Jaw-Joint Disorders in Contact-sports' Athletes: Diagnosis and Prevention


ABSTRACT: Clinical and radiologic findings in contact-sports' athletes who sustained injury to the Vital Cranial Triad (VCT), a complex of bones consisting of the temporomandibular joint (TMJ), the tympanic temporal bone and the inferior surface of the petrous temporal bone, were studied. Approaches to the diagnosis of temporomandibular disorders focused on findings from patient history, clinical examination and a modified transcranial radiographic examination. Indications for radiography included headache, earache, nausea, vomiting, vertigo, impaired hearing and balance, muscle weakness, occlusal disturbances, TMJ pain and other symptoms associated with neurologic and circulatory deficit secondary to trauma. Radiologic findings included compression fractures, condylar neck fracture, degenerative condylar remodeling, obliteration of the articular eminence, atresia of the ear canal and other damage to the VCT. Structural damage to the VCT is described and the associated symptoms are discussed in the context of 4 exemplary cases. The use of a customized mandibular orthopedic repositioning appliance to prevent injuries to the VCT is discussed. Damage to the VCT leads to painful and tragic sequelae which can and must be prevented in contact sports.

KEYWORDS: vital cranial triad, temporomandibular disorders, modified transcranial radiography, intraoral jaw-joint protection appliance, strength testing, symptomatology, diagnosis, prevention, repetitive impact loading, sequelae, jaw-joint disorders, contact sports

The relationship of trauma to temporomandibular disorders (TMD) has, until recently, been ignored or underestimated by many health-care providers possibly because of difficulties in demonstrating TMJ damage with conventional radiographic techniques and possibly because of inadequate provider training in craniomandibular physiology.

Clinicians agree that TMD arises from several pathologic conditions which affect mandibular opening, deglutition, mastication and other neurologic functions in which pain and discomfort predominate. TMD is one of the health hazards uniquely associated with contact sports such as hockey, football, soccer and boxing, the most frequent cause being repeated subconcussive blows to the head, chin or jaw, directly or via the headgear. In practically all instances, the vital cranial triad (VCT), which includes the TMJ, the tympanic temporal bone and the inferior surface of the petrous temporal bone, is violated. This complex of bones houses and ports important cranial nerve trunks as they exit from the base of the brain; it also houses the blood supply to the brain and the auditory and balance mechanisms, among others. Specifically it performs three important functions: it permits multiplanar motion of the mandible; supports the mandible and a variety of hard and soft tissue structures at the base of the skull; and, most importantly, it serves as a protective conduit for cranial nerve trunks and blood vessels. Thus, it should come as no surprise that athletes with VCT injuries often present with symptoms reflecting neurologic and circulatory deficit. These symptoms include headache, earache, facial pain, bloodshot eyes, photosensitivity, muscle weakness, pain and numbness of extremities, vomiting, vertigo and impaired speech among others (Table 1).

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### Table I. Symptoms-Athletes Jaw Disorder

<table>
<thead>
<tr>
<th>EYES</th>
<th>MOUTH</th>
<th>HEAD</th>
<th>EARS</th>
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<tbody>
<tr>
<td>- Sensitivity to light</td>
<td>- Discomfort when chewing</td>
<td>- Radiating headache pain from forehead to eyebrow area</td>
<td>- Clogged or itching ear with no infection or foreign body</td>
</tr>
<tr>
<td>- Pulsating pain behind eyes</td>
<td>- Discomfort when at rest</td>
<td>- Pain and pressures similar to sinus problems</td>
<td>- Dizziness or vertigo; ringing, hissing, or buzzing sound</td>
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<tr>
<td>- Bloodshot eyes</td>
<td>- Pain when opening mouth</td>
<td>- Ache in temple area—above and in front of the ear</td>
<td>- Earache but no infection</td>
</tr>
<tr>
<td></td>
<td>- Clicking or popping when opening mouth</td>
<td>- Hair or scalp painful to touch</td>
<td>- Decrease in hearing capacity</td>
</tr>
<tr>
<td></td>
<td>- Limited opening of mouth</td>
<td>- Radiating pain in back of head</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Jaw jumps or deviates to one side when opened</td>
<td>- &quot;Migraine&quot; type headaches</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Jaw locks in open position when eating, yelling, or yawning</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>- Teeth do not seem to fit together properly; can't locate &quot;bite&quot;</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Unconscious grinding of teeth, during times of anxiety or asleep</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- &quot;Migraine&quot; type headaches</td>
<td></td>
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<tr>
<td></td>
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<tr>
<td></td>
<td>- Earache but no infection</td>
<td>- Decrease in hearing capacity</td>
<td>- Decrease in hearing capacity</td>
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</table>

There are a few studies which identify trauma as a major cause of TMD in the general population and few, if any, standardized approaches to diagnosis of TMD [1-11]. To our knowledge, there are no studies which link trauma and its sequelae to VCT injuries in male athletes. There are a variety of methods employed for visualizing the TMJ/VCT, each with specific indications, limitations, availability and attendant risks. Arthroscopy, for example, provides excellent visualization of adhesions, perforations and displacements of the articular disc, as well as an indication of the contour of the meniscus and its relationship to the condyle and the articular fossa [12-16]. Its use is complicated by pain, hemorrhage, lacerations, instrument breakage and limited field [12-18]. It is also invasive, expensive, not easily available and requires specialized equipment and training. Panoramic images provide good visualization of hard structures associated with mastication, but show a loss of the true shape of the condyle because of the lateral projection of the beam [19]. Moreover, articular fossa are often obscured if the patient has limited mandibular opening [12-15]. With tomography, bony changes and functional relationships are easily visualized and non-osseous structures can be seen without dye injection. However, its use is limited by cost, availability, high levels of radiation and limited definition [12,15,20]. MRI is used for soft tissue analysis. It is more accurate than arthrography in providing soft-tissue contrast, is virtually free of ionizing radiation and is noninvasive. However, its linear focus has a limited depth of field and poor definition of structures. Like most of the other methods, it is very expensive, not easily available and not adaptable for office use.

The most practical and most affordable alternative to the methods described is a modified transcranial radiographic technique which we have developed. It is also relatively inexpensive, noninvasive, easily adaptable for use in medical, dental and sports facilities, and carries less risk of ionizing radiation than other methods. There is also less distortion due to overlapping structures than with conventional radiography, and sharper definition of bony structures. In addition, the pathologic results of trauma to the VCT are radiologically demonstrable with this technique. In this communication the pathologic results of trauma to the VCT of athletes are described and the symptoms associated with VCT damage are discussed in the context of 4 exemplary cases.

### METHODS AND MATERIALS

Bilateral transcranial radiographs of 31 male and 3 female athletes, ages 17-43 years were selected for study of destructive changes of the VCT. The patients, all referrals, had presented with one or more symptoms of pain, dysfunction, clicking, locking, occlusal difficulties, vertigo, tinnitus.
crepitus, and hearing impairment, among others (Tables 1 and 2). Headache, stiff neck and shoulder weakness were predominant symptoms. All had described a relationship between onset of symptoms and previously incurred trauma to the jaw.

Usually, symptoms became apparent 2 to 3 months after injury and had lasted for periods varying from 3 months and beyond. In some, symptoms became apparent immediately. All patients were evaluated by standard methods including a full mouth and transcranial radiographic examination, physical examination of the head, neck, jaw and shoulder areas and a medical history. In addition, two simple strength tests were conducted using the Nicholas MMT (Model 01160). Both were used to ascertain loss of strength in athletes with VCT injury. Injury to the VCT results in the loss in strength and physical endurance because of localized neurologic impairment at the nerve trunk. Essentially the condyle was unloaded by moving the lower jaw by mechanical means to an inferior and anterior position away from the injured VCT. Various muscle groups were then tested for strength loss or gain and the results were recorded.

### CLINICAL SYMPTOMS

<table>
<thead>
<tr>
<th>Athletes Jaw-Joint Disorders (n=34)</th>
<th>FOOTBALL (n=12)</th>
<th>BOXING (n=12)</th>
<th>SOCCER (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stiff neck</td>
<td>12</td>
<td>12</td>
<td>9</td>
</tr>
<tr>
<td>Headache</td>
<td>12</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>Ear sounds</td>
<td>10</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>Blackout</td>
<td>12</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>VCT sounds</td>
<td>8</td>
<td>11</td>
<td>4</td>
</tr>
<tr>
<td>Sinus pain</td>
<td>9</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Rotator cuff</td>
<td>12</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Locking jaw</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

Table 2

### RADIOGRAPHY

Transcranial radiographs were made with a Bennett Head Unit. The patient’s head was positioned laterally, with the imaginary ala-tragus line parallel to the floor. The tragus was positioned so that the lateral view of the TMJ/VCT would include the external auditory meatus, the total translatory space of the condyle, and the tympanic temporal bone to exclude unwanted cranial bones from the radiograph. Angulation was also described and standardized for the cassette holder and the radiation source to minimize angulation changes that would adversely affect the radiograph. Semi-axial lateral projections were made with both open-and closed-mouth positions. An 8 x 10 inch or a 24 x 30 mm cassette with screen was placed on the horizontal plane of

![Fig. 1](image1.png)  
![Fig. 2](image2.png)
the cassette holder and an adapter was used. The X-ray source was collimated to the aperture of
the adapter to create 2 circular exposures of the same film. An X-ray grid was used to eliminate
scatter radiation effects on the film. The mAs (mA x time), was calibrated with the kVp at 70 to
80. The films were exposed, processed, traced, and examined. The volume of joint space and the
destructive changes in and around the VCT area were studied and compared with dried skull
specimens and the reference transcranial radiographs described as normal.

Radiographs of a 53 year-old male volunteer (Fig.1) which approximated normal VCT findings
were used as reference. These were then compared with a series of dried skulls. A representative
dried skull was chosen (Fig.2) which showed anatomical markings typical of a so-called “normal”
VCT complex. A comparison of Figs. 1 and 2 reveals a relatively normal condyle morphology
except for an early change at the antero-superior head of the condyle (Fig. 1). Glenoid fossae are
congruent with the condylar heads, the tympanic temporal bone forms two well-formed walls
and floor of the external auditory meatus, i.e., the anterior wall (0-1), the inferior portion of the
wall (0-2) and part of the posterior wall (0-3). The anterior wall of the tympanic temporal bone
was convex facing the condyle; the posterior surface of the condyle is convex facing the temporal
tympanic bone; the antero-superior surface of the anterior wall of the tympanic temporal bone is
contiguous (G) with the infero-posterior or surface of the postglenoid process (H); the articular
eminence (D) is well-defined with rounding slopes, and the assumed disc space (F) suggests
adequate disc thickness.

Case Reports
Case 1. A 24-year-old football player complained of shoulder pains, frequent stiff neck, gurgling
and raspy voice patterns, headaches of unknown origin, periodic tingling and smarting sensa-
tions of the upper extremities and fingertips, and a steady decline in the amount of weight he
could lift on bench press. The right transcranial radiograph (Fig.3) reveals an increase in the
sclerotic density of the tympanic temporal bone (O-1, O-2); traumatic displaced fracture (G) of the
supero-anterior surface of the tympanic temporal bone and the postero-inferior surface of the
postglenoid process (H). A change in the condyle/fossa congruence; flattening of the superior
surface of the condyle (A); pyramidal reshaping of the of the fossa (E); change in the definition of
the external auditory meatus (B) as a result of the green-stick fracture of the anterior wall of the
tympanic temporal bone; degenerative changes of the posterior slope of the glenoid fossa
HEAD AND NECK INJURIES IN SPORTS

(nondisplaced healed fracture)(2 open arrows); and increase in sclerotic density and thickening of the postglenoid process. In some instances such blows can force the condyle into the external ear canal.

The left transcranial study (Fig 4) shows structural damage similar to that seen on the right condyle as a result of repeated trauma to the tympanic temporal bone, the condyle, and the fossa. Note the folding effect of the anterior wall of the tympanic temporal bone posteriorly; increased radiodensity (opacity) of the anterior floor and posterior wall of the tympanic temporal bone; condyle-to-eminentia interference during condylar motions of translation as determined by the osseous destruction of the articular eminence. There is also evidence of destructive osseous changes which causes obliteration of the articular eminence and the fracture of the posterior incline of the glenoid fossa (2 open arrows), and increased radiolucency at the tympanomastoid fissure (2:1 opposing arrows).

With the insertion of the jaw-joint protective appliance, the patient experienced an increase in strength during workouts and a notable decrease in the frequency and intensity of headaches and facial pain after play.

Case 2. A 28-year-old male heavyweight boxer with a ring history of 6 years, presented with symptoms of middle ear pain and undiagnosed severe debilitating headache. The right transcranial radiograph (Fig. 5) shows a tubercle-like extension on the postero-superior surface of the condylar head (A); reshaping of the posterior slope of the glenoid fossa(E); remodelling of the anterior wall of the tympanic temporal bone(O) and the posterior surface of the condylar head due to trauma of the anterior surface, producing an indentation congruent with the posterior surface of the condyle. There is also definite indication of early thinning of the tympanic temporal bone characterized by changes which tend towards concavity (facing the condyle), and loss of osseous integrity of the inferior slope of the articular eminence, indicating interference during condylar translation. Also seen is the fracture(G) of the union between the tympanic temporal and the postglenoid process (2 arrows).
The left transcranial radiograph (Fig. 6) reveals the following: early progressive thinning of the tympanic temporal bone (O); increased radiolucency of the fracture of the union between the tympanic temporal bone and the postglenoid process (G); structural change of the glenoid fossa with the loss in radiographic definition of the posterior slope; functional interference between the postglenoid process and the postero-superior surface of the condyle (open arrows), and loss in the integrity of the inferior slope of the articular eminence. (D).

Here too, the jaw-joint protective appliance proved to be of considerable benefit. All symptoms disappeared upon the insertion and utilization of the appliance.
Case 3. This patient, aged 43 years, had a ring history of over 20 years. He was unable to open his mouth more than 22 mm, his speech was soft and slurred, his voice was raspy, he exhibited parkinson-like tremors, and he suffered from impaired balance as evidenced by his "stagger-step" gait. The right transcranial radiograph (Fig. 7) shows a severely thinned anterior wall and floor of the tympanic temporal bone (0-1); loss of definition of the mastoid process; displaced fracture and chronic scarring of the posterior slope of the glenoid fossa (E). In addition, a shallow recess of the fossa and degenerative changes of the articular eminence (2 arrows). There is also a loss of structural definition of the postglenoid process (H).

![Right Transcranial](image)

The left transcranial radiograph (Fig. 8) reveals a slender and diminished condylar head with degenerative changes in the neck of the condyle(A); displaced and nondisplaced fractures of the posterior wall; fracture of the surface of the anterior wall(O-1) and inferior surface(O-2) of the

![Left Transcranial](image)
tympanic temporal bone. Also identified are shallow glenoid fossa (E) with a displaced healed fracture of its posterior slope; change in the morphology of the postglenoid process interfering with the posterior surface of the condyle at rest (open arrows); concavity of the anterior wall of the tympanic temporal bone (0-1) facing the condyle (A); increased radiolucency of the tympanomastoid fissure (3 arrows) and increased radiolucency of the posterior wall of the tympanic temporal bone. Present also are changes in the mastoid air cells (j); a severely eroded tympanic temporal bone, sharply exposing to view the underlying petrous temporal bone and the internal carotid canal (D); and a fracture of the posterior wall of the tympanic temporal bone (3 small arrows).

In this case the jaw-joint protective appliance proved to be of limited benefit although the tremors and the stagger-step gait decreased dramatically with its use.

Fig. 9 Right Transcranial

Case 4. A twenty three year-old female soccer player with field play experience of thirteen years, presented with migraine-like headaches, facial pain, deviated jaw movements and a past history of headaches and jaw problems which appeared after games and continued to worsen with time. She had previously undergone a condylectomy with no apparent benefit. The right transcranial radiograph (Fig. 9) reveals the condyle (A) in a higher than usual position in the recess of the fossa (E); feathering of the articular eminence (D), indicating interference during translation. Also visible are green-stick (O) fracture of the anterior wall of the temporal tympanic bone; sclerotic changes in the temporal tympanic bone, indicative of repetitive loading of that bone; and increased opacity of the squamotympanic fissure.

The left transcranial radiograph (Fig. 10) reveals the condylectomy (A) restricted translation during opening; feathering of the articular eminence; a nondisplaced fracture at the postglenoid process; and increased opacity of the temporal tympanic bone and squamotympanic fissure.

With the use of the jaw-joint protection appliance during play and training, the patient volunteered that her headaches diminished in frequency and intensity and that she experienced greater facial comfort after the athletic event.
DISCUSSION

Head and facial trauma as the underlying cause of TMD, mandibular and condylar fractures and dislocations, and related bone damage has not been widely reported in the past, possibly because of difficulties encountered in demonstrating TMJ/VCT damage with conventional radiographic techniques. The recent literature [1,3,21-25] reports a significant percentage of patients with internal derangements with a history of facial trauma before the onset of the TMD. The disorders included osseous fractures, disc interference, degenerative joint changes, and condylar dislocations. In our study, we encountered similarly devastating effects of head and facial trauma more often than in a normal population because of the homogeneity of the patient population. All were athletes, either football, soccer players or boxers, who were repeatedly exposed to the punishing effects of head and facial trauma. All had used accepted or mandated types of mouthguards. Much of the damage sustained was radiographically visible.

Among the taller football players, we observed diminished disc space with upward condylar positioning and pyramidal-shaped fossae; tympanic plate fractures resulting in atresia of the external auditory meatus; condylar morphology changes and posterior displacement of the condyle. While condylar damage and dislocations as well as other damage to the TMJ/VCT and surrounding areas have been observed in the general population [1,22-28], atresia of the external auditory meatus per se, has not been reported.

The reports of Akers and co-workers [21] and those of Worthington [22] mention involvement of the external auditory meatus in patients who sustained injuries in automobile accidents. Akers observed posterior dislocation of the condyloid process into the external ear canal, and later, partial stenosis of the external ear canal in the same patient. (Female patients encountered in our practice who were victims of spousal abuse, exhibited similar clinical symptoms and findings). Worthington in his report of a patient with multiple injuries, including a fractured mandible, observed that “...on occasions circumstances may so combine as to allow posterior dislocation with fracture of the bony tympanic plate and disruption of the external auditory meatus.” In all contact sports the VCT is repeatedly exposed to potentially injurious forces beyond the tolerable load design of the jaw-joint mechanism. This repetitive impact loading superimposed on already weakened or compromised VCT structure will predispose the athlete to more serious injuries.

In football, damage to the VCT is induced by repeated blows to the chin, faceguard, and helmet. The relative force exerted at the point of impact (considering the body mass of the individuals involved, their combined speed, and the angle of impact) is more than the VCT can reasonably absorb. The four
point chin strap contributes to the progression of damage, because its positioning, which secures the helmet to the head, also pulls the mandible superiorly and posteriorly. This antagonistic positioning of the condyles, adversely influences the neurologic and physiologic functions of the delicate structures of the vital cranial triad. This impacts the characteristic potential for injury. Contact against the faceguard also greatly contributes to VCT damage. Furthermore, in this critical area, repetitive load forces are intensified. These forces are directly transmitted from a bone of higher mass and density (the condyle) to bones and tissue of lesser mass and density (the undercarriage of the skull and brain).

Similar destructive changes were noted and identified in boxers. Radiographic studies revealed osseous changes, including bone thinning resulting from microfractures of the tympanic temporal bone and posterior surface of the condyle. The progressive erosion of the anterior and medial walls of the tympanic temporal bone as well as the erosion of the floor and posterior wall are attributable to microfractures induced by the cumulative effects of multiple blows to the jaw forcefully driving the condyle posteriorly and medially. In addition, the thinning of the temporal bones rendered the VCT more vulnerable to fracture of the glenoid fossa, cranial vault and exposure to the cranial vault and blood supply.

Copenhaver and colleagues [3] described a fracture of the glenoid fossa and dislocation of the mandibular condyle into the middle cranial fossa of a young patient who sustained trauma to the chin after falling from a bicycle. After surgery, the patient recovered and showed no signs of neurologic deficit. They stated that although craniofacial structures may serve to prevent violation of the cranial cavity, occasional condylar dislocations occur which may be related to several factors, including the magnitude and direction of the trauma, morphological abnormalities of the condyle and a particularly thin glenoid fossa roof. Orban [2], on the other hand, observed that "...the thinness of the bone in the articular fossa is responsible for fractures if the mandibular head is driven into the fossa by a heavy blow. In such cases injuries of the dura mater (subdural hematomas) and brain have been reported."

These changes may explain the headaches and intracranial pressures experienced in athletes engaged in contact sports. It may also explain, to some degree, symptoms suggestive of encephalopathy as in Case 3, where radiographic evidence pointed to massive damage to the VCT, including the medial encroachment of the condyle on cranial nerve trunks and the internal carotid artery which produce symptoms that mimic brain damage in boxers. The insertion of an appliance which positions the condyle anteriorly and inferiorly to the fossa, unloads the joint and relieves pressure on the vital structures, was used in the boxer with parkinsonian-like tremors. The severity and extent of his tremors decreased and his gait and balance improved. Considering the long professional career of this boxer, it is not at all unlikely that neurologic symptoms may have developed concurrently with, as a result of, or independently of damage to the VCT.

Brain damage and brain atrophy in boxers is discussed elsewhere in the literature [4,29-31]. It should be noted, that in all instances (except for Case 3) the insertion of an appliance which unloads the joint, elicited beneficial responses. Symptoms were ameliorated, impaired neurologic function was improved and significant muscular strength and resistance was restored.

Clinical experience points to an unusually high incidence of VCT damage among soccer players and focuses our attention to the net effects of "heading the ball" while contending with an opponent in the field. As Fig. 11 illustrates, the risk of injury to the VCT in soccer players may be as great as in football, hockey and boxing. Attention must also be focused on the assumption that there are now more women participating in physical sports than ever before. Injuries to the delicate area of the VCT may have a more intensely adverse influence on the physical functions of the female. Further studies in this area are indicated.

The unusual nature of the symptoms reported by the athletes in this communication requires further comment. As can be seen from Table 1 there were a variety of symptoms reported which are not usually associated with TMD, but are unique to athletes with jaw-joint disorders and VCT damage. In all likelihood, these symptoms arose as a result of cranial nerve trunk and blood vessel damage, following traumatic displacement of the condyles (particularly the left condyle because most boxers are right-handed and inflict power blows to the left side of their opponents' jaw) from a blow with a
posterior, medial and superior force vector. Following such a blow, the left condyle moves posteriorly, medially and superiorly into the area of the temporal tympanic bone and the inferior surface of the petrous temporal bone, thus encroaching on the area of the jugular foramen. Porting the jugular foramen directly off the brain stem are three cranial nerve trunks: (1) the glossopharyngeal, which influences the muscles of the pharynx, the stylopharyngeus and muscles of the soft palate and pharynx, which affect gag reflex, taste, swallowing patterns, and tongue sensations; (2) the vagus, which influences the voice box thus affecting speech and speech patterns. The vagus also branches to form the superior laryngeal and recurrent pharyngeal nerves which influence changes in voice volume and tone (in boxers this is characterized by low volume conversational tone, rasping voice, etc). The vagus also supplies heart, diaphragm and the gastrointestinal area. The consequent trauma can produce a vagal-vagal response such as nausea and vomiting (the chief symptoms of the boxer previously described). A large portion of the vagus nerve is afferent, sending impulses to reflex centers of the brain affecting many other bodily functions. It should also be noted that the vagus nerve (through its reflex functions) may be the principal cranial nerve involved in the recapture of strength loss experienced by athletes with VCT injuries. (3) the spinal accessory nerves which supply the sternocleidomastoidus and trapezius muscles. These muscles directly affect head and shoulder movement, limit abduction and adduction of the upper appendages and inhibit subscapular rotation, causing painful movements of the upper appendages. This is often misdiagnosed as a rotator-cuff injury. A fourth cranial nerve (the hypoglossal nerve) ports through this area after leaving the hypoglossal canal into the jugular sheath. Injury to this nerve affects tongue movement, causing slurring speech and thick-tongue syndrome. In juxtaposition to the jugular foramen lies the carotid canal which houses the internal carotid artery, one of the principal arteries which supply the brain. Trauma to this area adversely affects the nerve trunks and the internal carotid. Repeated trauma to the artery will produce fissures of the intimal surface, which predisposes this tissue to localized atherosclerotic development. Complicating this development, is the inability of the injured carotid artery (after making a turn into the bony sheath of the internal carotid canal) to accommodate the progressive accumulation of plaque. What follows is the occlusion of the artery and a diminution of the blood and oxygen supply to the brain thereby, predisposing the athlete to stroke and other encephalopathies seen among retired contact-sports’ athletes.

Recent developments in the management of jaw-joint injuries in athletes make particularly timely an appraisal of mouthguards in current use. While conventional mouthguards prevent dental damage, they also potentiate damage to the VCT and, as we have observed, the protection they offer to the VCT is far from satisfactory. It is important to recognize that the mouthguard design must provide stabilization of the jaw-joint. Clinical stability of the jaw-joint is defined as the ability of the condyle to limit its patterns of displacement of physiological loads to prevent damage, interference, or irritation.
to any of the structures of the vital cranial triad. Condylar instability/displacement perpetuates disc
disease and degenerative changes of the vital cranial triad. Obviously a compelling need has always
existed for an appliance which could effect and sustain adequate protection of the VCT to reduce risk
factors during offensive exchanges in the ring or the athletic arena. Lateral loading of the condyle can
produce hyperextension of the condylar capsule and impingement of the medial surface of the
condyle onto the protective conduit of the bones of the VCT. With repeated injurious lateral forces, the
capsular compliance is lost. This increases condylar displacement, producing destruction or fractures
of the protective conduit bones and damage to cranial nerves and blood supply.

Our observations of a newly designed Jaw-Joint Protection Appliance, WIPSS™, indicate that
damage to the VCT can be prevented and risk factors reduced. The WIPSS Appliance is designed to
create a buffer or recoil space between the condyle and the VCT by repositioning the condyles
(Fig.12,13).

Fig. 12  Condyle Position with Mouthguard

Fig. 13  Condyle Position with Jaw-Joint Protection Appliance
The recoil space created by the insertion of WIPSS, and the WIPSS appliance with the dental arches act in concert to absorb and dissipate the impact of any blow to the head, chin or face. It also reduces traumatic lateral displacement of the lower jaw (Figs.14,15), thus protecting the temporal bone complex, the base of the cranium, the dental arches and the dentition against fractures. Most importantly, the repositioning of the condyles, anterior to the injured structures, permits the athlete to function at optimal physical levels (increased muscular strength, resistance and thereby decreased fatigue) thus decreasing the likelihood of cervical and other facial injuries.

CONCLUSIONS
Contact sports are hazardous. Protective equipment such as helmets and face shields cannot protect athletes from all injuries to the head, face and jaw-joint. In fact, existing equipment can and does potentiate jaw-joint injuries. In sports, where retention performance of the helmet is based on a chin cup or the face shield in contact with the chin, impact loading to the helmet or face shield transmits the load force directly to the jaw-joint. The resultant injuries include bone fractures, damage to the delicate tissue at the base of the skull and repeated bleeding episodes at the base of the brain, i.e. subdural hematoma. After careful study and evaluation of the mechanisms and forces involved in head injuries and recognition of the pervasiveness of jaw-joint injuries, safety specifications for jaw-joint protective equipment are warranted. Consideration must be given to the dentition of the upper and lower arch and related structures, and to the vital cranial triad. The intent is to maximize clinical stability and minimize traumatic jaw displacement, as well as prevent injury to the delicate structures of the VCT, when subjected to load force. The result of utilizing a jaw-joint protective appliance (WIPSS) does indeed dramatically reduce jaw-joint and related head injuries.
REFERENCES


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