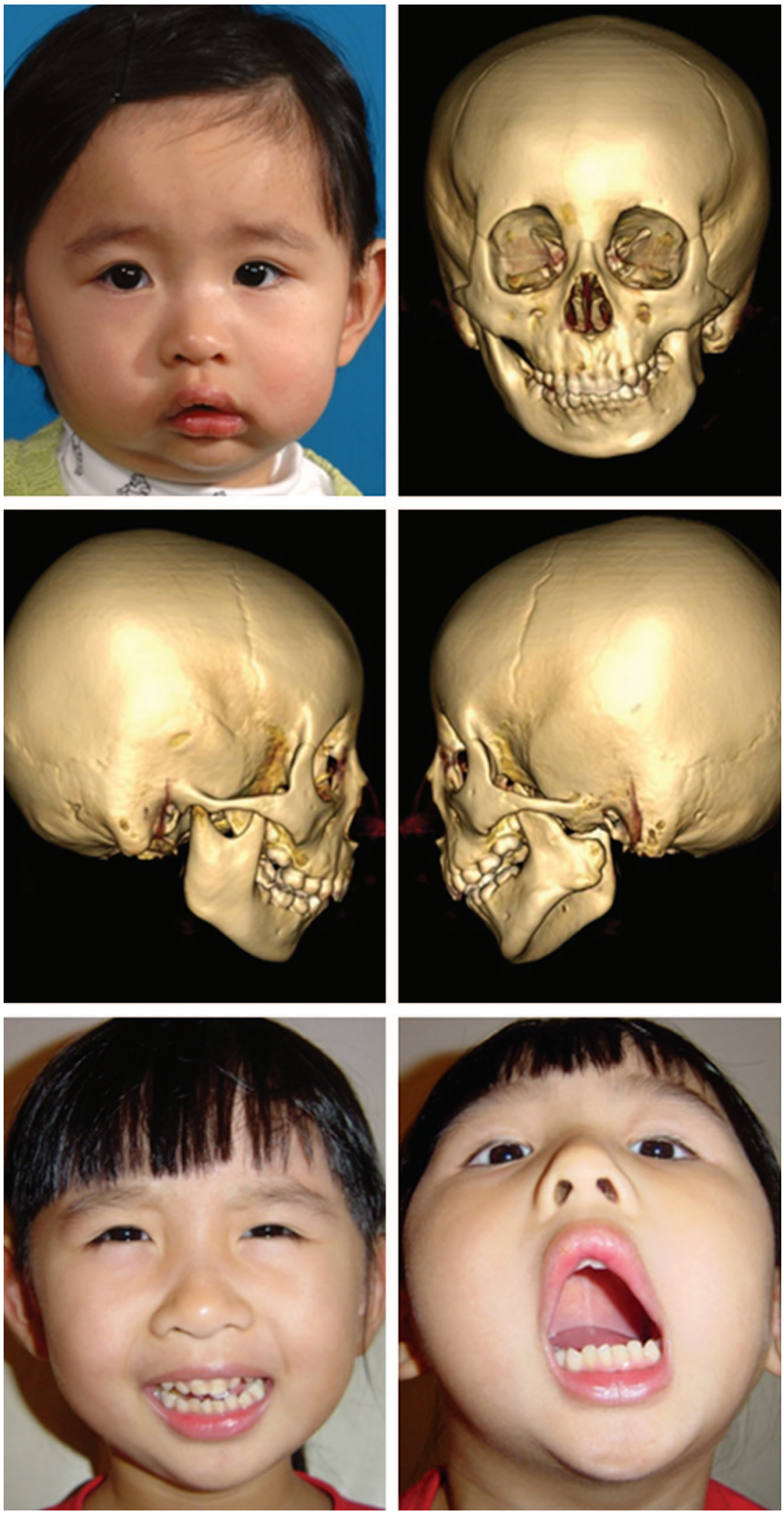

Fig. 5. Case 2. Preoperative images (*above* and *center*) of a 22-month-old girl (patient 18) who acquired an infection resulting in intracapsular ankylosis. Computed tomography

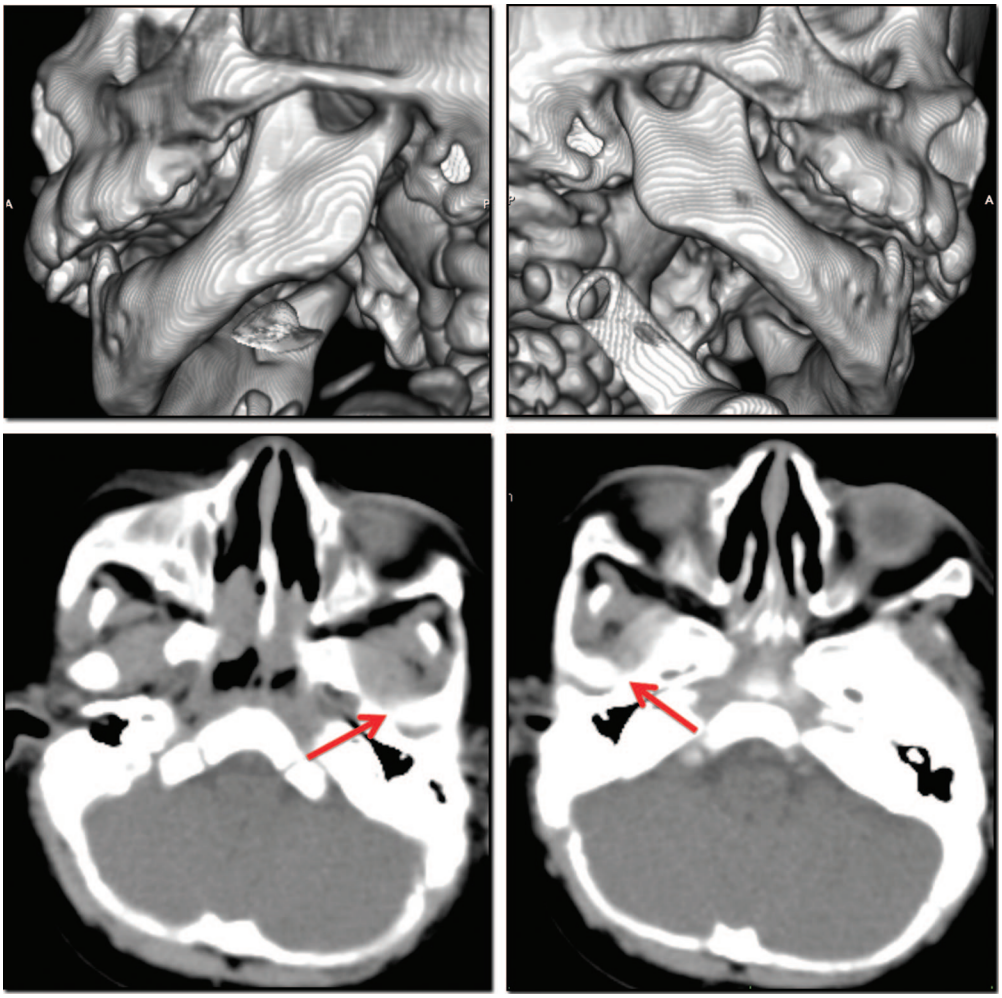

Fig. 6. Case 3. Two- and three-dimensional reconstructed computed tomographic scans of a female infant (patient 5) with congenital (bilateral) temporomandibular joint ankyloses. *Arrows* indicate the sites of the ankyloses.

remove the maxillomandibular fusion and a buccal fat pad flap was pedicled and interposed between the raw edges of the maxilla and the mandible. Postoperatively, the patient had 15 mm of interincisal opening.

DISCUSSION

On initial survey, the data from this review are consistent with previously published population studies that largely attribute pediatric temporomandibular joint disorders to acquired abnormality, specifically trauma (or surgery) and infection. However, in contrast, we observed a significant percentage (50 percent) of congenital temporomandibular joint ankyloses. Classification based on computed tomographic study of joint involvement revealed that the majority of congenital cases involved extracapsular abnormality, such as coronoid hypertrophy, whereas only a minority of congenital cases involved intracapsular ankyloses, as characterized by glenoid-condylar fibro-osseous fusion. In contrast, the majority of *acquired* cases of temporomandibular joint ankylosis were attributable to intracapsular abnormality.

Fig. 5. (*Continued*) demonstrated left temporomandibular joint ankylosis, with complete bony fusion of the mandibular condyle to the cranial base. There is coronoid process hypertrophy. The patient was treated with ipsilateral condylectomy (gap arthroplasty), reverse-L mandibular osteotomy, transport distraction osteogenesis, and coronoidectomy, followed by physiotherapy. At 2-year follow-up, she had an interincisal opening of 24 mm (*below*).

Epidemiology and Cause

The phrase "temporomandibular joint disorders" is a broad umbrella diagnosis that encompasses functional disturbances and anatomical

Fig. 7. Case 4. Preoperative images (*above* and *center*) of a 1-month-old girl (patient 9) with Hecht syndrome and maxillomandibular fusion extending from the left mandibular coronoid

disorders of the temporomandibular joint. Functional disturbances are characterized by restricted active motion in the context of normal passive range of motion. These are usually secondary to pain resulting from myofascial pain dysfunction syndrome, internal derangement (malposition of the disk), or capsulitis. In contrast, anatomical causes of temporomandibular joint disorders result in partially or totally restricted motion caused by either deformation of the joint itself or mechanical restriction by pathologic involvement of the bone or soft tissue.

Organic diseases that may directly affect the joint include rheumatoid arthritis, osteoarthritis, degenerative joint disease, psoriatic arthritis, and ankylosing spondylitis. Local or systemic infection may result in capsulitis, osteomyelitis, and suppurative arthritis. Trauma can result in fracture, ligamentous injury, and anterior joint dislocation. Similarly, injury from surgery, radiation therapy, or embolization therapy may result in soft-tissue scarring or fibrosis that can restrict the motion of the joint. Both bony and soft-tissue cancers may directly invade the joint or externally restrict mandibular excursion. The most common tumors are benign and include chondromas, osteomas, osteochondromas, chondroblastomas, and fibrous dysplasia. Malignant fibrosarcomas and chondrosarcomas are less frequently reported as causes of temporomandibular joint dysfunction. Finally, numerous congenital and developmental anomalies may affect mandibular growth and condylar development, resulting in temporomandibular joint dysfunction: condylar agenesis or hypoplasia, condylar hyperplasia, craniofacial microsomia, and coronoid hypertrophy.

The most morbid and disabling manifestation of temporomandibular joint dysfunction is immobility caused by ankylotic fusion of the mandible to the cranial base or zygoma. Although temporomandibular joint ankylosis is relatively rare, its incidence in the general population is unknown. Nevertheless, hundreds of cases of adult and pediatric temporomandibular joint ankylosis have been described in the literature over the past century. Temporomandibular joint ankylosis may occur in any age group, although children are at higher risk because of unique anatomical and physiologic differences at the joint, including a broader condyle and thinner cortical bone, and because of the higher incidence of nasopharyngeal and middle ear infections.[1–3] The most prevalent causes are infection and trauma (approximately 40 to 60 percent each in most series), followed by systemic illnesses, rheumatologic disease, arthritis, and cancer. Infectious cases were predominantly attributable to otitis media, mastoiditis, parotitis, or osteomyelitis of the temporal bone or mandible.[1,4–13] Reported pathogens include *Staphylococcus aureus*, *Streptococcus pyogenes*, *Haemophilus influenzae*, *Neisseria gonorrhoeae*, *Klebsiella pneumoniae*, paramyxovirus (measles and mumps), *Mycobacterium tuberculosis*, and *Variola*. With the introduction of antibiotic treatment, a demographic shift has occurred, with trauma surpassing infection as the predominant cause.[1]

The existence of congenital temporomandibular joint ankylosis has been the topic of much debate. Although a congenital form of temporomandibular joint ankylosis has been appreciated by several authors, others assert that these "congenital" cases are attributable, instead, to undocumented and unappreciated perinatal injury, and thus induced by trauma.[14–17] Certainly, congenital temporomandibular joint ankylosis is much rarer than any of the acquired forms, and its true prevalence is unknown. Our series, however, featured a high percentage (50 percent) of congenital cases because of a selection bias in the referral patterns to the Institute of Reconstructive Plastic Surgery. In all congenital cases in our series, the diagnosis was very clearly documented in the hospital record. For example, immediately after birth, the infant was found to have poor (or absent) mandibular excursion, making tracheal intubation difficult. Follow-up examination and radiographic imaging identified bony ankylosis within the first year of life.

Fig. 7. (*Continued*) process to the area of the left first bicuspid. Computed tomography demonstrated complete bony fusion. At 5 years of age, the patient underwent a type I maxillectomy to remove the maxillomandibular fusion and a buccal fat pad flap was pedicled and interposed between the raw edges of the maxilla and mandible. Postoperatively, the patient had 15 mm of interincisal opening (*below*).

Anatomy

The temporomandibular joint is a complex structure, allowing articulation between the condylar head of the mandible inferiorly and the mandibular fossa and articular tubercle of the temporal bone superiorly. It consists of two synovial cavities divided by an articular disk (Fig. 1). Allowing for both hinge-like rotation and anteroposterior gliding motion, the double-joint structure permits movements ranging from elevation (closing the mouth and biting) and depres-

sion (opening the mouth), to protrusion or retrusion, to asymmetric rotation (side-to-side and grinding motions). Small movements typically involve only the lower joint (hinge action), whereas large openings involve both joints (hinge and gliding actions). The joint is covered by an articular capsule that extends from the cranial base to the mandibular neck and is contiguous with the articular disk. The sphenomandibular and temporomandibular ligaments provide medial and lateral support, respectively. Finally, the discomalleolar ligament—a vestigial structure—connects the superior temporomandibular joint space with the middle ear, explaining the propensity for the development of temporomandibular joint ankylosis following severe otitis media.[18]

Mandibular movements are controlled by the coordinated actions of the muscles of mastication. Elevation of the mandible is enabled by the medial pterygoid, masseter, and temporalis muscles; the mylohyoid, suprahyoid, infrahyoid, and anterior digastric act in concert with gravity to depress the mandible; the lateral pterygoid muscles cause protrusion; and the posterior fibers of the temporalis muscle cause retrusion.

The blood supply includes branches of the maxillary, superficial temporal, and ascending pharyngeal arteries and veins. Neighboring vascular structures include the inferior alveolar and transverse facial arteries and veins. All of these vascular structures are at risk during mandibular and temporomandibular joint injury/surgery.

Several anatomical differences between the adult and pediatric mandible help to explain the increased incidence of temporomandibular joint ankylosis in children. The cortical bone in adults is quite dense, whereas in children it is thin. The condylar neck in adults is narrow, whereas in children it is relatively broad.[1] These factors predispose the adult mandible to subcondylar fracture, which is extracapsular, whereas fractures in children are comminuted and occur within the joint. Consequently, it is children, not adults, who typically have traumatic involvement of the joint, and the resulting hemarthrosis can undergo fibrinous organization that can become mineralized.[1,19]

Another anatomical difference lies in the anterior wall of the auditory meatus, which in children is composed of cartilage. Suppurative infections easily penetrate this weaker boundary and extend locally or track down the discomalleolar ligament to penetrate the joint capsule.[1]

Manifestations and Sequelae

The hallmark of temporomandibular joint disorders is decreased interincisal opening (typically <15 mm), with or without pain. In its severest form, it can be extremely incapacitating, resulting in limited mouth opening, malocclusion, interference with mastication, inability to form an oral seal, and thus difficulty achieving adequate nutrition and maintaining oral hygiene.

Temporomandibular joint ankylosis in the pediatric patient results in restricted mandibular growth and retrognathism. Specific manifestations are contingent on laterality: in unilateral ankylosis, the chin is deviated ipsilaterally/posteriorly because of mandibular ramal shortening on that side; in bilateral cases, there is mandibular retrusion without asymmetry.[1] Unilateral ankylosis typically results in a crossbite, whereas the more severe mandibular underdevelopment in bilateral cases typically results in micrognathia and an anterior open bite. In some cases, particularly in children, the mandibular condylar neck is shortened and the coronoid process extends above the level of the zygomatic arch. Pressure from lips and tongue-thrusting (when trying to form an oral seal) may adversely mold dentition. However, overall functional occlusion is rarely affected to any significant degree because of the adaptive capacity of the alveolus, which remodels to allow for dental repositioning.[1] Untreated unilateral ankyloses may result in bilateral disease, as restricted movement eventually causes a fibro-osseous union on the contralateral side.[1]

The severity of symptoms and specific sequelae are highly dependent on age at the time of onset of ankylosis, because injury in the growing child may have a pronounced effect on growth and development. The traditional teaching was that the more serious manifestations in children are attributable to damage to the condylar growth center.[1] However, more recent experimental evidence shows that this may not be true. For example, unlike the long bone growth centers, the cartilage cells in the condyle are not arranged in parallel rows,[20,21] and tritiated thymidine labeling studies found that this region contributed only minimally to new bone formation.[22] In a series of experiments in *Macaca rhesus* and *Saimiri sciureus* monkeys, Sarnat demonstrated that the craniofacial changes after condylectomy were comparable in both growing monkeys (with mandibular growth centers) and adult monkeys (without growth centers).[23–26] Similar findings were noted to occur regardless of the cause of joint injury

Table 2. New York University Classification for Etiologic and Anatomical Categorization of Temporomandibular Joint Disorders

		Acquired				
Classification	**Congenital**	**Secondary to Other Deformity or Defect**	**Trauma**	**Infection**	**Rheumatic or Vascular**	**Neoplasm**
Soft tissue		Disuse contracture	Pericapsular fibrosis (e.g., chemical and thermal injuries); postorthognathic surgical scar contracture	Periodontitis, parotitis	Myofascial pain dysfunction syndrome; ischemic injury; treatment-induced fibrosis (e.g., irradiation, sclerotherapy)	Tumor invasive of musculature
Skeletal						
Extracapsular	*True* developmental coronoid hypertrophy; fibrodysplasia ossificans progressiva (myositis ossificans)	*Relative* coronoid hypertrophy (acquired short ramus)	Malunited fracture of zygoma; restricting fibrous adhesions to coronoid process			Tumor invasive of coronoid process
Intracapsular	Congenital TMJ ankylosis (bony fusion)	Metachronous contralateral abnormality in cases of longstanding unilateral ankylosis and restricted excursion	Recurrent dislocation; intracapsular comminuted fracture (e.g., penetrating trauma, forceps delivery; postorthognathic surgical deformity; radiation-induced osteoradionecrosis)	Otitis media; mastoiditis; parotitis; pericapsular suppuration; osteomyelitis; paramyxovirus; *Streptococcus pyogenes*; *Variola*; actinomycosis	Ankylosing spondylitis; osteoarthritis; rheumatoid arthritis	Tumor invasive of joint and capsule

TMJ, temporomandibular joint.

(including condylar fracture, dislocation, irradiation, and infection).[27,28] Furthermore, replantation of the subcondylar growth center following its surgical removal was insufficient, by itself, to restore normal growth.[29,30]

These findings suggest that the more important factor is loss of the temporomandibular joint itself and disruption of its normal function, which leads to change in the magnitude and direction of muscular pull and subsequent remodeling of the mandibular ramus and adjacent bony structures.[24] Many authors now agree that it is the investing soft tissues, especially the muscles of mastication, and the forces they exert that serve as the primary impetus for a compensatory skeletal growth.[31–35] Adverse sequelae, such as facial asymmetry resulting from temporomandibular joint fracture or condylar resection in animals, were found to be prevented by adequate fracture reduction or reconstruction of the condyle.[30] Thus, the presence of a normally functioning joint reestablishes the proper balance of muscular tension that is necessary for adaptive growth and remodeling.[1]

Classification

Temporomandibular joint disorders can be broadly categorized into functional disturbances (e.g., tetany, myofascial pain syndrome) and anatomical abnormality. Anatomical disorders are subdivided into soft-tissue or skeletal defects, depending on which part of the anatomy (e.g., musculature, mucosa, and glands, versus joint and skeleton) is principally affected.

Temporomandibular joint skeletal disorders were first classified by Kazanjian in 1938,[16] with subsequent modification by Rowe.[1,36,37] According to this system, temporomandibular joint ankylosis is either a "true" case, involving bony fusion of the joint surfaces; a "false" case, in which the joint is normal, but joint dysfunction occurs because of extracapsular ankylosis (e.g., periarticular fibrosis, as might result from infection); or a "pseudo" ankylosis, in which joint immobility occurs because of mechanical interference (e.g., coronoid hypertrophy). We have found that most cases of pericapsular abnormality invariably lead to true joint involvement, thus converting pericapsular cases into intracapsular cases. Furthermore, diagnosis of pericapsular disorders is difficult prospectively and impossible retrospectively. Therefore, we suggest improving the utility of the anatomical classification scheme by considering only intracapsular or extracapsular pathologic findings. It is also possible to correlate the anatomical categorization with specific causes to yield an etiologic classification scheme (Table 2).

Whereas etiologic classification summarizes relevant history and clinical diagnosis, anatomical classification summarizes the pathophysiologic diagnosis derived from physical examination and radiographic workup, which can guide the choice of treatment modality. As demonstrated in our selected cases, we would encourage the use of both descriptors for a full classification (e.g., "congenital/intracapsular" or "acquired (trauma)/intracapsular").

CONCLUSIONS

The data reported in this review are, in part, consistent with previously published population studies that principally attribute pediatric temporomandibular joint disorders to acquired cause, specifically, trauma and infection. In contradistinction to prior published reports, however, we observed a significant percentage (50 percent) of congenital cases of temporomandibular joint skeletal disorders. Computed tomographic studies revealed that most of the congenital cases involved extracapsular disease, such as coronoid hypertrophy and maxillomandibular bony fusion. Only a minority of congenital cases represented intracapsular ankyloses, as characterized by glenoid-condylar fibro-osseous fusion.

Because precise diagnosis and classification directs the choice of therapy, we propose a new classification to differentiate between soft-tissue and skeletal abnormalities and to better characterize the extent of capsular involvement. Protocol-based preoperative computed tomographic scanning is recommended to aid in classification and the selection of treatment modalities. Operative protocols are varied, and these treatment modalities will be discussed in Part 2 of this series.

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PATIENT CONSENT

Parents or guardians provided written consent for the use of patient images.

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