THESE

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Lucile TUCHTAN TORRENTS

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Etude de la transmission des forces à l'extrémité céphalique suite à un impact facial

Laboratoire de Biomécanique Appliquée
– LBA ADES/AMU –

Présentée devant le jury composé de :

Pr Norbert TELMON	Université de Toulouse	Rapporteur
Pr Valery HEDOUIN	Université de Lille	Rapporteur
Pr Cristina CATTANEO	Université de Milan	Examinateur
Pr Pascal ADALIAN	AMU/ADES	Examinateur
Pr Marie Dominique PIERCECCHI MARTI	AMU/ADES	Directeur de thèse
Dr Lionel THOLLON	AMU/ADES	Co-Directeur de thèse

Résumé

Etablir le lien de causalité entre des violences physiques et le décès d'un individu est une problématique récurrente de la pratique médico-légale. Une grande partie des situations de violence ne pose pas de difficulté au médecin légiste. Toutefois, cette causalité n'est parfois que supposée sur un continuum temporel entre des faits de violence et le décès sans démontrer le mécanisme lésionnel. Cette situation peut provenir de l'absence de traduction anatomique de ce mécanisme lésionnel, illustrée par des cas de décès secondaires à des impacts faciaux, observés à l'institut médico legal où seules des lesions histologiques étaient objectivées.

Pour répondre à cette problématique, un premier modèle par éléments finis de tête a été réalisé initialement afin d'étudier la transmission des forces au crâne à partir d'un impact mandibulaire. Nous avons observé une diminution des transmissions des efforts à la base du crâne suite à un impact mandibulaire ainsi que des contraintes de Von Mises au niveau du tronc cérébral notamement. Ce modèle a été amélioré par l'ajout d'un cou et de la moelle épinière cervicale afin d'étudier plus précisemment les contraintes au niveau du tronc cérébral, lors d'impacts mandibulaires type uppercut ou antéro posterieur, des contraintes au niveau de la jonction cérébro spinale ainsi qu'une hyperextension de la moelle sont observées.

Ces mécanismes lésionnels mettant en jeu des contraintes dans la zone des pédoncules cérébraux, sont en accord avec nos résultats de simulations numériques par éléments finis. Ce niveau de contrainte observé, très proche de la valeur seuil traduisant des lésions cérébrales, laisse présager de possibles lésions axonales.

Les paramètres biologiques variant selon chaque individu, la modélisation numérique permet de les moduler à l'infini (forme de mandibule, dentition…) pour une approche réaliste d'applications médico-légales.

Mots clés : Modèle en éléments finis, Lésions cérébro spinales, Transmission des forces, Tronc cérébral, Impact mandibulaire.

Abstract

Establishing the relationship between the death of an individual and a violent event is a common practice in forensic science. Many cases of death by violence are relatively obvious for the medical examiner. However, the process of identifying the causes of the death is sometimes solely based on the time-event continuum between the violence act and the death without confirmation of the injury mechanisms. This situation may be the case when anatomical signs of the injury mechanisms are absent, illustrated by cases of death secondary to facial impact, observed at the Institut médico legal, where only histological lesions were objectified.

To answer this problematic, a first finite element model of head was realized initially to study the forces transmission to the skull starting from a mandibular impact. We observed a decrease of the efforts transmissions at the skull base following a mandibular impact as well as constraints of Von Mises at the level of the brainstem in particular. This model was enhanced by the addition of a neck and cervical spinal cord to more accurately study brainstem strains, uppercut or anteroposterior mandibular impact, cerebral junction and hyperextension of the cord are observed.

These lesional mechanisms involving stresses in the area of the cerebral peduncles, are in agreement with our results of numerical simulations by finite elements. This level of stress observed, very close to the threshold value reflecting brain lesions, suggests possible axonal lesions.

Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (jaw shape, dentition ...) for a realistic approach of forensic applications.

Keywords: Finite element model, Cerebrospinal injury, Force transmission, Brainstem, Mandibular impact.

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Partie I

Généralités des traumatismes cranio-faciaux

1. Contexte

Problématique des traumatismes cranio-faciaux

Etablir le lien de causalité entre des violences physiques et le décès d'un individu est une problématique récurrente de la pratique médico-légale. Une grande partie des situations de violence ne pose pas de difficulté au médecin légiste car les lésions létales s'expriment par un mécanisme hémorragique induisant le décès soit par déplétion sanguine, soit par compression d'un organe de voisinage. Toutefois, cette causalité n'est parfois que supposée sur un continuum temporel entre des faits de violence et le décès sans démontrer le mécanisme lésionnel. Cette situation peut provenir de l'absence de traduction anatomique de ce mécanisme lésionnel. C'est le cas des décès par mécanisme dit réflexe c'est-à-dire faisant intervenir une conduction nerveuse (décès par compression des glomus carotidiens, par impact thoracique, par compression des globes oculaires…) [1]. Cette situation peut aussi provenir de l'impossibilité d'identifier les lésions par décès rapide, c'est le cas des lésions du système nerveux central (dommage axonal). Dans cette dernière situation, il est formulé l'hypothèse de la transmission des efforts au cerveau.

Pour répondre à cette problématique, depuis quelques années, la modélisation par éléments finis du crâne a permis de réaliser des simulations numériques avec la possibilité de réaliser des études paramétriques [2–6].

Dans le service de médecine légale de Marseille, des décès secondaires à un impact facial, ont été rapportés, où seules des lésions cérébrales histologiques étaient retrouvées au niveau du tronc cérébral. L'équipe de Generalli et al[7], a étudié les conditions d'émergence des lésions axonales

diffuses par des tests effectués sur de grands singes. Elle a conclu que les conséquences fonctionnelles secondaires à la perte de connaissance étaient directement liées à la destruction des axones à l'impact. D'autres études (Besenski et al[8], Povlishock et al[9], Sahuquillo et al[10] et Buki et al[11]) ont démontré que la fonction des axones pouvait être altérée même s'ils n'étaient pas sectionnés. Ces lésions d'étirement sont retrouvées dans les zones de faibles résistances axonales : les zones de transitions cérébrales entre la substance grise et la substance blanche, la substance blanche péri ventriculaire, le corps calleux et à un haut degré de sévérité, le mésencéphale dorsal.

En 2005, Belingardi et al démontrent ainsi le rôle de pivot du tronc cérébral lors d'un impact facial et son altération importante lors de mouvement à type de cisaillement [3]. Sans fracture, le mouvement de la tête à l'impact peut à lui seul causer un dommage cérébral. Différents degrés de dommages sont possibles, de la simple paralysie locomotrice transitoire locale à diverses lésions parenchymateuses plus ou moins étendues. Ces effets d'inertie peuvent être observés lorsque la tête est secouée violemment sans impact direct (notamment en hyperextension) : Exemple, le conducteur subissant un coup du lapin lors d'un impact du véhicule par l'arrière, le rugbyman ou le footballeur poussé par derrière, le boxeur recevant un uppercut ou encore avec un impact subi par une tête casquée. [12,13].

Dans le secteur automobile, l'évaluation des critères de blessures de la tête par le HIC (Head Injury Criterion) est réalisée par l'utilisation de mannequins de chocs afin de prédire les risques lésionnels des usagers dans les transports, la réglementation, conditionnant la mise sur le marché des véhicules. Cependant, la partie faciale affectée à ces mannequins n'est ni géométriquement, ni mécaniquement représentative de la face humaine. De plus, le *Head Injury Criterion* (HIC), basé sur l'accélération linéaire de la tête est controversée et n'apparaît pas comme un bon critère d'évaluation des risques de fractures du crâne et de la face.

Si ces essais expérimentaux permettent un premier niveau de compréhension des mécanismes lésionnels mis en jeu, ils sont coûteux en temps et en argent et ne permettent pas de faire des études paramétriques.

Pour répondre à cette problématique, depuis quelques années, la modélisation par éléments finis du crâne a permis de réaliser des simulations numériques avec la possibilité de réaliser des études paramétriques sur les conditions de choc mais également de tenir compte de la variabilité biologique [2–6].

Ainsi, l'utilisation de ces modèles présente plusieurs avantages comme la possibilité d'une représentation biofidèle de la géométrie de la face ainsi que celle de simuler à l'infini, et à un moindre coût, des conditions d'impact diverses.

L'amélioration de ces modèles en éléments finis pour la prédiction des risques de blessures, lors d'un impact sur la face, nécessite une modélisation plus biofidèle de cette région, d'une part pour prédire les risques de blessures spécifiques à la face et d'autre part, pour évaluer correctement les risques de lésions du contenu intracrânien.

L'objectif de ce travail de thèse est de comprendre les mécanismes lésionnels mis en jeu lors d'un impact facial pouvant aboutir à une perte de connaissance voire au décès d'une personne suite à la propagation de l'onde de choc.

Afin de répondre à cet objectif, nous faisons tout d'abord un rappel anatomique, puis une revue de la littérature concernant les lésions.

Dans une seconde partie, nous présentons le développement du modèle complet de tête numérique par éléments finis, étudiant tout d'abord la transmission des efforts au crâne puis au cerveau jusqu'à

la moelle épinière, de la procédure d'acquisition numérique en éléments finis jusqu'à la validation mais aussi l'interprétation des mécanismes lésionnels.

Enfin, dans une troisième partie, nous présenterons les cas médico-légaux qui nous ont amenés à la réalisation de ce modèle.

2. Anatomie crâniofaciale

a.Structure osseuse

Le squelette céphalique se divise en deux parties : le crâne et la face qui sont solidement liés entre eux et, seule, la mandibule reste mobile.

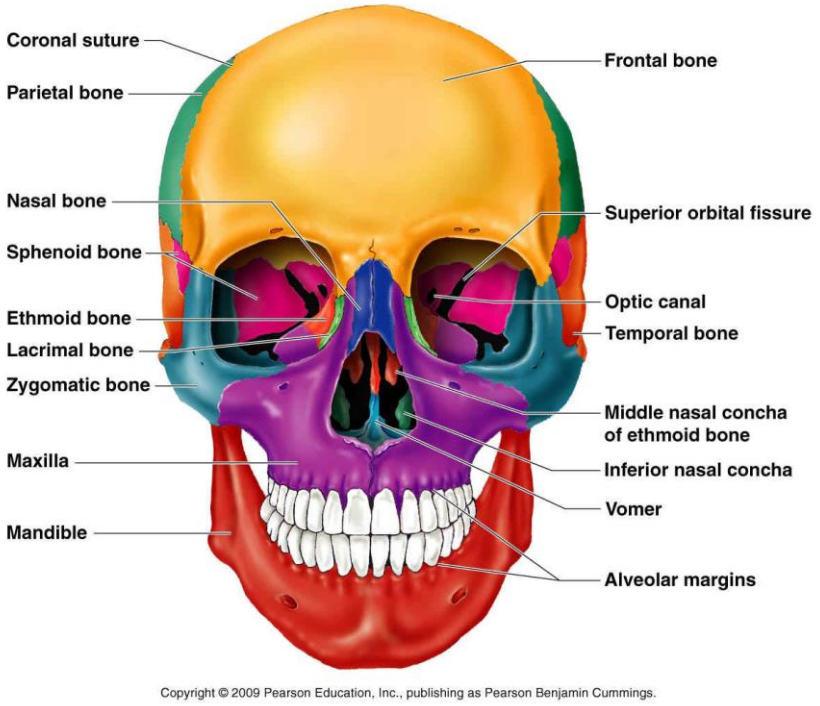


Figure 1 : Squelette céphalique vu de face

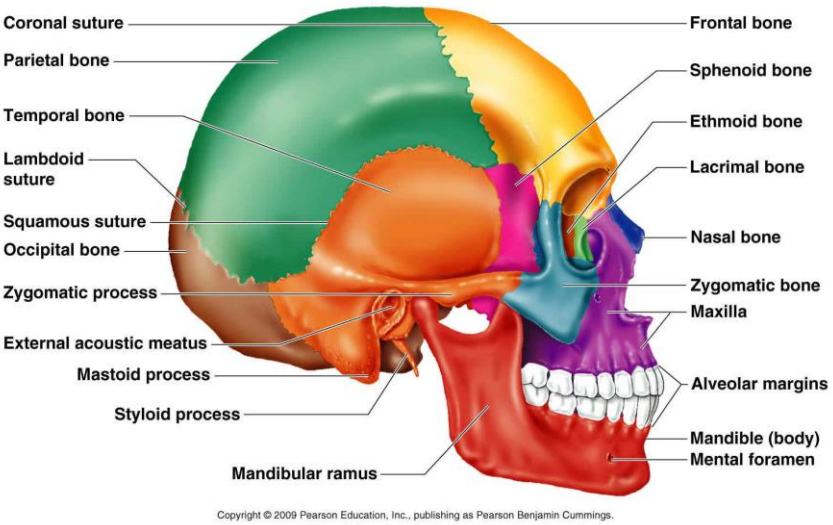


Figure 2 : Squelette céphalique vu de profil

1. *Architecture interne des os du crâne et de la face*

Le tissus osseux est constitué de lamelles de 3 à 7 microns, parallèles entre elles et aux fibres de collagène qu'elles contiennent. Cette structure lamellaire caractérise l'os secondaire et se trouve aussi bien dans l'os compact haversien que dans l'os spongieux trabéculaire.

L'os compact haversien, ou os cortical (Figure 3), est très dense; il est formé à partir d'unités élémentaires juxtaposées (ostéons). Ces ostéons, cylindriques et à grand axe parallèle à celui de l'os, sont formés de lamelles osseuses concentriques à un canal central vasculaire (canal de Havers). Sa répartition correspond à la schématisation des piliers à la face.

L'os spongieux trabéculaire est formé de travées séparées par du tissu conjonctivo-adipeux et vasculaire. La structure globale est une structure aréolaire tridimensionnelle. C'est typiquement l'os de la voûte palatine ou le diploë (Figure 3).

L'os papyracé représente une particularité de certains os plats où le spongieux fait défaut ne laissant qu'une seule lame mince d'os cortical, parfois même translucide, dont les qualités mécaniques sont moindres.

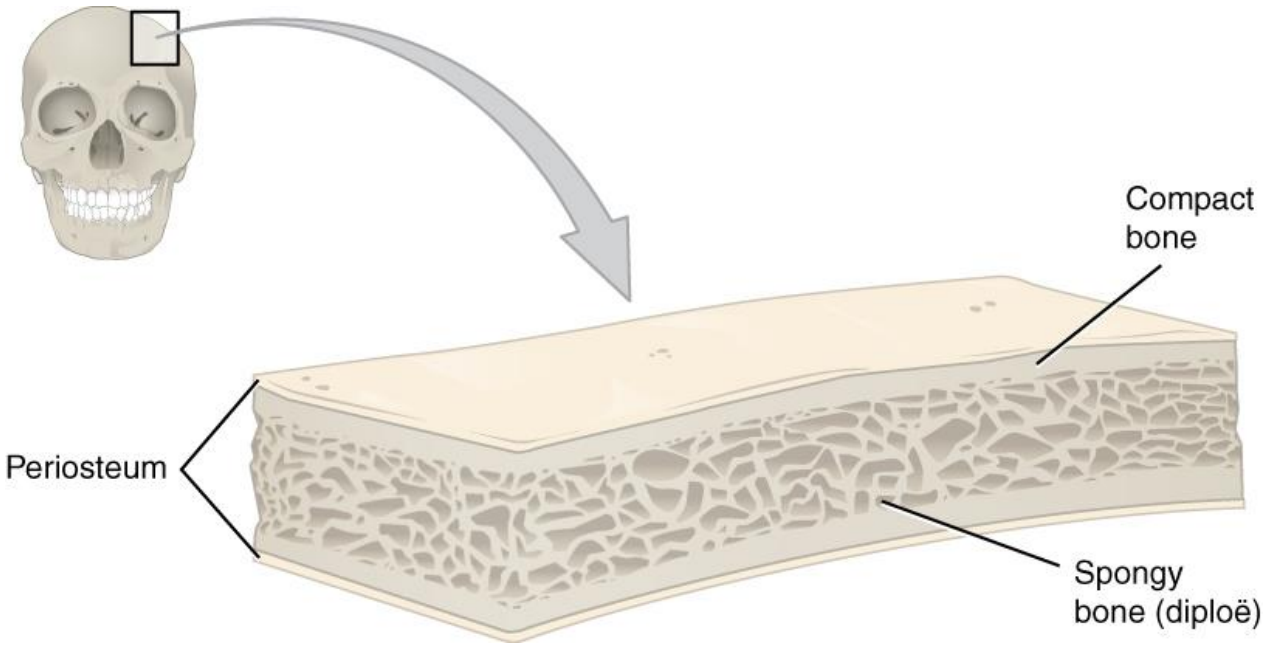


Figure 3 : Eléments constitutifs de l'os crânien (calvaria)

2. Le crâne

Le crâne (ou neuro-crâne) est une enveloppe osseuse qui contient et protège l'encéphale. Il est constitué de deux parties : une base (la base du crâne) et une voûte (la calvaria).

- *Systématisation biomécanique*

La calvaria est renforcée par des arcs longitudinaux et transversaux (Figure 4).

Les arcs longitudinaux sont :

- L'arc fronto-occipital impair et médian,

- L'arc latéral supérieur, pair et symétrique, qui unit le processus frontal de l'os zygomatique (apophyse orbitaire externe) au processus mastoïde,

- L'arc latéral inférieur (concentrique au précédent), pair et symétrique, joignant le tubercule sphénoïdal de la grande aile à la racine du processus zygomatique du temporal.

Les arcs transversaux sont :

- dans la région de la nuque, deux arcs semi-circulaires qui suivent les lignes nuchales supérieures et inférieures (lignes occipitales externes),

- en avant, un arc qui emprunte les bords supra- orbitaires (arcades orbitaires).

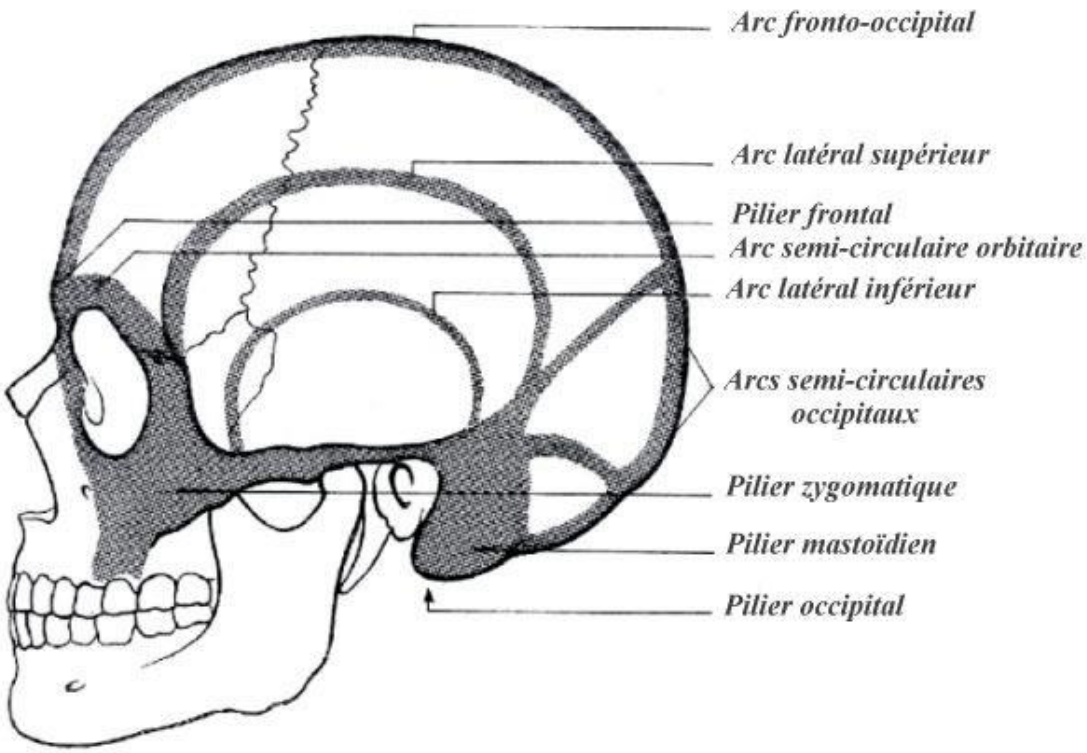


Figure 4 : Arcs de renforcement de la calvaria [14]

3. La base du crâne

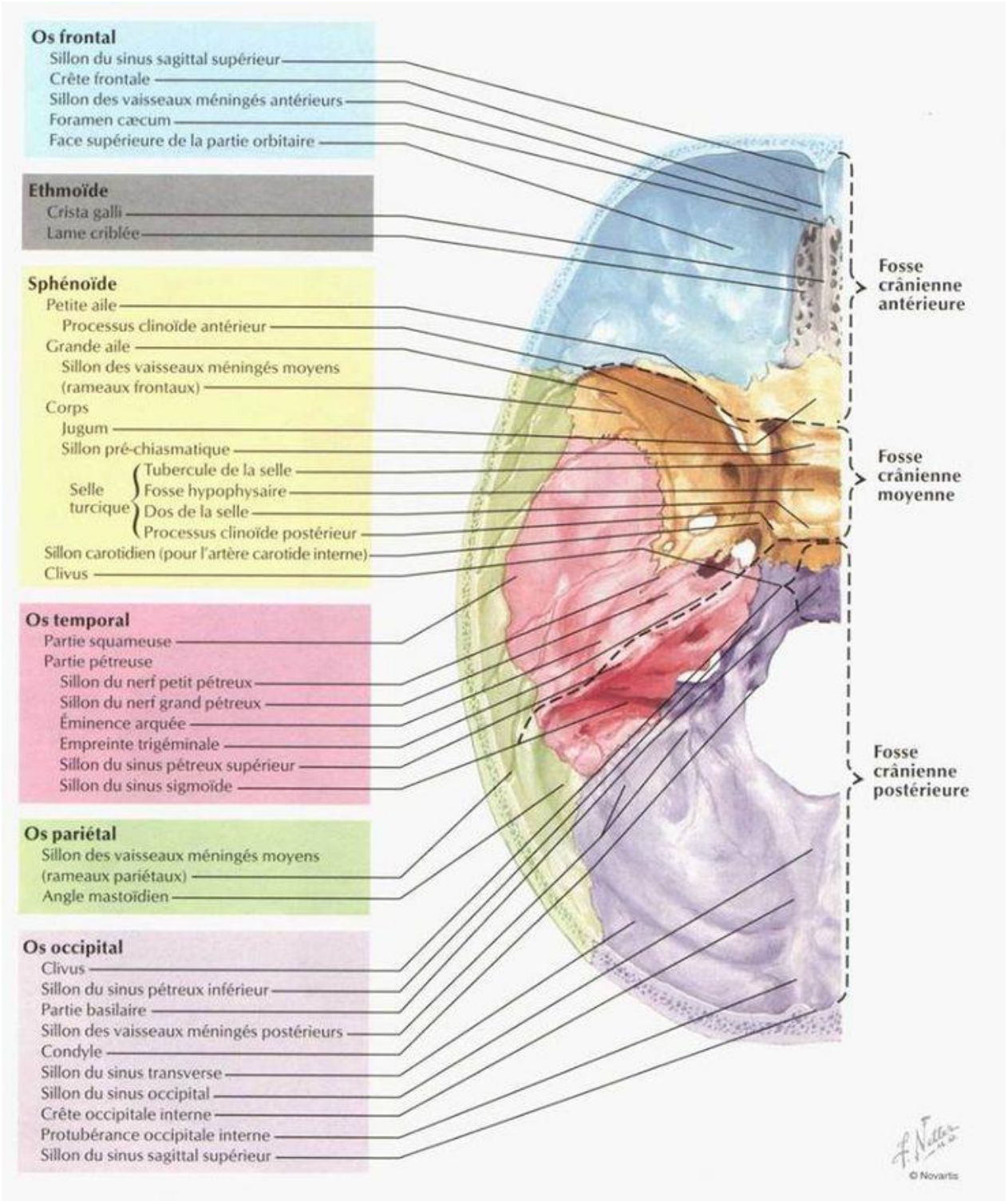


Figure 5 : La base du crâne : face externe [15]

- *Systématisation biomécanique*

La base du crâne présente des systèmes de renforcements (Figure 6) :

- Une crête fronto-ethmoïdale, médiane, impaire, de direction antéropostérieure. Elle se prolonge vers l'arrière en passant en pont sur le corps du sphénoïde, puis, elle se divise en deux, passant par les condyles occipitaux et entourant le foramen magnum (trou occipital), pour se terminer sur la ligne médiane au niveau de la protubérance occipitale interne.

- Un système sphéno-frontal antérieur, à direction transversale et se terminant latéralement sur les piliers zygomatiques [FER86]. Ce système comprend deux éléments; l'un emprunte le bord antérieur de la petite aile du sphénoïde, l'autre emprunte son bord postérieur.

- Un système de renforcement postérieur, avec deux renforts latéraux obliques de chaque côté: renforts pétreux, d'une part, qui passent par les bords antérieurs des pyramides pétro-tympaniques jusqu'aux piliers mastoïdiens (piliers dont l'existence est controversée [FER86]); renforts occipitaux, d'autre part, se dirigeant, à partir des condyles occipitaux, par les bords postérieurs des pyramides pétro-tympaniques, vers les sillons des sinus transverses.

Elle présente néanmoins des zones de faiblesse :

- En avant, au niveau des voûtes orbitaires.

- Puis, au niveau des grandes ailes du sphénoïde.

- Enfin, en arrière, au niveau des fosses cérébelleuses.

Le corps du sphénoïde, où convergent les différents systèmes, représente le centre de résistance du crâne.

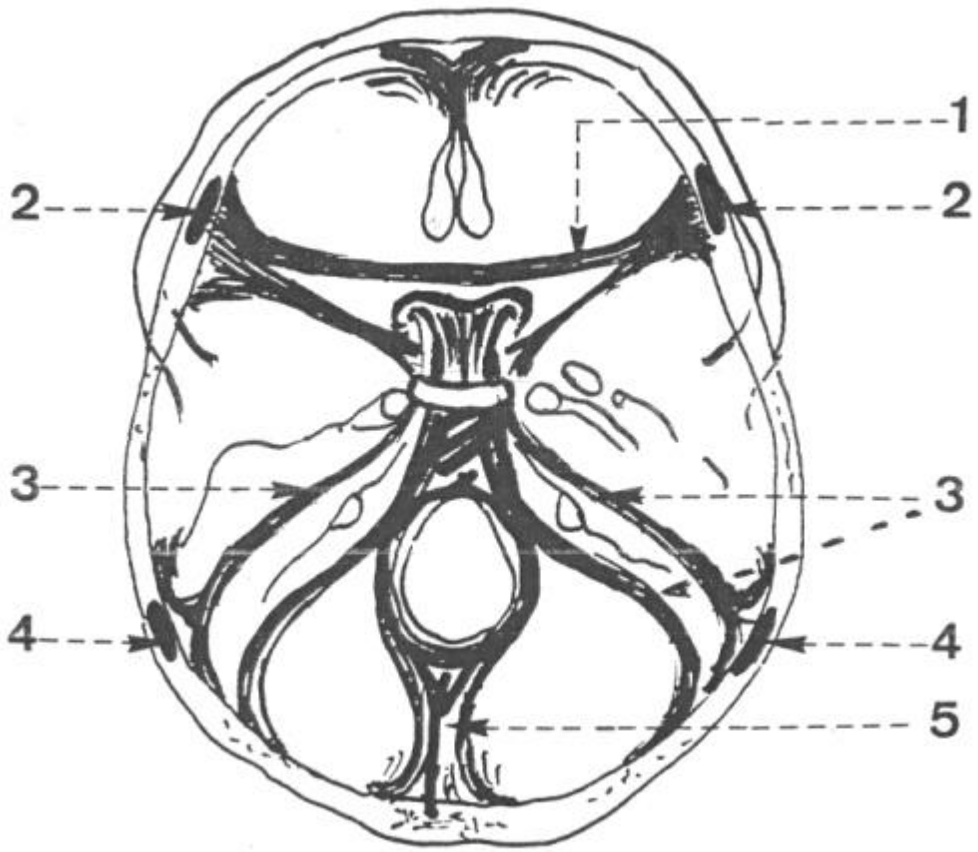


Figure 6 : Systématisation de la base du crâne [16] 1 et 2 : Poutres sphénofrontales. 3 : Poutre pétreuse. 4 : Piliers mastoïdiens. 5 : Arc fronto-occipital ou poutre occipitale postérieure.

4. La face

La face est un complexe neuro-sensoriel dont le rôle est considérable dans la perception, grâce aux organes des sens abrités au sein des cavités faciales qui sont toutes ouvertes vers l'avant : orbites, cavité nasale (fosses nasales) et cavité orale (cavité buccale). Le rôle joué par la face dans la communication et dans l'esthétique est primordial, et, à notre époque, toute atteinte à ce niveau aura des répercussions extrêmement importantes.

- *Anatomie morphologique*

La face est formée de quatorze os avec (Figure 1) :

- six os pairs et symétriques: dans le sens médio latéral et de haut en bas, on trouve l'os nasal, l'os lacrymal (entre les deux s'interpose le processus frontal de l'os maxillaire) et l'os zygomatique (os malaire); puis, le cornet nasal inférieur, l'os palatin et l'os maxillaire,

- deux os impairs et médian : en haut, le vomer ; en bas, formant à elle seule l'étage inférieur de la face, la mandibule.

Les cavités faciales (cavités «ouvertes», car directement en communication avec l'extérieur) sont à rattacher aux organes des sens avec :

Les sinus paranasaux sont des cavités profondes, paires, creusées dans les os. Ils sont en communication avec la cavité nasale (et, par son intermédiaire, avec l'extérieur). On distingue de chaque côté quatre sinus (Figure 7) :

Les os de la face présentent essentiellement deux types de structures : soit de l'os cortical uniquement (os nasal, os lacrymal, cornet nasal inférieur, vomer), soit de l'os cortical entourant de l'os spongieux (os maxillaire, os palatin, os zygomatique, mandibule).

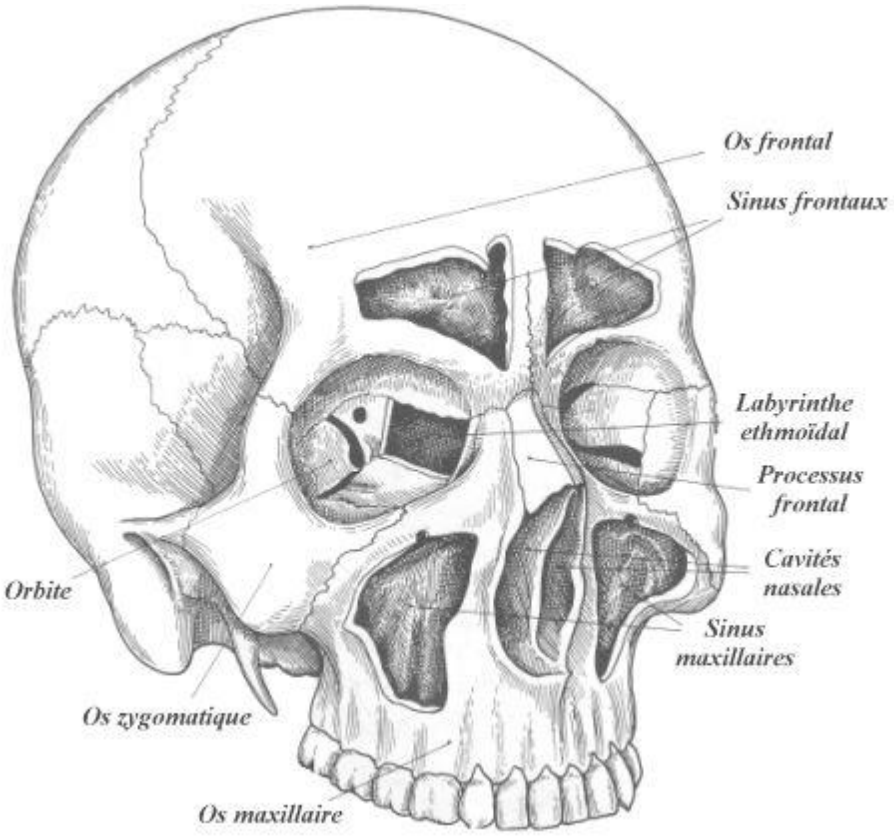


Figure 7 : Sinus et cavités faciales

- *Systématisation biomécanique*

La face possède des renforts verticaux qui sont (Figure 8) :

- Deux piliers antérieurs, naso-maxillaires ou canins. Issus de la canine supérieure, ils suivent le bord antérieur du maxillaire jusque dans le processus frontal et se terminent dans le cintre frontal sus-orbitaire.

- Deux piliers latéraux, zygomato-maxillaires. Issus de la première molaire, ils passent par le bord inférieur du processus zygomatique, ils se poursuivent dans le corps de l'os zygomatique jusque dans son processus frontal et se terminent dans le cintre frontal sus-orbitaire.

- Deux piliers postérieurs, ptérygo-sphéno-frontaux. Issus des processus ptérygoïdes, ils passent par les grandes ailes du sphénoïde et se terminent dans le cintre frontal sus-orbitaire.

- Deux piliers postéro latéraux, représentés par les branches de la mandibule.

La face possède également des renforcements horizontaux :

- Le cintre frontal sus-orbitaire.

- Deux renforcements (entretoises) sont issus des piliers latéraux (zygomato-maxillaires) au niveau de l'os zygomatique. Un renforcement sous-orbitaire rejoint le pilier antérieur homologue. Un renforcement zygomatique passe par les processus temporal puis zygomatique, se terminant en éventail à la racine temporale de l'arcade zygomatique.

- Le corps de la mandibule (arc antérieur mandibulaire).

Un système de caissons, renforcés par des cadres, traduit une organisation particulière des cavités faciales [16]:

- La cavité nasale, caisson de rupture, agit comme un amortisseur.

- Les orbites et les sinus maxillaires s'organisent, de chaque côté, en un caisson double stabilisé latéralement par les arcades zygomatiques.

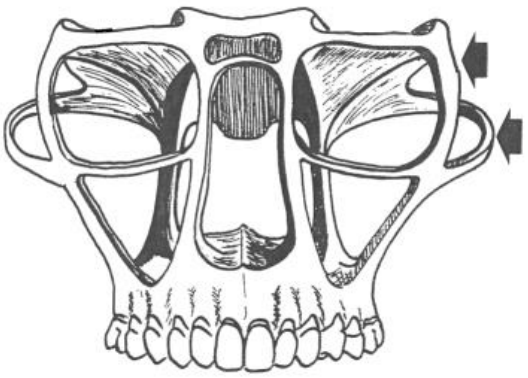
- Les cellules du labyrinthe ethmoïdal se présentent comme des raidisseurs de la cavité nasale médialement et des orbites et sinus maxillaires latéralement.

Les pare-chocs, zones saillantes de la face particulièrement exposées aux traumatismes, sont :

- Les pare-chocs latéraux, de grande résistance, sont représentés par les os zygomatiques et par les bords supra- orbitaires.

- Les pare-chocs médians, fragiles, sont représentés, de haut en bas, par la région glabellaire, la pyramide nasale, la région prémaxillaire et la protubérance mentonnière.

Cette systématisation biomécanique est une manière d'expliquer des lésions du squelette du crâne et de la face, par des hypothèses données par l'architecture.



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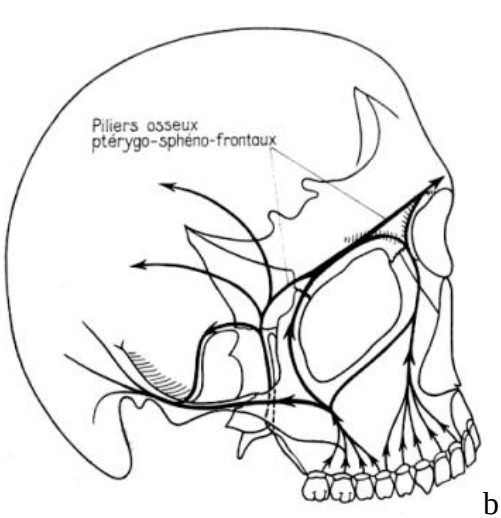

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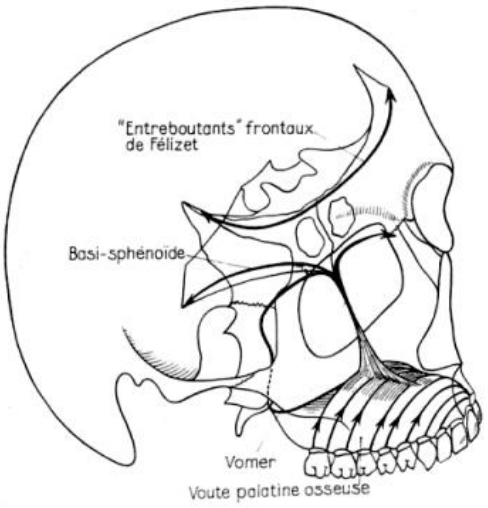

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Figure 8 : Systématisation biomécanique de la face

a. Les renforcements et caissons [16] (selon Deffez).

b. Distribution des efforts à travers les piliers verticaux [17].

c. Distribution des efforts à travers les piliers horizontaux [17].

b. Le cerveau et les méninges

1. Le cerveau

C'est l'étage le plus élevé dans la hiérarchie fonctionnelle du système nerveux central. Il est spécialement développé chez l'homme. Son poids moyen est de 1400 à 1800 grammes.

Le cerveau est placé dans la boîte crânienne où il repose sur la base du crâne et il est recouvert par la voûte.

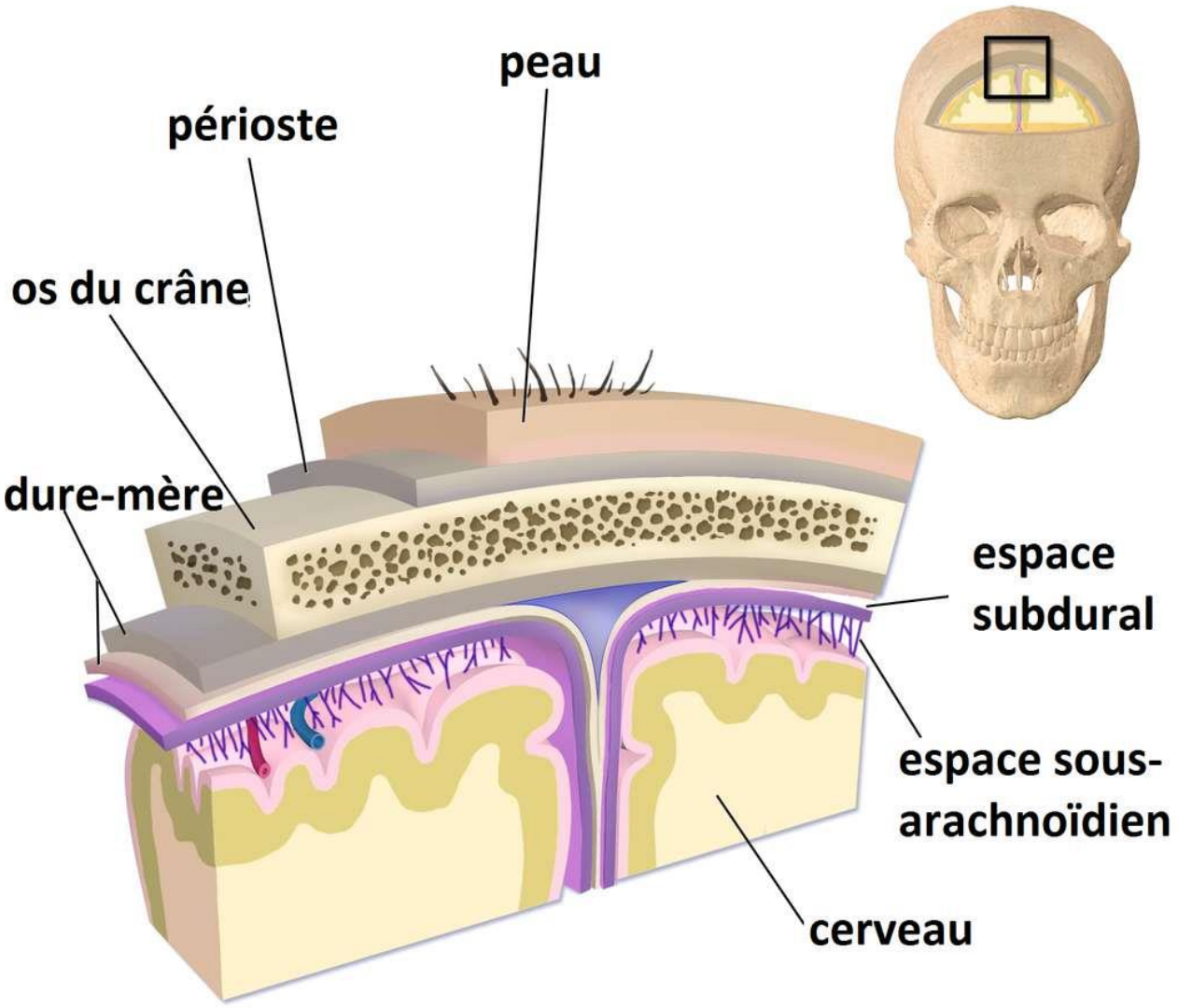

Figure 9 : Détail des couches cutanées, osseuses, méningées et cérébrales de la région frontale cranio-cérébrale.

Une toile fibreuse très épaisse appelée dure-mère tapisse la face interne du crâne et forme un repli sous le cerveau appelé : tente du cervelet. Elle forme aussi un repli vertico-sagittal entre les deux hémisphères du cerveau constituant une cloison médiane appelée : faux du cerveau. Ainsi se trouvent délimitées deux loges fibreuses : en haut la loge cérébrale qui contient les deux hémisphères du cerveau, en bas la loge cérébelleuse (ou fosse crânienne postérieure) qui contient le cervelet et le tronc cérébral.

2. Les méninges

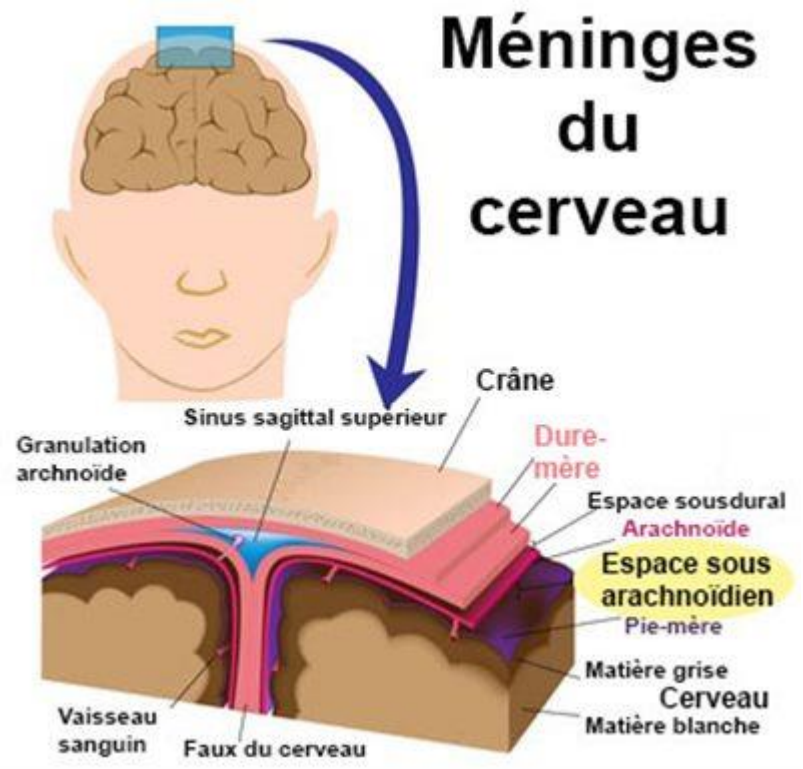

Figure 10 : Les méninges

Le cerveau est recouvert de trois méninges :

* la dure-mère est la méninge la plus épaisse (décrite plus haut).

* l'arachnoïde tapisse la face interne de la dure-mère

* la pie-mère tapisse la surface du cerveau en épousant étroitement les replis, les scissures et les circonvolutions du cerveau.

Entre l'arachnoïde et la pie-mère se trouve l'espace sub-arachnoïdien qui est occupé par le liquide cérébro-spinal.

3. Le liquide cérébrospinal

Le névraxe baigne totalement dans le liquide cérébro-spinal, qui est situé à l'extérieur et à l'intérieur du névraxe.

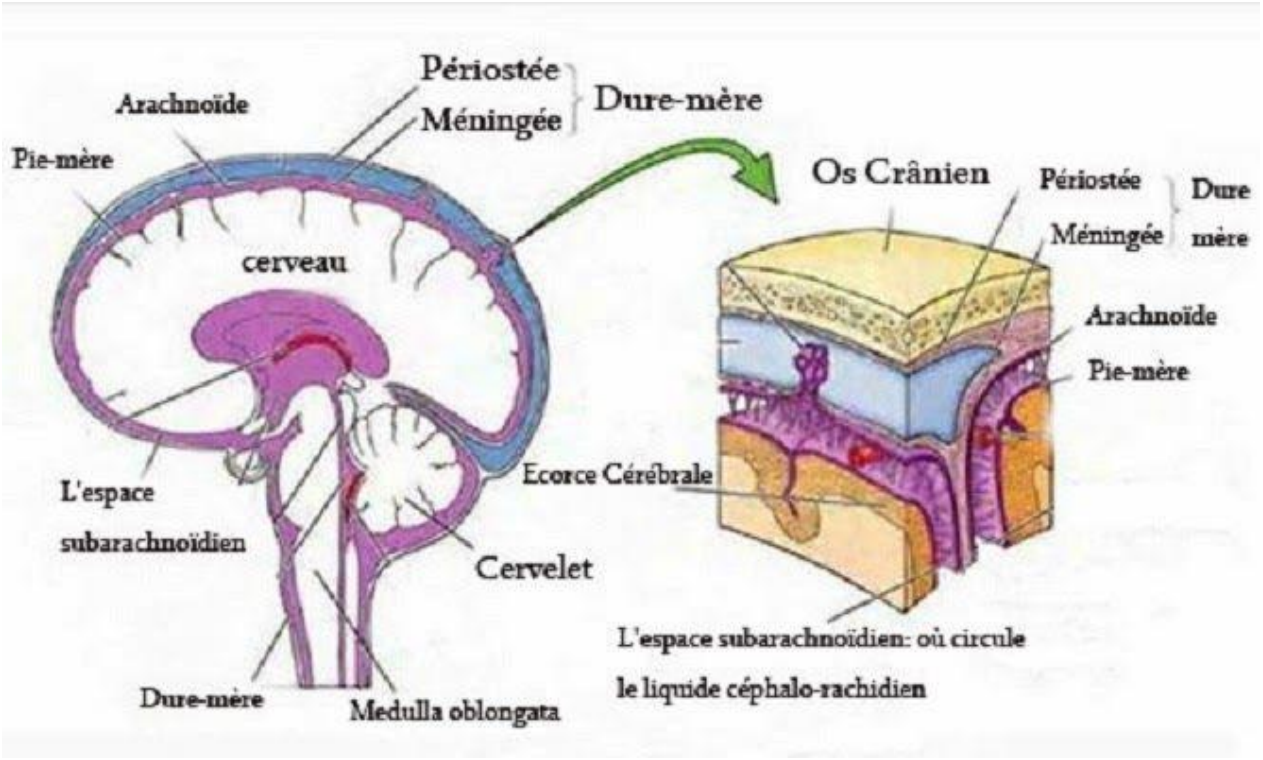

Figure 11 : Visualisation du liquide céphalo-rachidien

Il est sécrété en permanence par les plexus choroïdes, qui sont des formations névrogliques de structure glomérulaire. La filtration dans le sens sang / liquide cérébro-spinal est très sélective. Il existe donc une véritable barrière hémo-méningée qui assure, au système nerveux central, la stabilité de son milieu.

Le liquide cérébro-spinal circule en permanence et il est résorbé par voie veineuse au niveau du secteur périphérique.

Le cerveau et le tronc cérébral possèdent un système de cavité qui contient du liquide cérébrospinal. Le 4ème ventricule appartient au tronc cérébral. Le 3ème ventricule appartient au cerveau. Il est placé profondément sur la ligne médiane, entre les deux couches optiques. Il communique par un orifice étroit (trou de Monro) avec les ventricules latéraux qui sont des cavités placées au sein de chaque hémisphère.

c. Système nerveux et tronc cérébral

1. Tronc cérébral

Le tronc cérébral appartient au système nerveux central, et plus particulièrement à l'encéphale. Il est situé dans la fosse crânienne postérieure, sous le cerveau et en avant du cervelet. Il est structurellement continu avec la moelle épinière, qui commence à la première racine spinale. Le tronc cérébral est relié au cerveau, via les pédoncules cérébraux du mésencéphale, et au cervelet, via les pédoncules cérébelleux supérieurs (mésencéphale), moyens (pont) et inférieurs (moelle allongée). C'est également le lieu d'émergence de dix des douze paires de nerfs crâniens (de la IIIe paire à la XIIe).

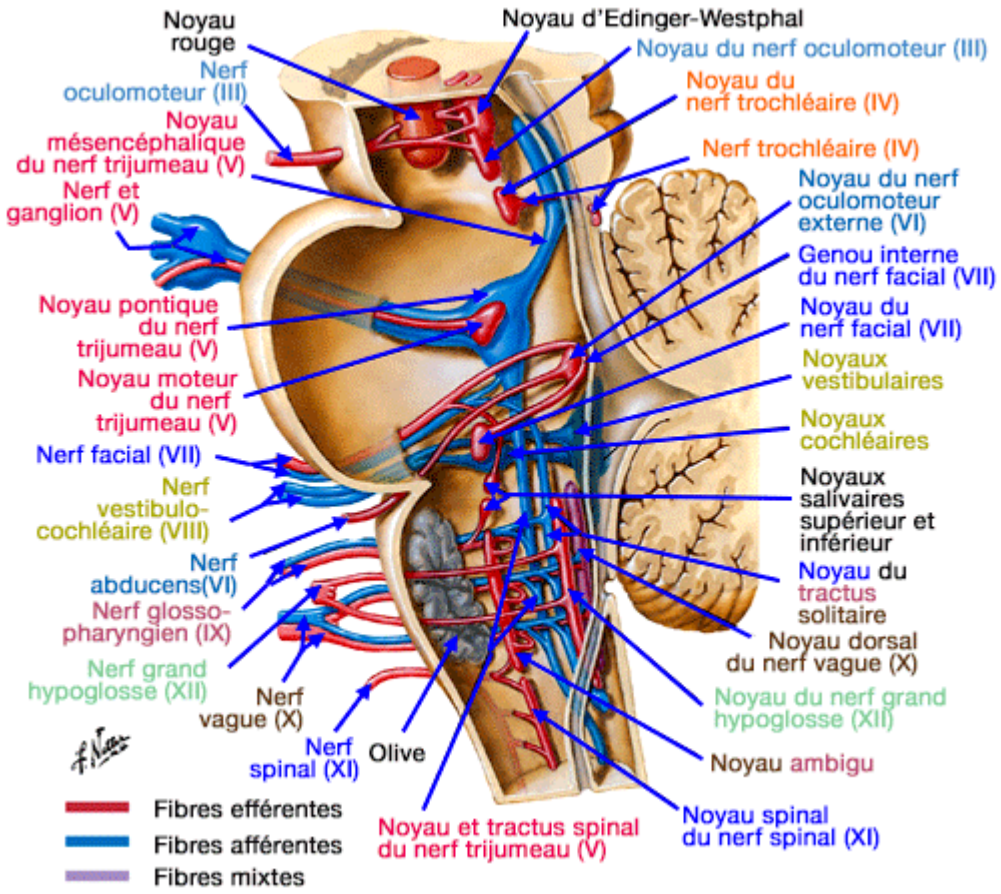

Figure 12 : Noyaux des nerfs crâniens dans le tronc cérébral

Le tronc cérébral est oblique en haut et en avant, et se compose de haut en bas :

- du mésencéphale (pédoncules cérébraux, tegmentum et tectum)
- du Pont
- de la Moelle allongée

Une partie des faces postérieures de la moelle allongée et du pont constitue le plancher du quatrième ventricule.

Le tronc cérébral est responsable de plusieurs fonctions dont la régulation de la respiration et du rythme cardiaque, la localisation des sons, etc. C'est également un centre de passage des voies motrices et sensitives, ainsi qu'un centre de contrôle de la douleur.

La moelle allongée ou bulbe rachidien

La moelle allongée (ou bulbe rachidien) est la partie inférieure du tronc cérébral. Elle est limitée en bas par la moelle épinière (au niveau de la première racine spinale) et en haut par le sillon ponto-médullaire.

Sur sa face médiale, elle se compose de deux cordons ventraux (ou pyramide), séparés par une fissure médiane ventrale (ou sillon médian). Les pyramides contiennent les fibres axonales du tractus cortico-spinal (ou tractus pyramidal), moteur.

La face dorsale présente deux cordons dorsaux, séparés par le sillon dorsal médian. Ces cordons s'écartent en haut pour border le plancher du quatrième ventricule. Ce plancher contient, au niveau de la moelle allongée, les noyaux des nerfs crâniens X, XI et XII.

Le pont

Le pont constitue la portion moyenne du tronc cérébral. Il est limité en bas par le sillon ponto-médullaire et en haut par le sillon ponto-pédonculaire.

Le mésencéphale

Le mésencéphale forme la région supérieure du tronc cérébral, constituant le cerveau moyen. Il est limité en bas par le sillon ponto-pédonculaire et en haut par le tractus optique. Il est constitué de trois parties principales : les deux pédoncules cérébraux, le tegmentum mesencephali (ou calotte du mésencéphale) et le tectum mesencephali (ou toit du mésencéphale).

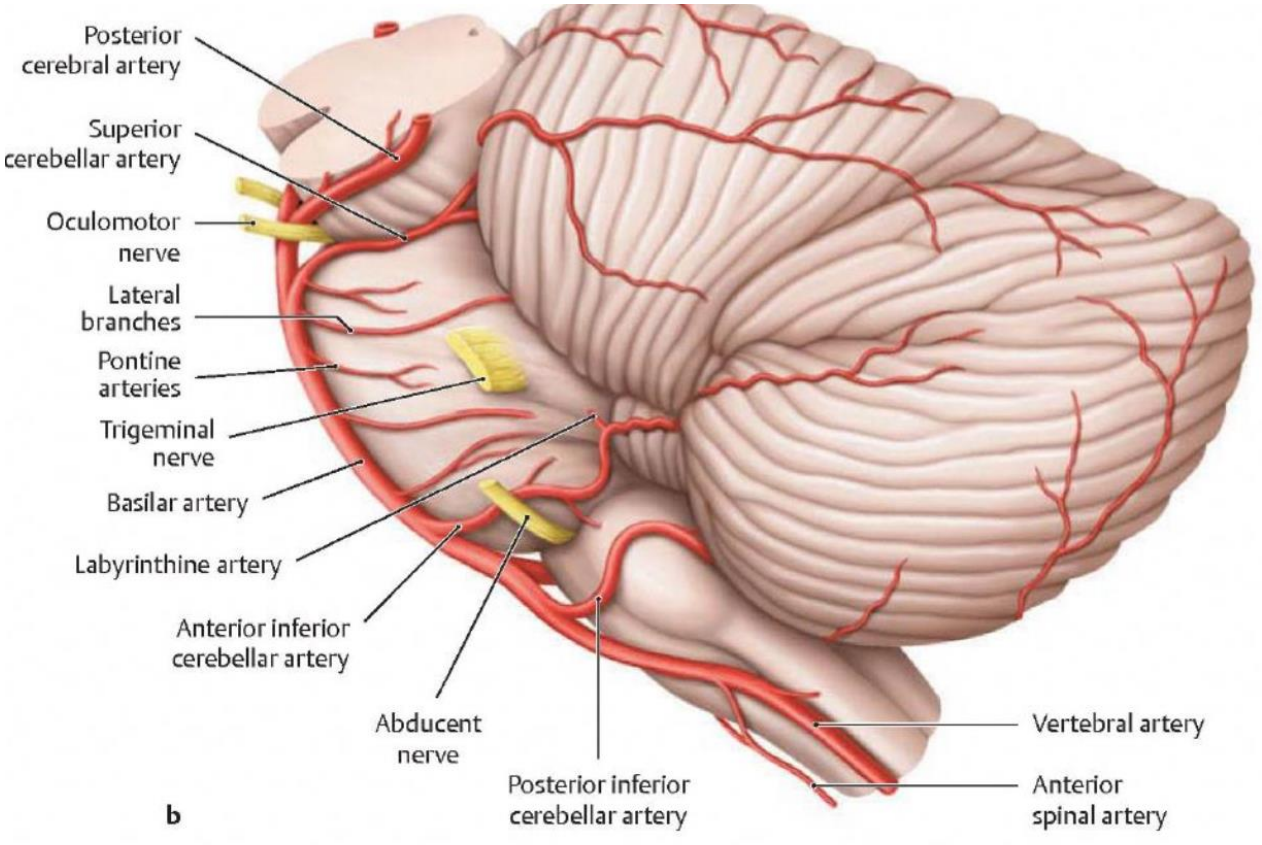

Figure 13 : Morphologie et vascularisation du tronc cérébral.

2.Le nerf vague

Le nerf vague (X), également appelé nerf pneumogastrique, nerf cardio-pneumo-entérique, nerf parasympathique ou nerf cardiaque, est le dixième nerf crânien. C'est une voie très importante de la régulation végétative (digestion, fréquence cardiaque…) mais aussi du contrôle sensorimoteur du larynx et donc de la phonation.

Au sein du système parasympathique, il constitue la principale innervation efférente du cœur. En 1921, Otto Loewi a, le premier, mis en évidence la transmission synaptique en montrant que la stimulation du nerf vague entraînait une sécrétion d'acétylcholine qui induisait un ralentissement du rythme cardiaque.

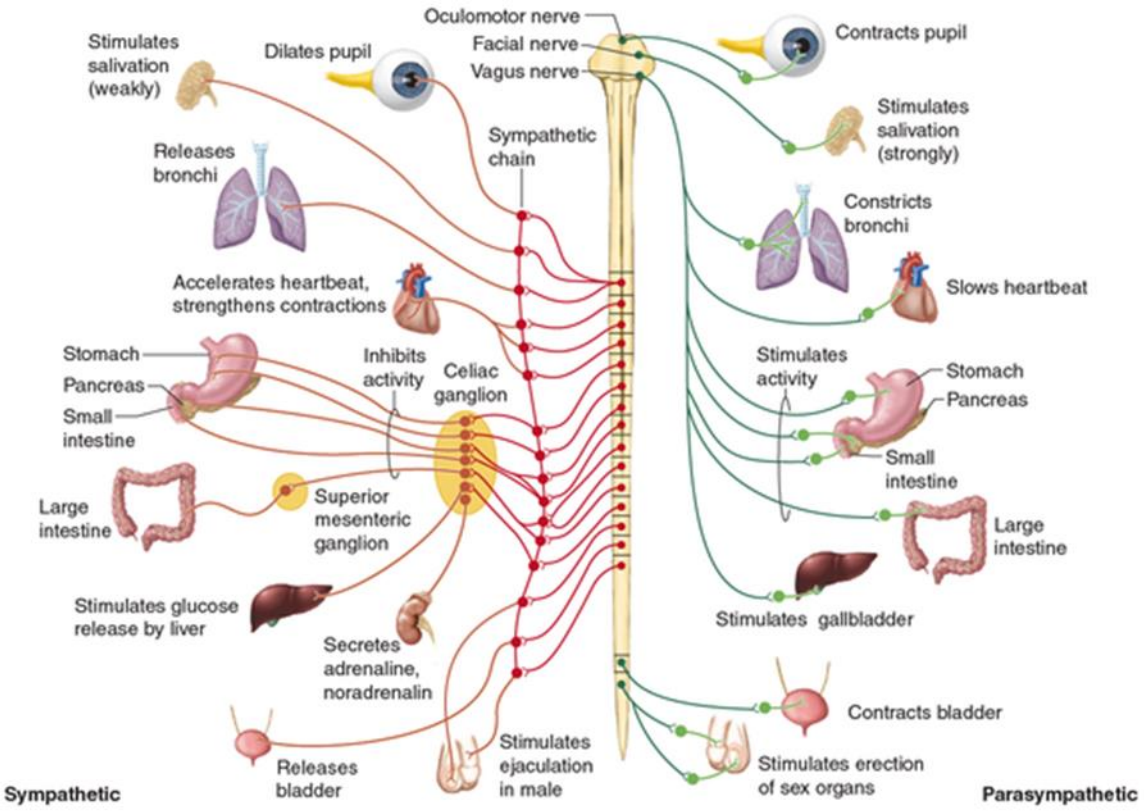

Figure 14 : Mode d'action du système parasympathique et sympathique

3.La perte de connaissance

Le tronc cérébral est constitué d'une multitude de noyaux. La formation réticulaire est une coulée de cellules et de fibres, parsemé de nombreux noyaux dont la substance noire (locus niger) et le noyau rouge sont les plus importants.[18]

Elle s'étend de la moelle épinière au diencéphale, occupant dans le tronc cérébral l'espace situé entre les grandes voies en avant et les noyaux des nerfs crâniens en arrière.

Elle est constituée par un réseau dense de fibres (réticulum) enserrant des groupements cellulaires organisés en agglomérats juxtaposés.

Le système réticulaire ascendant :

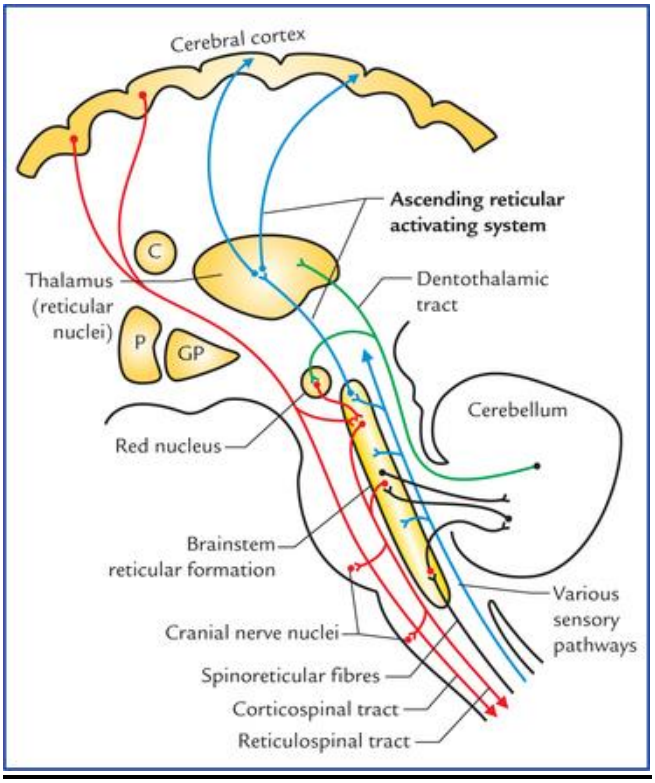

Figure 15 : Le système réticulaire ascendant

Les noyaux médians du raphé bulbo-ponto-mésencéphaliques avec leurs afférences sensitivo-sensorielles et leurs projections sur l'hypothalamus et le cortex constituent le système réticulaire ascendant ou système d'éveil et de sommeil.

Une lésion du système réticulaire ascendant entraine des troubles de la vigilance- éveil ou du sommeil.[18]

Le coma, trouble de la vigilance avec non éveil, est dû à une atteinte des noyaux du raphé médians mésencéphaliques et protubérantiels supérieurs.

Dans les traumatismes crâniens, le coma d'emblée est dû à une contusion du tronc cérébral qui s'écrase sur le clivus et plus particulièrement du mésencéphale qui s'écrase sur le dorsum sellae (section physiologique).[18]

Le système réticulaire végétatif :

Les noyaux dorso bulbaires, tenant sous leur dépendance les grandes fonctions végétatives respiratoire, cardio-vasculaire, digestive, température… constituent le système réticulaire végétatif.

Une lésion du système réticulaire végétatif entraine des troubles des grandes fonctions végétatives. L'orage végétatif de la contusion du tronc cérébral avec troubles respiratoires (ataxie respiratoire avec encombrement), cardiaques (troubles vaso-moteurs, accès hypertensif ou collapsus), digestifs (vomissements, hoquets…) métaboliques, dérèglements thermiques…est dû à une contusion des noyaux dorsaux de la formation réticulaire bulbaire.

Il accompagne la compression bulbaire par engagement des tonsilles (amygdales) cérébelleuses dans l'Hypertension intra crânienne de la fosse postérieure par tumeur par exemple. Il est alors, à ce moment-là, étonnamment pur, sans troubles de la conscience. Une ponction lombaire intempestive dans une tumeur de la fosse postérieure peut l'entrainer. La mort brutale est possible dans l'engagement cérébelleux.[18]

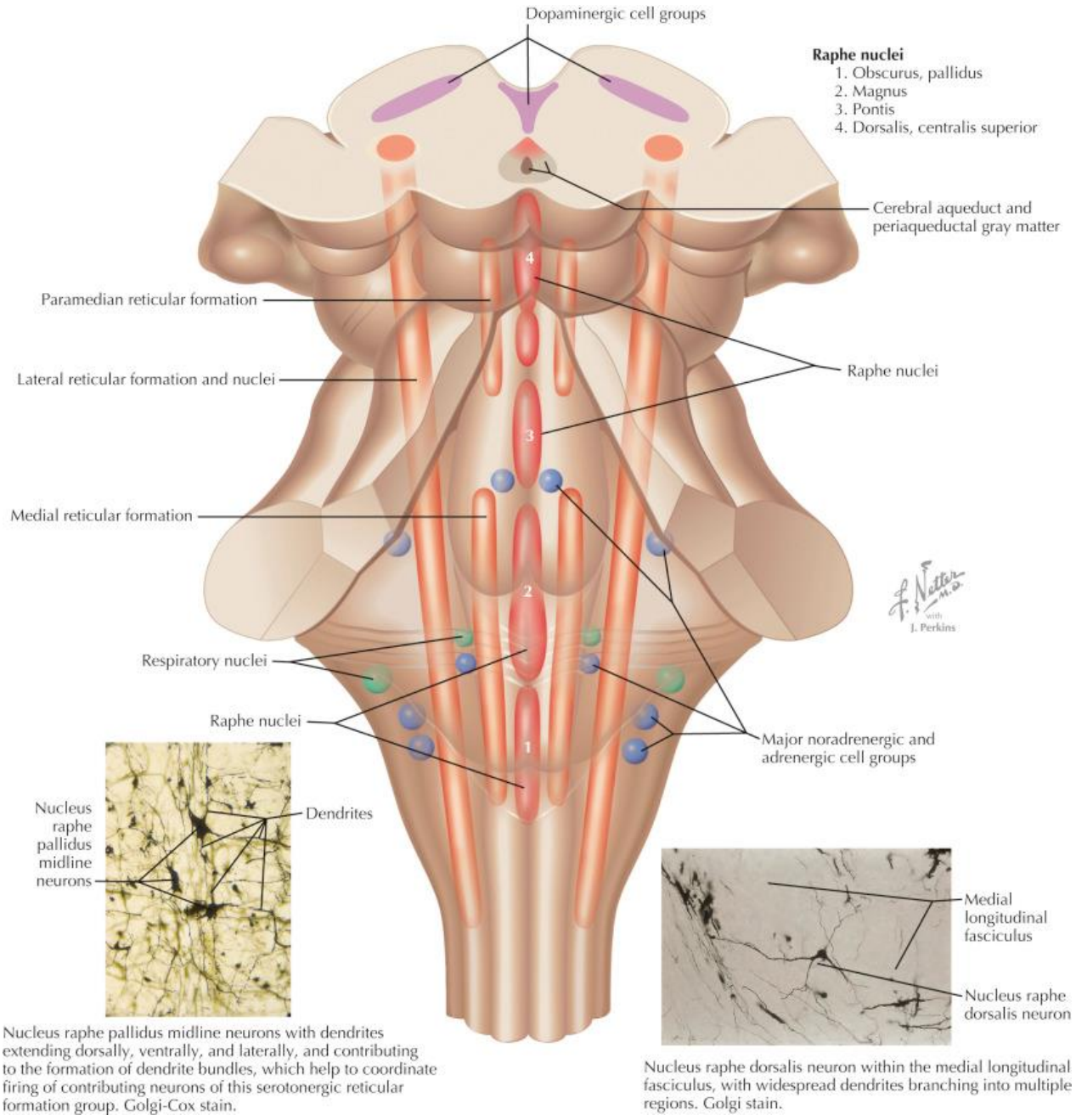

Figure 16 : Noyaux des nerfs crâniens

3. Epidémiologie des traumatismes craniofaciaux

Les **accidents de la voie publique (ou AVP), les chutes, les rixes et les activités sportives** [19] représentent les principales causes de lésions crâniofaciales. De nombreuses études décrivent simplement les lésions faciales, sans les corrélées aux lésions cérébrales potentiellement associées.

Lésions crâniofaciales

Les accidents de la voie publique causent principalement des fractures du crâne (50% des fractures dues aux AVP), tandis que les chutes et les rixes impliquent des lésions moins graves, surtout des fractures du nez (respectivement 47 et 60%) [20]. Les fractures du nez représentent la majorité des cas de fractures crâniofaciales (44%), suivies des fractures du crâne (24%), de la mandibule (15%), de l'os zygomatique (13%), de l'orbite (2%) et du maxillaire (2%).[20].

La plupart des études s'accordent sur deux points : les victimes de rixes sont souvent des hommes (2/3 cas), avec un pic entre 20 et 30 ans et plus de la moitié des patients ont absorbé de l'alcool [20,21].

Les mêmes auteurs précisent que, parmi les fractures crâniofaciales observées, les fractures du nez sont les plus fréquentes (60%), suivies de fractures de l'os zygomatique, et que les lésions des tissus mous concernent le front, le nez, les lèvres et le menton.

Sur la période de 1985 à 2012, 24.4% des fractures maxillo-faciales étaient dues à des violences inter individuelles, 16.7% étaient à l'origine de fractures des condyles et 17% de la symphyse mandibulaire, soit en deuxième et troisième position après les fractures orbito-zygomatiques [22].

Les activités sportives sont à l'origine de 10% de traumatismes faciaux en Europe, alors que relativement rares dans le reste du monde [23]. Selon les données obtenues par Hussain et al. [20,21] et Gaw et al, le football est en cause dans 33 à 47% des blessures crâniofaciales, le rugby dans 14%, puis viennent le squash et le hockey. Dans le cas des activités sportives, 17% des

blessures crâniofaciales sont des fractures : elles touchent le nez et la mandibule. Celles-ci sont essentiellement concernées par le football (73%) et le rugby (18%) [20].

Les traumatismes par mécanisme de faible vélocité, définis comme les chutes de sa hauteur, les sports à vitesse faible (football ou rugby) ou les agressions sont responsables de 56% des fractures de Lefort I. Les traumatismes à haute vélocité, définis comme les chutes de hauteur supérieure à un individu, les sports à haute vitesse (cyclisme, ski…) ou des accidents de véhicule à haute vitesse, sont associés à des Lefort II et III [24–26].

Lésions crâniofaciales et traumatisme cérébral

Le taux d'associations lésions cranio faciales et lésions cérébrales est très variable selon les études allant de 5.4% à 79, 4% voire 86% selon Martin et al et Hayter et al, respectivement[27–30]. Les études de Zandi et al, et Ghosh et al, rapportent qu'environ 20% de leur cohorte présentaient des lésions cérébrales dont 18% associées à des hémorragies cérébrales [31,32]. Ces résultats d'une extrême variance sont dus aux différences de populations étudiées, méthodologies utilisées, recueil des données, etc…

L'une des études de Zandi et al, a montré que 13% de leur cohorte présentaient des lésions cérébrales sans lésions osseuses, uniquement cutanées, que 25% présentait des lésions cérébrales et des fractures crâniennes, essentiellement des fractures de la base suivie des fractures frontales. Seulement 18% des patients qui présentaient des lésions cérébrales, avaient des hémorragies intracrâniennes [31].

Haug et al rapporte que les mandibules sont plus souvent fracturées lorsqu'il y a des lésions faciales cutanées et intracrâniennes alors que les fractures du milieu de la face sont plus fréquemment associées à des lésions intracrâniennes uniquement, s'expliquant par le fait que les structures du milieu de la face sont plus près du cerveau.[33]

Corrélation entre traumatisme facial, fractures mandibulaires et perte de connaissance ?

Dans la littérature, des divergences se retrouvent concernant le rôle protecteur des os de la face et en particulier de la mandibule aux lésions cérébrales [34]. Cependant quelques études descriptives s'accordent sur la dissipation des forces par les fractures mandibulaires laissant passer une énergie résiduelle au cerveau [35].

Traumatisme facial et lésions cervicales

Un taux élevé de lésions cervicales spinales est décrit chez des patients présentant des lésions de la face et intracrâniennes par rapport aux patients présentant des lésions faciales seules[31,36]. Les impacts mandibulaires entrainent un violent mouvement de la tête, à l'origine de dislocations cranio cervicales instantanées qui peuvent être à l'origine de lésions indirectes du tronc cérébral, le plus souvent ponto médullaires, parce que la jonction ponto-médullaire est anatomiquement la partie la plus fine du tronc cérébral et donc la plus faible. [37]. Comme décrit dans notre précèdent article, lors d'un impact mandibulaire, l'énergie cinétique est transmise de la mandibule aux articulations temporo mandibulaires puis à la base du crâne et au cerveau [38]. Selon Zivkovic et al, dans les cas d'impacts mandibulaires, la majorité ne sont pas associés à des fractures de la base du crâne et quelques-unes sont associées à des fractures isolées en anneau (hinge, ring).et près de la moitié sont associées à des lésions ponto médullaires[37]. Dans les deux situations nous avons une décroissance de la transmission de la force d'impact. Cela signifie que l'énergie est suffisante pour produire une lésion ponto médullaire mais insuffisante pour produire une fracture de la base du crâne.

4. Critères lésionnels

Caractérisation de la sévérité d'une blessure crâniofaciale :

a. Description générale de l'Abbreviate Injury Scale (AIS)

Une blessure en elle-même n'est pas un bon indicateur des risques encourus pour le sujet qui la subit. Il faut tenir compte de l'influence du type de blessure, de son étendue, de sa localisation et de l'état physiologique général du blessé. La sévérité d'une lésion se mesure en termes d'atteintes à l'intégrité physique et fonctionnelle de l'individu en distinguant les courts, moyens et longs termes.

L'Abbreviate Injury Scale (AIS), introduit en 1971, établit un code de caractérisation de la sévérité d'une lésion, en fonction de paramètres tels que la menace pour la vie, les dommages permanents, et la durée des soins prodigués à l'individu. Plusieurs révisions de ce code ont été proposées, la dernière datant de 2015. L'AIS utilise 7 chiffres pour décrire la blessure et son niveau de gravité: Le niveau de sévérité (ou gravité AIS) est donné sur une échelle de 0 à 6.

Niveau de gravité des lésions (AIS)

0 Pas de lésion

1 Lésion mineure

2 Lésion modérée

3 Lésion sérieuse qui ne met pas la vie en danger

4 Lésion sévère qui met la vie en danger

5 Lésion critique à survie incertaine

6 Lésion fatale

La combinaison des AIS des organes atteints est l'ISS (Injury Severity Score), c'est la somme des carrés des codes AIS les plus élevés pour les trois régions les plus touchées. L'ISS varie de 1 à 75, sachant que toute blessure ayant un AIS égal à 6 donne automatiquement un ISS égal à 75.

b. Application à la structure osseuse crâniofaciale

Des exemples de blessures crâniofaciales et leur niveau de gravité selon l'échelle AIS sont présentées ci-dessous. Il faut noter que cette échelle détermine seulement la sévérité des lésions pour la survie de l'individu, mais ne prend pas en compte les séquelles esthétiques qui, dans le cas de la face, sont préjudiciables.

Exemples de lésions de la face et de code AIS associé

1(mineure) Fracture de la mandibule, du nez, des dents

2(modérée)

- **Fractures linéaires de la voûte crânienne**
- **Fractures du maxillaire, de l'orbite, de l'os zygomatique**
- **Lefort I, II**

3(sévère)

- **Fractures de la base du crâne**
- **Fractures pénétrantes de la voûte crânienne**
- **Lefort III**

4(sérieuse)

- **Fractures de la base du crâne avec exposition des tissus cérébraux**
- **Fractures pénétrantes de la voûte crânienne avec exposition des tissus cérébraux**

c. Critères de blessures de la tête

Pour la prédiction des risques lésionnels des usagers des transports, les critères de blessures sont classiquement basés sur des grandeurs mécaniques qui caractérisent de manière globale la sévérité du choc. Il peut s'agir, par exemple, d'effort d'impact, d'accélération ou de déflection. Les seuils

de tolérance associés à ces critères sont alors déterminés expérimentalement sur des sujets d'anatomie ou des animaux.

Plus récemment, des critères basés sur des grandeurs mécaniques locales ont été développés. Ces grandeurs locales sont par exemple des contraintes ou des déformations, calculées à l'aide de modèles numériques en éléments finis de la tête.

d. Critères basés sur des mesures globales

Le critère utilisé dans la réglementation pour évaluer les risques de blessures lors d'un choc sur le crâne ou la face est le Head Injury Criterion (HIC). Le HIC a été proposé par la NHTSA en 1972 suite à un ensemble de travaux réalisés précédemment par la Wayne State University.

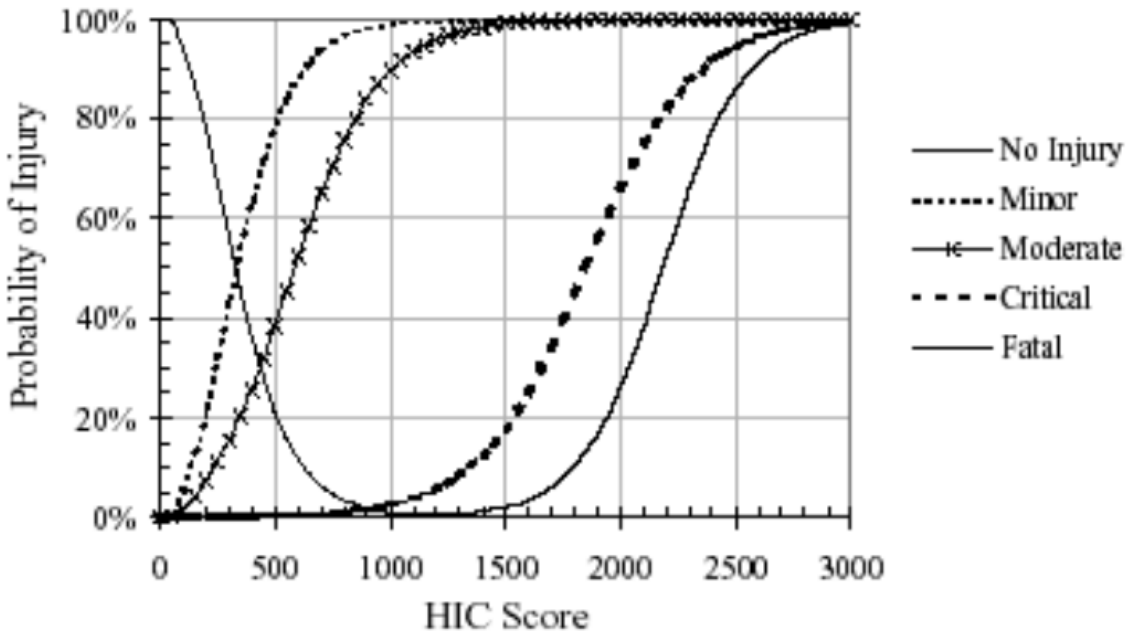

Figure 17 : Probabilité de lésions selon le score du Head Injury Criterium

Ce critère est basé sur l'accélération linéaire de la tête enregistrée au cours du choc :

Un seuil de tolérance égal à 1000 est défini. Au-delà de cette valeur, les risques de lésions cérébrales sont considérés comme trop importants.

Cependant, le HIC est un critère controversé. Plus précisément, il est apparu indispensable de prendre en compte l'effet de l'accélération angulaire de la tête dans la prédiction des lésions cérébrales[39].

Par ailleurs, plusieurs auteurs ont proposés des seuils de tolérances pour la prédiction des fractures de certains os du crâne et de la face [40,41]. Ces seuils sont généralement exprimés en termes d'effort d'impact ou d'énergie d'impactante. Cependant, ils sont très liés au type de sollicitation exercée sur les pièces anatomiques.

En particulier, les surfaces, les masses et les vitesses impactantes conditionnent l'apparition de fractures des os du crâne et de la face ainsi que leur type.

5. Modèles en éléments finis de la tête humaine

Les modèles numériques, en particulier les modèles éléments finis (MEF), sont extrêmement utiles pour comprendre les mécanismes lésionnels mis en jeu lors d'un trauma. Ce sont des outils permettant de déterminer la réponse mécanique des tissus cérébraux lors des impacts, qu'ils soient locaux ou diffus. En segmentant la tête humaine en plusieurs éléments plus petits, la réponse mécanique du cerveau peut être assimilée à un système d'équations algébriques.

Une fois créé, l'étape finale consiste à évaluer le MEF en comparant les résultats obtenus avec des essais expérimentaux effectuées sur des corps donnés à la science. Une fois validé, les résultats de ces modèles peuvent être extrapolés à d'autres cas.

Un des premiers MEF utilisé pour analyser les traumatismes au niveau de la tête a été développé par King et al [39]. Ces premiers modèles grossiers permettaient tout de même d'avoir un premier niveau de réponse, au regard la puissance informatique qui ne permettait pas de réaliser des simulations trop complexe. Depuis lors, la recherche a progressé dans ce domaine et le calcul numérique a connu un essor considérable ces dernières années, permettant une meilleure description des éléments anatomiques composant l'extrémité céphalique et ainsi un raffinement des MEF [42–44].

La validation des modèles numériques par rapport aux données expérimentales est une étape importante permettant de s'assurer que le modèle numérique est en accord avec la réalité.

Les principaux modèles éléments finis de la tête

- Le **modèle développé par Shugar** [45] utilise une géométrie basée sur des mesures relevées sur un crâne sec.

Le maillage, réalisé en éléments volumiques (Figure 18), comporte le crâne, la face, le cerveau, la tente, la faux et l'espace subarachnoïdien. La variabilité de l'épaisseur du crâne est prise en compte, ainsi que sa structure sandwich : les tables interne et externe ont chacune une épaisseur égale à ¼ de l'épaisseur totale.

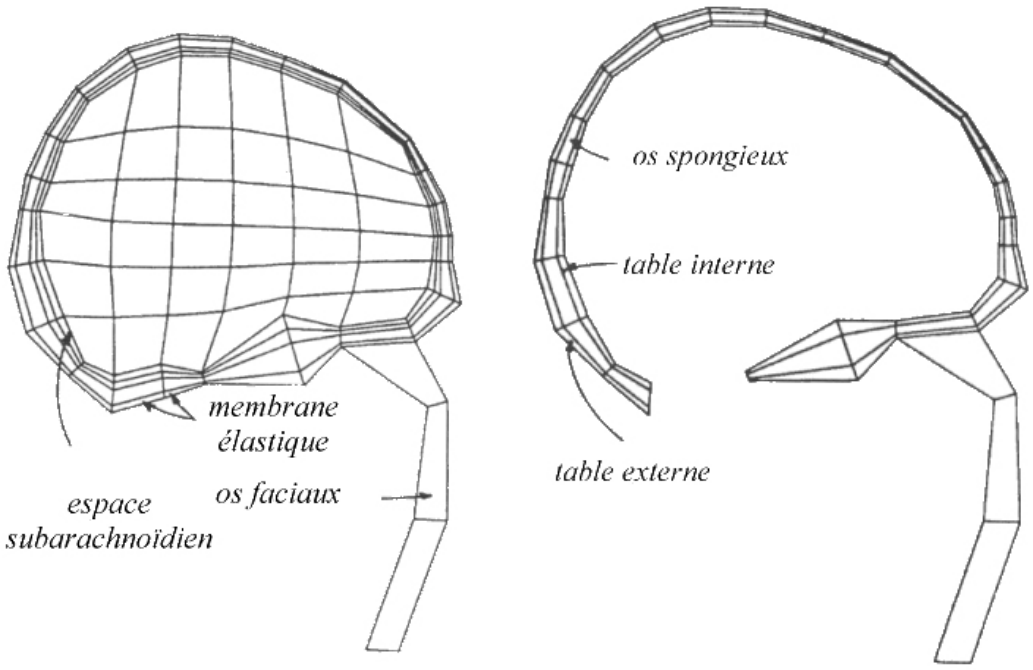

Figure 18 : Modèle en éléments finis de la tête réalisé par Shugar [45]

a. Modèle complet : coupe latérale.

b. Maillage du crâne : coupe latérale.

- Le **modèle développé par Ruan** [46] s'appuie sur une géométrie inspirée de celle de Shugar, relevée sur crâne sec [45].

Ce modèle comprend le crâne (os compact et os spongieux), le cerveau, le fluide cérébro-spinal, la peau, la dure-mère et la faux. Le modèle ainsi finalisé compte un peu plus de 7300 éléments et 6000 nœuds. Le cerveau, le crâne (constitué de 3 couches) et le CSF sont maillés en éléments volumiques

(hexaèdres), tandis que le reste est maillé en éléments surfaciques (coques). Le modèle est validé à l'aide des essais réalisés par Nahum et al. (1977) [46].

Ce même modèle est ensuite modifié par Zhou [47] (Figure 19) qui améliore la modélisation du contenu intracrânien : le maillage du cerveau est densifié (nombre d'éléments multiplié par 5) et des tissus cérébraux sont ajoutés (matière grise, matière blanche, cervelet, tronc cérébral, ventricules, pie-mère, veines). La géométrie de ces nouveaux éléments s'appuie sur un atlas et des coupes anatomiques, sur lesquelles sont digitalisés les contours.

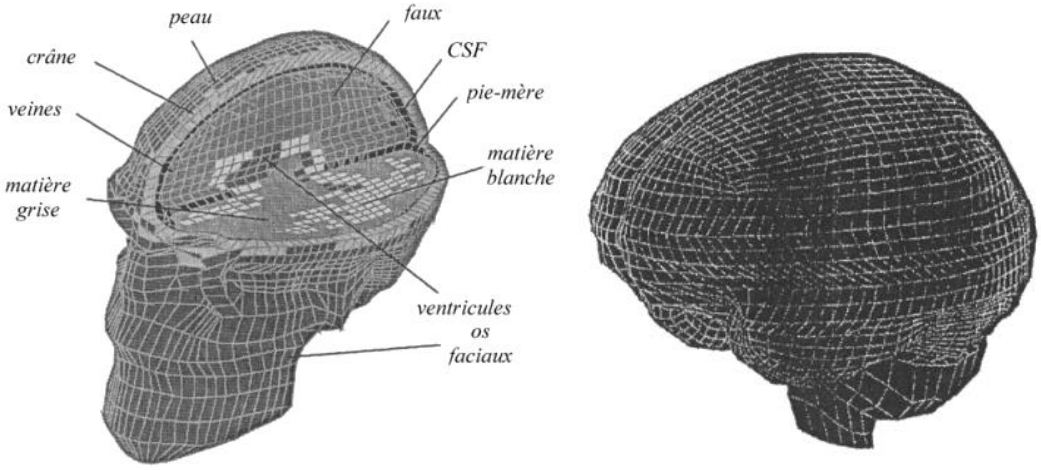

Figure 19 : Modèle en éléments finis de la tête modifié par Zhou [47].

a. Maillage complet de la tête.

b. Maillage du cerveau.

Le maillage ainsi amélioré compte environ 23000 éléments et 18000 noeuds. Le crâne, considéré comme homogène, les constituants du cerveau (matière grise, matière blanche, cervelet, tronc cérébral, ventricules), et le CSF sont maillés en éléments volumiques (hexaèdres). La peau (épaisseur égale à 6mm), la dure-mère, la pie-mère, la faux, la tente et la face sont maillées en éléments surfaciques (coques). Des éléments 1D (ressorts) modélisent les veines.

La masse totale de la tête est égale à 4,37 kg, celle du cerveau est égale à 1,41 kg. La validation du modèle est basée sur les essais réalisés par Nahum [48] et ceux réalisés par Abel et al en 1978 (tests sur animaux).

Ce même modèle a enfin été modifié par Newman [49] : il compte 37000 éléments et 29000 noeuds. La masse totale du modèle est égale à 4.22 kg, dont 1.45 kg pour le cerveau.

- Le **modèle élaboré par Claessens** [50] se base sur la géométrie (images scanner et IRM) de la base de données « visible human dataset ». Les surfaces délimitant les différents tissus permettent de construire des éléments volumiques par une méthode de projection. Le maillage ainsi obtenu compte un peu plus de 12000 éléments volumiques hexaédriques et tétraédriques, représentant le crâne, la face, la tente, la faux, le cerveau, le cervelet, le tronc cérébral (Figure 20). La validation de ce modèle est réalisée sur la base des essais effectués par Nahum et al. (1977) [50].

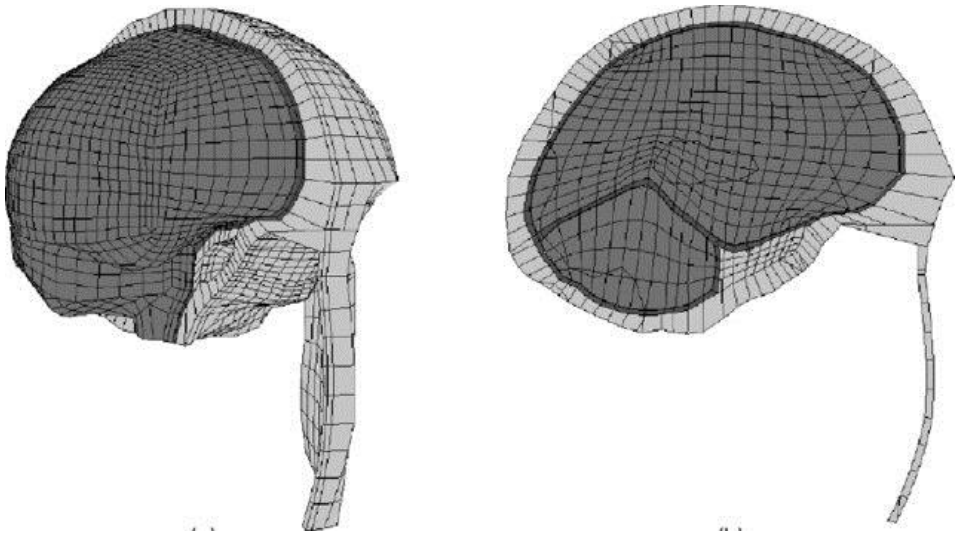

Figure 20 : Modèle en éléments finis de la tête développé par Claessens [50]

- Le **modèle développé à l'Université Louis Pasteur par l'équipe de Willinger** [6,51] s'appuie sur la géométrie d'un crâne sec, dont les contours internes et externes ont été digitalisés à l'aide d'un bras de mesure 3D (Figure 21).

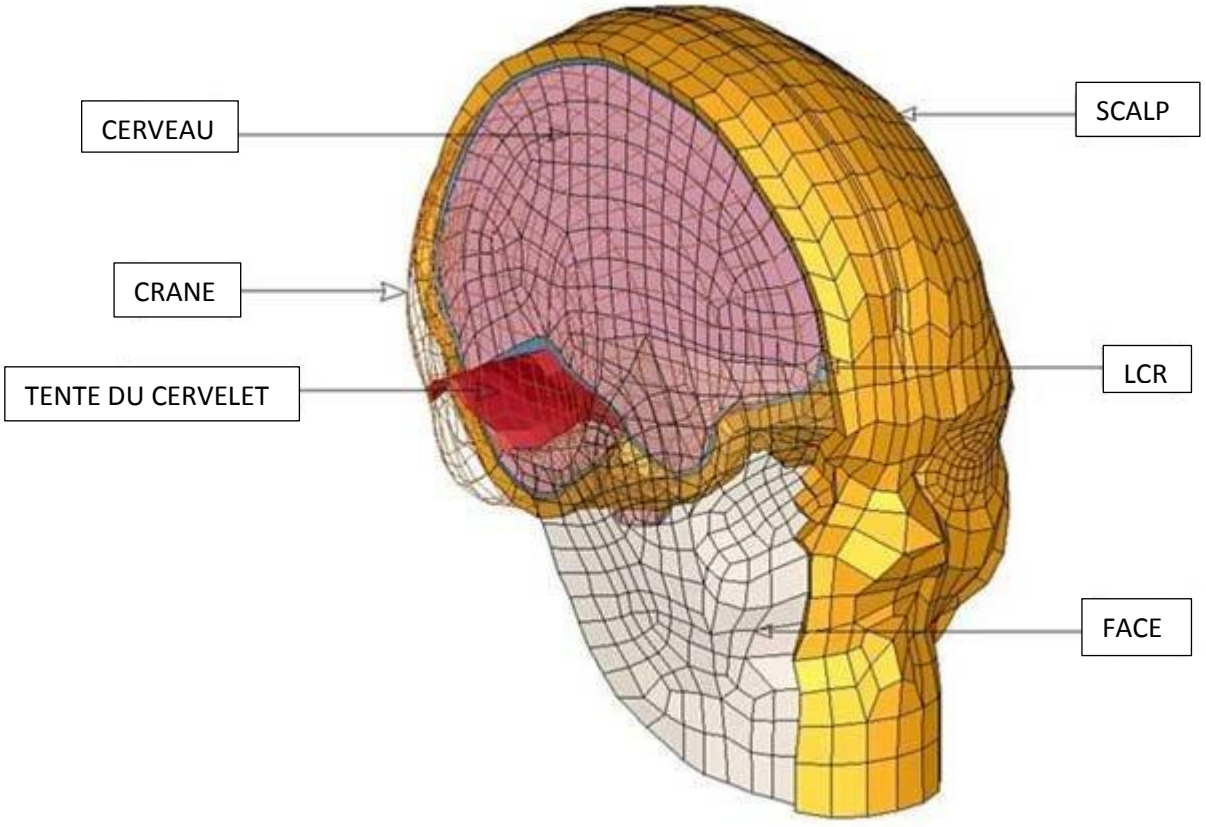

Figure 21 : Modèle en éléments finis de la tête de l'Université Louis Pasteur [51,52]

Il est composé du crâne, des os faciaux, du cerveau, de la peau, de l'espace sub-arachnoïdien, et des membranes de la tente et de la faux. Ceci représente un total de 12000 noeuds et 13000 éléments: 2800 éléments surfaciques (crâne et membranes de la tente et de la faux) et 10400 éléments volumiques (hexaèdres). La masse totale du modèle est égale à 4,77 kg.

Le modèle est validé à l'aide des essais réalisés par Nahum et al. (1977), Yoganandan et al. (1994), et Trosseille et al. (1992) [6,51].

- Le **modèle développé par Camacho** [53] représente la tête et le cou. La géométrie de la tête s'appuie sur des images scanner issues de la base de données « visible human project ». Les contours externes du crâne sont repérés et la surface basée sur ces contours est maillée en éléments surfaciques (coques) (Figure 22). Le modèle compte environ 1300 noeuds, 640 éléments surfacique rigides (4 noeuds), 448 éléments déformables (4 noeuds), 25 ressorts et 25 amortisseurs (cou). Le contenu intracrânien n'est pas modélisé : une masse ajoutée de 1,59 kg, avec un système ressort-amortisseur, est assignée au centre de gravité. La face est modélisée en éléments rigides, avec une masse ajoutée de 4,38 kg. Le modèle est validé à l'aide des essais réalisés par Nightingale et al. [54].

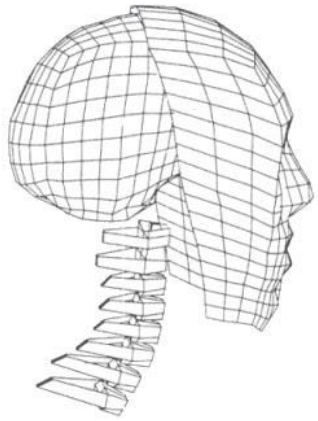

Figure 22 : Modèle en éléments finis de la tête réalisé par Camacho [53]

- **Le modèle KTH**

Le modèle de tête numérique [55–57] connu sous le nom de Kungliga Tekniska Hogskolan (KTH), développé par Kleiven s'appuie sur des images scanner, IRM, et des photos de coupes sériées obtenues par « human visible database » (Figure 23). La construction des surfaces délimitant les différents matériaux permet de réaliser le maillage, avec une mesure de l'épaisseur réelle du crâne.

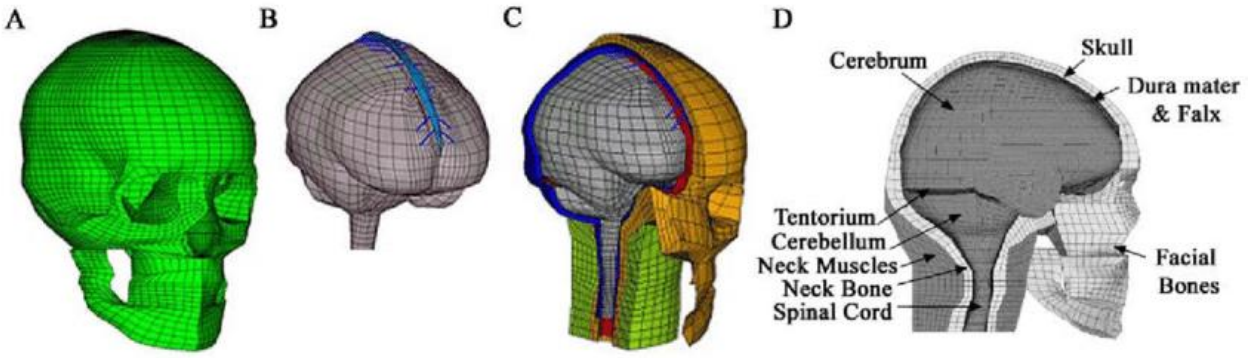

Figure 23 : Modèle en éléments finis de la tête développé par Kleiven

L'os compact, les os de la face, ainsi que la peau sont maillés en éléments surfacique (coques à 4 nœuds). L'os spongieux, le cerveau, le cervelet et la moelle épinière sont maillés en éléments volumique (hexaèdre). Les autres tissus sont constitués d'éléments membranes à 4 noeuds. La validation est réalisée sur la base des essais de Nahum et al. (1977).

- **Modèle de tête WSU**

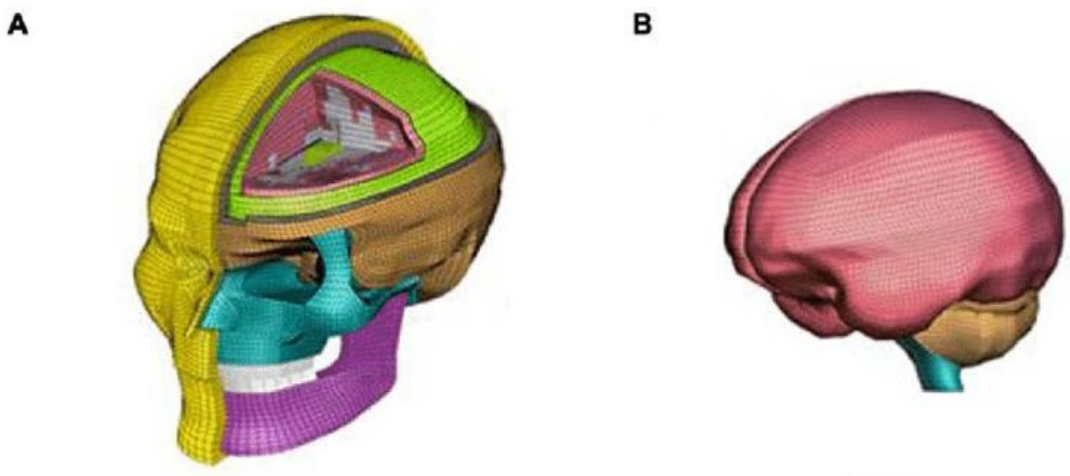

Figure 24 : Modèle en élément fini de tête de WSU

Un autre modèle populaire est le Wayne State University (Brain Injury Medecine) de l'Université Wayne State (WSUBIM)[39,58]. Le modèle comprend environ 315000 éléments et comprend le crâne, le cuir chevelu, la dure-mère, la faux du cerveau, la tente du cervelet, la pie mère, le sinus, le LCR, le cerveau, le cervelet, le tronc cérébral, les ventricules et les veines ponts. Le LCR a été

modélisé avec des éléments volumiques incompressibles, élastiques. En outre, les données IRM et TDM ont été utilisées pour recréer un modèle réaliste du visage avec 14 os et 36 400 éléments. Le modèle de visage détaillé a permis la simulation des impacts sur les os du nez où un modèle de dommage a été inclus pour le matériel osseux.

- **Modèle de tête GHBMC**

Le Global Human Body Model Consortium (GHBMC) a été recréé en utilisant une approche multibloc [59,60]. Le modèle comporte 271000 éléments et est intégré à d'autres modèles de composants du corps pour former un corps entier destiné aux simulations d'accident de voiture. Le modèle comprend le cerveau, le cervelet, le tronc cérébral, les ventricules, les veines pontant, le LCR, le crâne, les os du visage, les méninges, la faux du cerveau et la tente du cervelet. Une interface numérique de collage a été utilisée entre la dure-mère et les couches arachnoïdiennes pour assurer la stabilité du modèle. Une loi viscoélastique linéaire a été choisie pour les tissus de la face, du cerveau et du LCR alors que les autres tissus ont été modélisés comme élastiques linéaires.
Le modèle a été validé à partir d'un nombre important d'études expérimentales, montrant de bonnes capacités prédictives.

- **Modèle principal Ying et Ostoja-Starzewski**

Ying et Ostoja-Starzewski utilisent les données IRM pour convertir des voxels d'image directement en éléments hexaédriques avec une taille d'élément de 1,33 mm x 1,33 mm x 1,30 mm - identique à la résolution de l'image - conduisant à un maillage de plus d'un million d' éléments [61]. L'IRM à base de voxel offre un rendu plus précis et complet de la tête. Le modèle représente le cuir chevelu, le crâne, le LCR, la matière grise et la substance blanche [62]. Les caractéristiques anatomiques fines telles que les membranes, les veines ponts et le réseau vasculaire ont été négligés dans la première version du modèle.

Apres validation du modèle, les simulations montraient lors d'**une chute de la tête de 2 cm, un déplacement de 2-3 mm dans le cerveau.**

La résolution fine de ce modèle permet d'observer que des impacts contondants donnent lieu non seulement à une onde de pression rapide, mais aussi à un cisaillement lent et potentiellement beaucoup plus dommageable [62].

- **Le modèle de tête de Ghajari, Hellyer et Sharp**

Un modèle précis de la tête humaine a été généré à partir de données IRM d'un homme âgé de 34 ans [63]. Le modèle comprend près d'un million d'éléments volumiques (hexaèdres) et un quart de million d'éléments surfaciques (coques à 4 nœuds) , comprenant le cuir chevelu, le crâne, le cerveau, les méninges, l'espace sous-arachnoïdien et les ventricules. Sillon, girations et ventricules ont également été inclus dans le modèle.

L'inclusion d'une géométrie de sillons précise a permis une étude détaillée de la survenue de lésions dans ces régions au cours de trois cas d'impact.

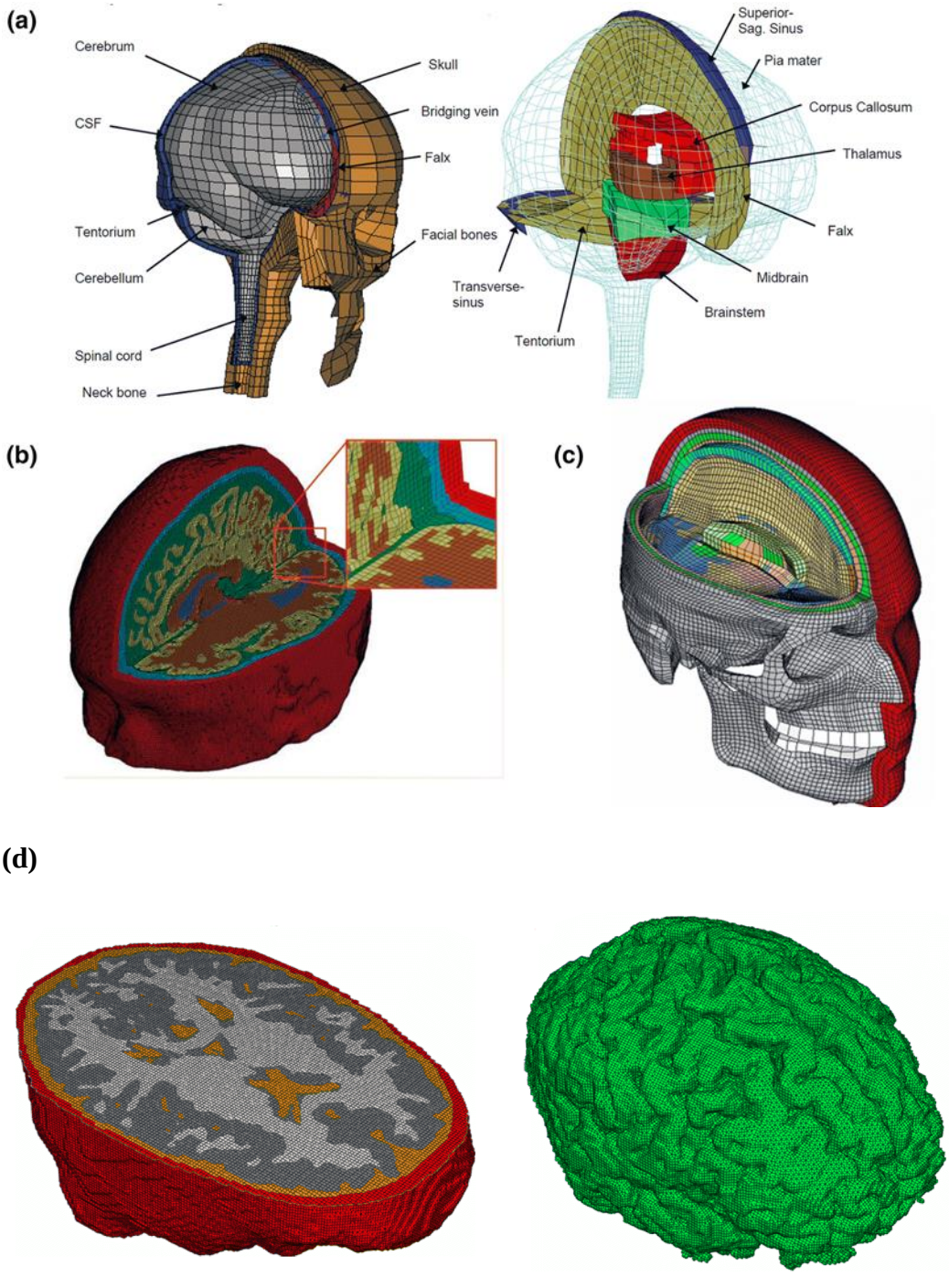

Figure 25 : Différents modèles par éléments finis décrits : (a) modèle de tête KTH, (b) modèle de tête de Ghajari, Hellyer et Sharp, (c) modèle de tête GHBMC, (d) modèle de tête de Ying et Ostoja Starzewski.

- **Le modèle de Tse.**

Très récemment un modèle de tête complet (tête, face et cou), validé à partir de 3 essais expérimentaux a été publié (Tse et al, 2014). Ce modèle comprend la face, la tête, le cerveau ainsi que les vertèbres cervicales fixées à leurs bases. La moelle épinière n'est pas représentée.

La validation du modèle était réalisée en comparaison avec la littérature :

- Nahum et al : masse cylindrique (152.4 mm de diamètre et 40 mm d'épaisseur) de 5.59 kg et une vitesse de 9.94 m/s sur le front.
- Trosseille et al : 6 tests sur 3 cadavres, masse de 23kg en antéropostérieur à la vitesse de 7 m/s.
- Hardy et al : tests frontaux sur bloc de 25mm de large sur 250 mm de long, impact à 3m/s, mesure de déplacement du cerveau par des cibles de densité neutre et déformation du cadavre par radio scopie. (35 tests sur 8 têtes et cou).

Les pressions étaient mesurées sur 5 localisations : tissu cérébral frontal, pariétal postérieur et supérieur, occipital et fosse postérieure.),

A partir de ce modèle validé, 9 tests craniofaciaux ont été réalisés, avec un cylindre rigide (28.66mm de diamètre et 100 mm d'épaisseur) de 3.2 kg à la vitesse de 2.5 m/s.

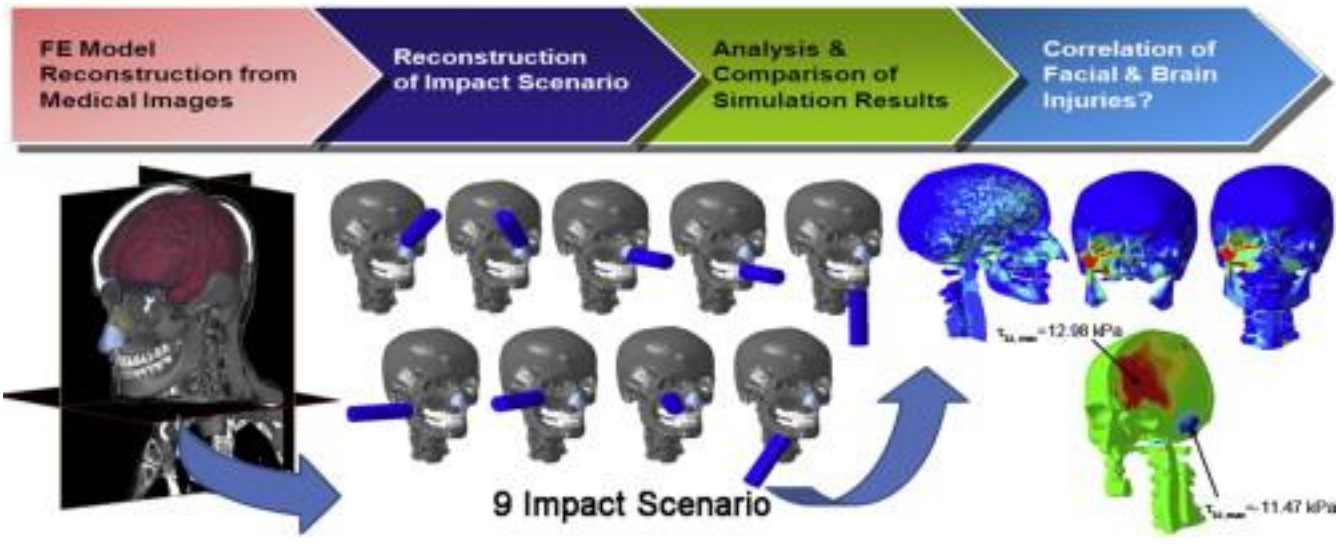

Figure 26 : Modèle de tête de Tse et al.

Conclusion de cette étude bibliographique

La face et le crâne ont tous deux fait l'objet de systématisation biomécanique par les anatomistes : ils sont constitués d'arcs de renforcement et ont donc un rôle de structure protectrice. Ainsi la face abrite les organes des sens et le crâne abrite cet organe vital qu'est le cerveau.

Les fractures crâniofaciales observées dans les services hospitaliers sont principalement causées par des accidents de la voie publique, des chutes, des rixes et des activités sportives. La description des lésions crâniofaciales abonde mais plus rares sont les corrélations aux lésions potentielles cérébrales. De même, des lésions cérébro-cervicales sont fréquemment décrites suite à des impacts faciaux majeurs mais rarement face aux impacts mandibulaires de faible intensité.

Les travaux de modélisation pour la prédiction des risques de blessures se concentrent essentiellement sur la prédiction des blessures du contenu intracrânien.

Cependant, en cas de choc sur la face, l'appréciation correcte de la réponse mécanique de la boîte crânienne et de son contenu repose sur celle de la face. C'est pourquoi il est nécessaire de mieux simuler le comportement de la face et du cou, et cela en réalisant un modèle géométriquement biofidèle, ayant des propriétés mécaniques représentatives, et validé expérimentalement, afin de comprendre les mécanismes lésionnels au niveau cérébral et de la jonction cérébrospinale.

Problématique : utilisation d'une approche numérique pour appréhender les traumatismes craniofaciaux en Médecine légale

Partie II

Modélisation en élèments finis

Il ne s'agit ici que de donner une définition simplifiée et résumée de la méthode des éléments finis. Loin d'être exhaustif, cette définition se base sur le travail de 2014 du Pr Vincent Manet intitulé « méthode des éléments finis ; vulgarisation des aspects mathématiques et illustration de la méthode », destiné à l'enseignement des élèves ingénieurs, et sur les mémoires d'habilitation à diriger la recherche de 2013 du Pr Yannick Tillier « De la caractérisation mécanique du comportement des matériaux à la modélisation biomécanique par éléments finis » et de 2015 du Pr Thierry Serre « Modélisation géométrique du corps humain et simulation numérique de l'accident routier de l'usager vulnérable » , qui peuvent être consultés librement :

Manet V : https://cel.archives-ouvertes.fr/cel-00763690v7/document

Serre T : https://hal.archives-ouvertes.fr/tel-01267155/document

Tillier Y : https://tel.archives-ouvertes.fr/tel-00847642/document

Manet résume ainsi la démarche complète de l'ingénieur numéricien :

1. Modélisation et mise en équations- Construction du problème continu (système d'équations aux dérivés partielles)
2. Analyse mathématique du problème posé- Existence, unicités, propriétés des solutions.
3. Conception d'une méthode numérique- Construction d'un problème discrétisé.
4. Analyse numérique – Questions de stabilité, convergence, précision.
5. Algorithmique- choix de méthodes de résolution en dimension finie.
6. Mise en œuvre sur ordinateur- Programmation.
7. Pré et Post traitement (maillages/ visualisation)- Interpolation, extrapolation, outils de la CAO.

Dans le contexte de la simulation numérique appliquée à l'étude de la biomécanique des chocs, la méthode des éléments finis permet une description fine du corps humain (Serre T, 2015). Le point de départ est d'obtenir une représentation géométrique tridimensionnelle du corps et à la découper en un nombre fini d'éléments (de formes triangulaires, quadrangulaires) qui peuvent être surfaciques ou volumiques qui sont par la suite associés à des lois de comportements adaptées et représentatives de chaque organe anatomique.

Ces modèles permettent une étude ciblée du comportement du corps, plus particulièrement la simulation de la déformation de la structure humaine au cours du choc. La notion de traumatologie virtuelle peut alors être abordée et l'identification des lésions ou des mécanismes lésionnels approchées.

L'élaboration d'un modèle numérique nécessite la connaissance de deux grands types de données : une représentation géométrique du corps humain et des lois mathématiques caractérisant le comportement des matériaux biologiques. Le modèle doit ensuite être validé avant d'être utilisé.

1. Concernant la géométrie, il s'agit d'obtenir une représentation dans l'espace la plus proche possible de l'anatomie humaine c'est-à-dire la plus bio fidèle. L'acquisition de cette géométrie s'appuie alors principalement sur les techniques d'imagerie médicale (scanner dans le cadre de notre étude).
2. Les caractéristiques mécaniques des éléments anatomiques constitutifs du modèle doivent être déterminées afin de simuler leur tolérance au choc. Il s'agit ici d'établir les lois mathématiques régissant le comportement des organes soumis à une sollicitation. L'objectif est alors de caractériser par exemple la rigidité d'un os, la souplesse ou l'élasticité d'un tissu, etc. dans le cas de ce travail de thèse, les caractéristiques utilisées ont été celles déjà disponibles dans la littérature. L'acquisition de ce type de données s'appuie généralement sur des expérimentations réalisées sur des corps

données à la science ou sur volontaires. Ces lois sont alors intégrées dans la géométrie pour former le modèle numérique complet.

3. La validation des modèles est faite par comparaison avec des essais expérimentaux. Dans le cas de notre étude, la validation s'est effectuée par comparaison avec la littérature et les essais expérimentaux sur corps donnés à la science.

II

1. Matériels et Méthodes utilsés dans ce travail

Le modèle utilisé, validé lors d'une précédente étude conduite au sein de notre laboratoire [2], est issu d'une reconstruction 3D d'un crâne humain à partir de coupes scanographiques.

Cette approche numérique a nécessité préalablement d'améliorer et de développer le modèle au niveau de la face, de le valider au travers des données issues de la littérature et de nos essais expérimentaux et enfin, d'évaluer l'influence des conditions de chocs et de la variabilité biologique.

- le premier modèle numérique par éléments finis

Il s'agissait de compléter la numération de la peau, des os de la face et de l'encéphale. La géométrie du crâne a été développée à partir de coupes scanographiques de 1 mm d'un homme de 30 ans à l'aide de MICMICS 12,3 ® (logiciel de Matérialise, Louvain, Belgique). Les principales caractéristiques anatomiques (cuir chevelu, espace sous-arachnoïdien et le cerveau) ont été ajoutées au cours de la phase de maillage. Le modèle a été maillé à l'aide du logiciel Hypermesh ® (Altair Engineering, Inc., Detroit, MI, USA) et mis en donnée avec le code de calcul Radioss ® (Altair Engineering, Inc., Detroit, MI, USA). La dimension moyenne d'un élément était égale à 2 mm. Le crâne a été reconstitué selon trois couches représentant l'os compact et l'os spongieux. L'os compact a été modélisé avec des éléments surfaciques (coque à trois nœuds) et l'os spongieux avec des éléments volumiques (tétraédriques). Le cerveau a été modélisé avec des éléments volumiques (tétraédriques) et l'espace sous-arachnoïdien, compris entre le cerveau et le crâne pour simuler le liquide céphalo-rachidien, a été modélisé par une couche d'éléments volumique (tétraédriques). Le cuir chevelu a été modélisé avec deux couches d'éléments volumiques

(hexaédriques).

La complexité anatomique de la face a nécessité la modélisation de chaque os séparément avec un paramétrage selon la dimension et l'épaisseur corticale. Les sinus et les dents ont été ajoutés et dimensionnés selon les données de la littérature [51,64–70]. La mandibule, son articulation ainsi que le disque articulaire ont été définis. (Figure 27).

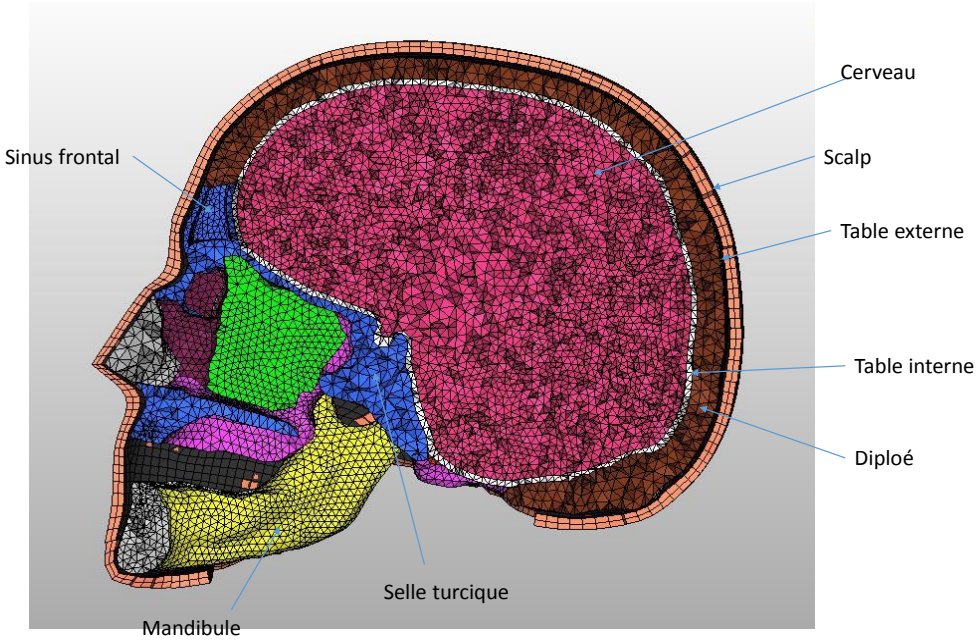

Figure 27 : Modèle de tête en élément fini (coupe sagittale)

- Validation du modèle

Le modèle développé était validé selon:

- les tests expérimentaux réalisés **par Schneider et al (1972)** [40] : Celui-ci recherchait le niveau de tolérance des os de la face et ainsi de la mandibule, à partir de 106 tests sur 17 crânes donnés à la science (embaumés ou non). Concernant la mandibule, les tests étaient effectués en position antéro-postérieure et latérale.

- les tests expérimentaux **de Viano et al (2005)** [58] étudiant l'impact suite à un coup de poing

de boxeurs. Les boxeurs olympiques devaient réaliser 4 coups différents (uppercut, jaw ou antéro-postérieur, Hook ou latéral et frontal), sur un mannequin Hybrid III mesurant ainsi les efforts sur le mannequin et sur les mains des boxeurs.

Nous avons également évalué notre modèle numérique à partir d'essais réalisés au sein du laboratoire de biomécanique appliquée. Trois tests expérimentaux sur des corps donnés à la science ont été effectués afin de mieux appréhender les mécanismes lésionnels mis en jeu et de renforcer la validation de notre modèle numérique. Les sujets, 2 hommes et une femme, âgés de plus 75 ans, étaient assis dans un siège baquet inclinable selon un angle défini en fonction de la taille du sujet. Un impact était pratiqué sur la symphyse mandibulaire, afin de simuler un coup de poing de type uppercut, par une masse de 5kg à la vitesse de 5m.s-1. La masse utilisée était un cylindre métallique (diamètre 76.5mm et longueur 458mm). Le pendule était tenu par des câbles métalliques et relâché à une hauteur prédéfinie permettant d'atteindre la vitesse souhaitée. Un capteur d'effort était positionné sur le pendule afin d'identifier l'effort appliqué au niveau de la mandibule lors de l'impact. Un accéléromètre était fixé sur le front afin d'identifier la décélération subit lors de l'impact. Une caméra rapide à 1000 images par seconde était utilisée permettant une analyse de la cinématique après l'essai.

Après chaque essai expérimental, une dissection était réalisée de la partie inférieure de la face de chaque individu afin de localiser les fractures occasionnées. Chaque individu était préalablement pesé et, lors de la dissection, la longueur de la branche montante et du corps de la mandibule a été mesurée.

- Étude de la variabilité

Une étude paramétrique a été réalisée faisant varier les conditions de choc et les paramètres biomécaniques de la mandibule afin de vérifier leur influence sur la transmission des efforts au niveau de la région mésencéphalique. Pour toutes ces simulations, l'épaisseur corticale de la mandibule était de 1.5 mm, le module d'Young de 13700 MPa, une vitesse d'impact de 6.7 $m.s^{-1}$, une masse d'impact de 1.5 kg et une densité de 2500 $kg.m^{-3}$.

Les conditions de chocs :

- zone d'impact : uppercut, face menton, latéral

- vitesse d'impact : 4, 5, 6.7, et 8 $m.s^{-1}$

- masse d'impact (ensemble gant de boxe / main) : 1.2, 1.7 et 2.2 kg.

La masse de référence utilisée était ainsi de 1.7 kg, en accord avec l'équipe de Viano et al [58] qui avait une masse moyenne à 1.67 kg.

Concernant la variabilité biologique, il a été évalué l'influence :

- de l'épaisseur corticale: 1,5 mm, 3 mm, et 5 mm,

- de la « morphologie » de la mandibule : édenté complet, haut, bas, et dentition complète,

- du module d'Young : 10000 et 13700 MPa

- de la densité osseuse : 1500 et 2500 $kg.m^{-3}$.

Pour chaque simulation, il a été analysé l'effort d'impact au niveau de la mandibule, la transmission de l'effort à plusieurs niveaux de la face (ex: condyles) jusqu'à la zone du mésencéphale. De plus, il a été mesuré la décélération au centre de gravité de la tête. Enfin, il a été visualisé les fractures.

- Caractéristiques du second modèle.

Ce second modèle éléments finis développé est une combinaison de notre premier modèle de tête développé par le Laboratoire de Biomécanique Appliquée (LBA) et d'un modèle de cou développé dans le cadre d'une collaboration avec l'École Polytechnique Montréal (EPM). La jonction tronc cérébral/moelle épinière a été modélisée dans la continuité des éléments du tronc cérébral représenté par des éléments tétraédriques. Dans le modèle de cou issu de notre laboratoire (LBA, IFSTTAR en collaboration avec ILABSPINE), seuls les éléments des corps vertébraux et la moelle épinière ont été conservés.

La complexité anatomique du cerveau, des méninges et du cou a nécessité la modélisation de chaque élément (pie mère, dure mère, faux du cerveau, tente de cervelet, hémisphères, cervelet, tronc cérébral, moelle épinière cervicale, vertèbres cervicales et ligaments) selon leurs différentes propriétés mécaniques. Un maillage continu a été utilisé.

Le crâne a été reconstitué selon trois couches représentant l'os compact (table externe et interne) et l'os spongieux (diploé), modélisées avec des éléments tétraédriques. Le cerveau et l'espace sous-arachnoïdien, compris entre le cerveau et le crâne pour simuler le liquide céphalo-rachidien, ont été modélisés par des éléments tétraédriques. La dure mère, la faux du cerveau et la tente du cervelet ont été modélisées avec des éléments coques à trois nœuds. Le cervelet ainsi que le tronc cérébral ont été individualisés. Le modèle a été fixé au niveau de l'extrémité distale du cou.

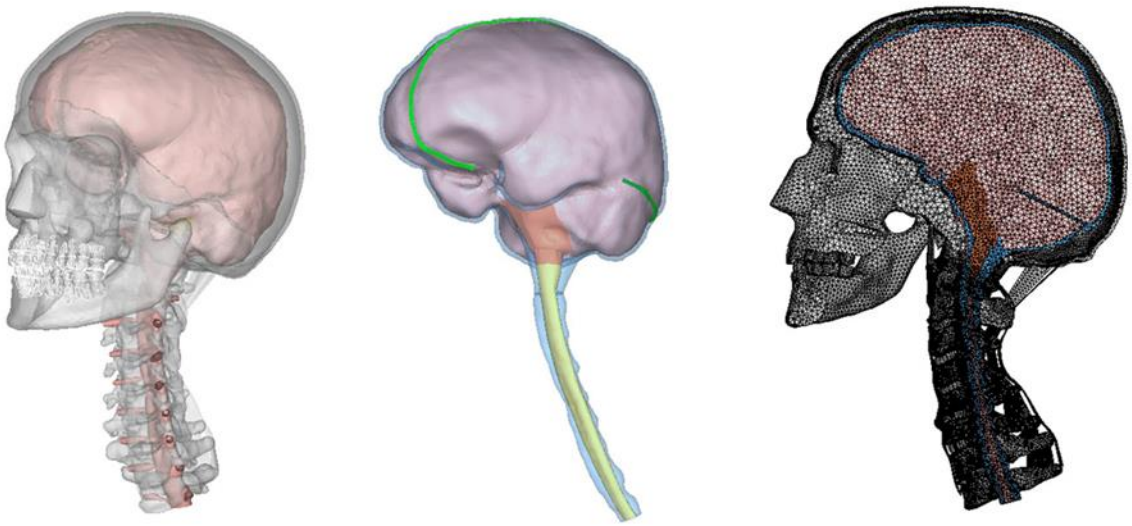

Figure 28 : Modèle de tête et cou par éléments finis : modèle complet, cerveau et moelle spinale et coupe sagittale du modele.

- Validation du modèle

Le modèle a été évalué à plusieurs niveaux : 4 sources de validation différentes (étude de Nahum, Trosseille, Viano (Figure 29) et essais expérimentaux que nous avons réalisés au laboratoire) basées selon les expérimentations les plus pertinentes de la littérature et selon 3 configurations différentes (3 impacts mandibulaires : uppercut, crochet et antéro-postérieur) afin d'étudier l'influence du site d'impact au niveau mandibulaire.

1. **Nahum et al** [48] **(essai 37):** impact avec une barre cylindrique rigide (masse 5,59 kg et vitesse d'impact de 9,94 m/s) sur la région frontale d'un PMHS assis, le torse maintenu. L'impact a été pratiqué sur un sujet sur un plan sagittal, en antéro-postérieur, avec la tête inclinée en avant de 45° selon le plan de Frankfort. (Fig.2).
2. **Trosseille et al** [4] : impact avec une barre de fer rigide (masse 23,4kg et vitesse d'impact 7m/s) sur la face d'un PMHS assis en direction antéro-postérieure (MS 428-2). (Figure 29)

Afin d'évaluer la réponse de notre modèle, nous avons comparé la force d'impact au niveau de la zone frontale, et l'accélération de la tête au centre de gravité, avec les essais de Nahum. Les pressions en région frontale et occipitale ont été mesurées et évaluées selon les essais de Nahum et Trosseille.

3. **Viano et al** [58] : impacts effectués sur 3 zones mandibulaires selon différentes vitesses d'impact avec a hand mass de 1,67 kg : jaw (9,2 m/s), hook (11m/s) et uppercut (6,7 m/s). (Fig. 2)

4. **Essais expérimentaux réalisés en 2015 dans le laboratoire** : impact avec un cylindre rigide (masse de 5kg et vitesse d'impact de 5m/s) sur un PMHS afin d'évaluer la force d'impact au niveau de la symphyse mandibulaire et évaluer le type de fracture observée [38].

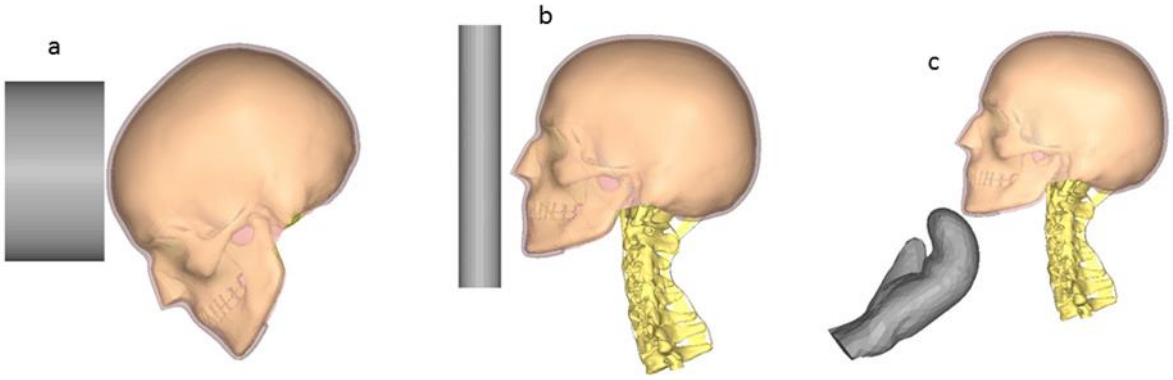

Figure 29 : Trois tests de validation numérique : Nahum et al (a), Trosseille et al (b), Viano et al (c).

II

2. Etude de la transmission des forces au crâne : développement du modèle crâne et face

Afin de répondre à notre première interrogation à savoir la transmission des forces au crâne suite à un impact mandibulaire, nous avons créé un premier modèle de tête qui a été publié par notre équipe en 2015.

Afin de comprendre les mécanismes lésionnels mis en jeu, nous avons conduit une étude expérimentale et numérique selon un modèle éléments finis (MEF) tête-face. Dans un premier temps, nous avons démontré qu'il existe une transmission des forces appliquées de la mandibule à l'encéphale et en particulier au mésencéphale. Dans un second temps, nous avons évalué les paramètres influençant potentiellement cette transmission : La vitesse de l'impact, la position et la masse d'impact, mais également la morphologie de la mandibule ainsi que les propriétés biomécaniques de l'individu (module de rigidité ou module d'Young, épaisseur corticale, densité osseuse…).

Cet article a été publié au Forensic Sciences International (cf annexe 1)

Forces transmission to the skull in case of mandibular impact

Lucile Tuchtan[a,d], Marie-Dominique Piercecchi-Marti[a,d], Christophe Bartoli[a,d], Dominic Boisclair[c], Pascal Adalian[c], Georges Léonetti[a], Michel Behr[c], Lionel Thollon[b]

a. APHM, CHU Timone, Service de Médecine Légale et Droit de la Santé, 13385 Cedex 5, Marseille, France

b. Aix-Marseille Univ, LBA, 13916 Cedex 20, Marseille, France

c. IFSTTAR, LBA, 13916 Cedex 20, Marseille, France

d. Aix-Marseille Univ, UMR 6578, 13385 Cedex 5, Marseille, France

The e-mail address, telephone and fax numbers of the corresponding author:

E-mail address: lucile.tuchtan@ap-hm.fr

Tel: (33)+4.91.48.22.45

Fax: (33)+4.91.92.33.31

Abstract

Background: Loss of consciousness and cardiac arrests resulting from direct mandible impact has been reported through forensic investigations. How forces are transferred to the skull remains however unclear. We conducted a study on the level of energy required to create a mandible fracture and on the energy dispersion phenomenon to the skull and to the brain.

Material and Methods: This study combines an experimental and numerical approach. Mandible strike was studied with experimental trials made on post-mortem human subjects. A finite element model of the head and face of a male was also developed based on tomodensitometry scans. The model was validated with literature data and experimental trials. A parametric study was then performed in order to study the effect of diverse variables such as the dentition integrity, cortical bone thickness, etc.

Results: The forces measured on our reference model were 3000 N on the chin, 1800 N at the condyles and 970 N in the occiput. Of all the results, we observe a decrease of about one-third of efforts chin to the base of the skull and a lower half still forces at the occiput, except in edentulous and when lateral and frontal impact where the force is transmitted directly to the skull base area.

Conclusion: This study allowed us to create a 3D model of the mandible and face bones to better understanding force transfer mechanisms into and from the mandible. Model's parameters may be modified to suit individual characteristics for forensic investigations and legal matters.

Keywords: Finite element modeling, Mandible, Uppercut, Forensic investigations

Introduction

Establishing the relation between the death of an individual and violent events is a common practice in forensic science. Many cases of death by violence are relatively obvious and are the result of major blood loss or organs compression caused by hemorrhage. However, the process of identifying the causes of the death is sometime solely based on the time-event continuum between the violent act and the death without confirmation of the injury mechanisms. This may be the case when anatomical signs of the injury mechanisms are absent such as death by reflex reactions related to nervous conduction (death by carotid compression, thoracic impact, ocular globes compression, etc.) [1] or injuries to the central nervous system (axonal damage).The hypothesis that forces are internally transferred to the brain and more specifically the brainstem is sometime promoted for the latter case. At the Marseille forensic service, two deaths related to uppercut were recorded and brain histological examination only showed midbrain's lesions. Genarelli and al (1982) studied the diffuse axonal lesions emergence conditions by apes testing [2]. They conclude the functional consequences, resulting in loss of consciousness, is directly due to axons'destruction at impact. Others (Besenski et al Povlishock et al, et al Sahuquillo, Buki et al ….) showed that the axon functionnal role can be altered even if it is not cut [3-6]. These stretched lesions are found in lower resistance axons areas: the brains area transitions between gray matter and white matter, periventricular white matter, corpus callosum and with a higher severity degree the dorsal mesencephalon (Sahuquillo et al 1989) [7].Belingardi et al. demonstrated that the brainstem pivots upon facial impact and suffers alterations by following shearing mechanisms [8]. Without fracture, the skull movement at the impact, may still have caused a brain direct contusion. Different degrees of contusion are possible from simple transient local vasomotor paralysis, to various extensive parenchymal tears, inevitably concerning vascular elements. We refer to "blow" as opposed to lesions of "blacklash" that the inertia cause away from the impact point. These inertial effects can be observed when the head is violently set in motion without impact, in less direct: the driver undergoing a "whiplash"

when the vehicle is buffered from behind, rugby player or football player pushed in the back, the boxer receiving an uppercut, or even with impacts suffered by a helmeted head [9]. Our study is primarily concerned with the understanding of energy transmission upon impact of the mandibular area to the rear of the face, through the condyles. We try to show how during a single impact, like an uppercut, residual energy (some is absorbed by the face with broken bones) arrives to brainstem.

Brain injuries sustained by boxers and American football players following a mandible impact have been reported recently [10, 11].

Finite element models of the head have been proposed recently [12-15] in order to study this issue through parametrical analysis.

We decided to investigate effects of mandible impact resulting from fighting.

In order to understand this injury mechanism, we conducted an experimental and numerical study with a validated head-face finite element model (FEM). We first demonstrate that forces are transmitted from the mandible to the skull and more especially to the condyles. Part of the residual energy spreads to the brain and brain stem and we will illustrate this energy transfer phenomenon. We assess how some parameters may affect this transmission, namely the impact velocity, position and mass but also the mandible physiology and biomechanical properties (Young's modulus, cortical bone thickness, bone density, etc.).

Material and Methods

Experimental investigation

Three experimental trials were conducted on post-mortem human subjects (PMHS) in order to measure forces involved and to validate the FEM. Two man and one woman aged above 75 years old were sat in a tilting seat with a preset angle defined by the subjects' height. Two out of three subjects were toothless. Subjects' anthropometric data are reported in table 1. An uppercut strike was simulated by impacting the subjects on the mandibular symphysis with a 5 kg mass and impact velocity of 5m/s. The impactor mass, a metallic cylinder of 76.5 mm in diameter and 458 mm long, was mounted on a swing and dropped at a predefined height. A load cell placed on the impactor measured the forces transferred to the mandible and an accelerometer placed on the subjects' forehead measured its acceleration upon impact. Trials were filmed with a high speed camera (1000 fps) to record the impact kinematic.

Lower end of the subjects' face were dissected after the impact to identify any potential fracture.

Numerical investigation

A previously validated 3D FEM of a human head created based on tomodensitometry scans was used in this study [12]. The model was first improved by adding the face and validated with available literature data and our experimental trials. Effects of variations in impact and biological parameters were then assessed.

a) FEM description

Skull geometry was created with tomodensitometry scan with slice thickness of 1 mm and numerically reconstructed with MICMICS 12.3 software (Materialise, Louvain, Belgium). Main anatomical components (scalp, subarachanoid space and brain) were added in the meshing process. Meshing was made with Hypermesh software and the model properties were managed with Radioss (Altair Engineering, Inc., Detroit, MI, USA). Average element size was fixed to

2 mm. Skull was constituted of three layers to recreate trabecular and cortical bone. Cortical bone mesh used shell elements with 3 nodes and trabecular bone used tetrahedral elements. Brain and subarachanoid space acting as the cerebrospinal fluid situated between the brain and skull were modeled with tetrahedral elements. Scalp was modeled with two layers of brick elements.

Facial bones were separately modeled with a parametric approach to define size and cortical bone thickness. Sinus, teeth, mandible and articulation discs were added and sized based on literature data [16-22]. Model is presented in figure 1.

The FEM is composed of 700 000 volumetric elements (tetrahedral and brick) and 80 000 shell elements (3 and 4 nodes). Total mass is 4.2 kg. All mechanical properties (table 3) were taken from literature data [16-20, 23, 24].

b) FEM validation

Model was validated according to:

- Experimental trials conducted by Schneider et al. [25]. Resistance of facial bones and mandible was assessed with 106 trials on 17 PMHS skulls. Two different positions were used to impact the mandibles: antero-posterior and lateral. Two plexiglass cross members were affixed to the drop assembly and contained nylon bushings to minimize any frictional reaction with the steel guidewires. In addition, the heads were severed at the seventh cervical vertebra in the latter experiments to facilitate the placement of the skull necessary for Antero –posterior mandibular impacts. The head, in all cases, was supported by wedges of soft polyurethane padding. (Conditions de test, limits) (Table 4)

- Experimental trials conducted by Viano et al. [10] where the effect of a boxer punch was studied: Eleven Olympic boxers weighing 51 kg (112lb) to 130kg (285lb) were included in the study. They were instructed to strike the instrumented Hybrid III head with their gloved first two times with four different punches, straight punches to the forehead and jaw and a hook and

uppercut. Accelerometers was placed in the boxer's clenched hand. (Two Endevco 7264-2k accelerometers) The Hybrid III was equipped with the standard triaxial accelerometers (Endevco 7264-2k). (Table 4).

- Experimental trials described previously in this study.

Results

Experimental investigation

First PMHS trial (subject n°1) resulted in a 2 cm long laceration to the chin, linear fracture of the mandible symphysis and bilateral mandible necks linear fracture (Figure 2). No fracture at the temporal regions was found. Second trial (subject n°2) resulted in a laceration to the chin, a complete oblique linear fracture of the right side mandible and bilateral mandible necks linear fracture. Last trial (subject n°3) resulted in a laceration to the chin and bilateral mandible neck oblique linear fractures. Average acceleration of the 3 tests was measured as 22 G. All forces measured are presented in table 2.

Numerical investigation

a) Model validation

Validation results are shown in table 4. Our results are comparable to above studies and therefore the FEM may be a suitable method.

b) Variability study

A parametric study with varying impact conditions and mandible's biomechanical properties was performed in order to assess how forces transmission to the skull is affected. Following parameters were used as a comparison baseline: cortical bone thickness (1.5 mm), Young's modulus (13.7 GPa), impact velocity (6.7 m/s), impactor mass (1.5 kg) and density (2500 kg/m^3).

Impact conditions variations were:

- Impacted zone: uppercut, chin, lateral
- Velocity: 4, 5, 6.7 and 8 m/s.
- Impactor mass (simulating hand and glove): 1.2, 1.7 and 2.2 kg.

Reference impactor mass 1.7 kg is in accordance with previous work of Viano et al. [10] who used a mass of 1.67 kg.

Mandible's biomechanical properties variations were:

- Cortical bone thickness: 1.5, 3 and 5 mm.
- Dentition: toothless, complete upper dentition, complete lower dentition or full dentition.
- Young's modulus: 10 or 13.7 GPa.
- Bone density: 1500 or 2500 kg/m³.

Impact forces at the mandible and forces transferred to various face regions up to the skull were analyzed for each simulations. Deceleration at the center of the head was assessed and visual fracture incidence documented.

Fifteen simulations were conducted. All simulations were done with Radioss software (Altair Engineering, Inc., Detroit, MI, USA). Forces were measured at three levels: chin, section through mandibular condyles and skull base, and one measurement at the occiput. Results are presented in table 5. Force curves were filtered at 600 Hz and acceleration curves at 1000 Hz.

b.1) Effect of impact site location

Forces measured at the impact site and at the condyles levels are close for chin and lateral strikes: forces are respectively 2200 N and 2300 N at the impact site and 2200 N at the condyles level. For the uppercut strike, force at the impact site was 3000 N and 1800 N at the condyles.

b.2) Effect of biomechanical variation

Following parameters are associated to the victim:

- Cortical bone thickness: forces measured at the chin were 3000 N for a cortical bone thickness of 1.5 mm and 2350 N for thickness of 3 and 5 mm. Forces measured at the condyles are 1850 N regardless of the cortical bone thickness. Forces are somewhat damped with greater cortical bone thickness.

- Dentition: forces at the condyles and at the skull base remained high (between 2800 N and 3600 N) for incomplete dentition and toothless models. Forces at the condyles dropped to 1800 N for the complete dentition model.

- Young's modulus: forces at the impact site were modestly lowered with the modulus of 10 GPa compared to the reference modulus (13.5 GPa). Forces at the condyles and occiput are similar regardless of the modulus used with the exception of the maximum load of 2200 N with the 10 GPa modulus.

- Bone density: Forces measured at the impact site was of 2250 N for the 1500 kg/m³ density. The forces increased to 3000 N with the reference bone density of 2500 kg/m³.

b.3) Impact conditions

Following parameters may be associated to the striker:

- Impact velocity: Forces found at the impact zone are similar between impact velocity and decrease progressively from the condyles to the occiput. Peak force of 780 N followed by a second peak of 1700 N was fond with an impact velocity of 8 m/s.

- Impactor mass: Increase in the impactor mass increased the forces at the chin from 2300N to 3200 N.

In our study, we aimed to assess force dispersion in the mandible but also at the brain level. To do so, a visual inspection of the Von Mises stress distribution on our FEM was done. Figure 3 shows stress distribution for various time steps following the impact. Significant forces are found at the base of the skull and specifically at the midbrain level which is similar to what Viano et al. [10] found.

All subjects sustained a fracture at the condyles and mandible symphysis.

Discussion

This study confirms that a mandible impact causes cranial bone damage and may generate brain injuries. Forces measured in this study are similar to data reported on anthropometric surrogate and PMHS which validate our model.

FEM was validated with experimental trials. FEM's parameters may be modified to suit biomechanical variability of the victim or striker which is a significant benefit for forensic investigations.

Previous FEM study investigated impacts to the face (Bruyère et al, Yoganandan et al, Allsop et al, Schneider et al, etc.) [25-34]. Frontal or maxillary impacts resulting from road crashes (Yoganandan et al [27,29], Hampson [28]) with contact to the driving wheel (Hodgson et al [31]) were the focus of great attention. Previous studies on uppercut strikes were concerned by the mandible resistance to impact [21, 35], mechanical properties [20] or chewing [22].

Evidently, from our understanding, the forces are transmitted along the anteroposterior direction (frontal-temporal-occiput). Forces at the chin, skull base and occiput are significantly decreased with our model featuring a complete dentition.

a) Impact site effects

Our results show that frontal and lateral strikes transfer forces directly to the skull base and occiput, although they are slightly less for the latter. Our results at the impact site are similar to what Schneider et al. [25] have reported, albeit in this study, the mandible was impacted with a mass of 3.12 kg and velocity ranging between 4.8 and 5.4 m/s. Along the anteroposterior direction, reported forces at the impact site were ranging between 1890 and 4000 N depending of the individual. Forces at the impact site ranged between 820 and 3400 N for an impact mass of 3.8 kg and velocity ranging between 5.4 and 5.9 m/s for lateral mandible impacts. According to Viano et al. [10], impact force ranged between 1010 and 4090 N for a strike at the jaw and

between 994 and 9950 N for hook punch. Our results are within the same force ranges that are admittedly quite large, highlight the great variability of the impact conditions.

b) Biomechanical variations

b.1) Dentition

Forces were somewhat damped by the addition of teeth in our model. Partial dentition and toothless models were similar. Forces measured on our FEM are similar to our experimental trials. Toothless cases had reduced alveolar bone and therefore reduced spacing between the impactor and skull base. Walilko et al. [11] have shown that protective mouthpieces may half forces measured at the mandibular fossa with the added material increasing force damping. In our study, we chose to assess the difference between two extreme cases (complete dentition and toothless) and therefore our results must be adjusted to the condition of remaining teeth (altered or not, number, prosthetic materials, osteoporosis, etc.). Chosen parameters were linked to the presence or not of teeth and not to the subject's age which may, along with individual variability, affect force transfer mechanisms.

b.2) Cortical bone thickness

Similarly, increasing the cortical bone thickness decreases the force damping effects with forces being directly transferred to the skull base. For a cortical thickness of 5 mm, a peak appears which is most likely due to the increase bone stiffness and decrease damping due to early bone fracture.

b.3) Young's modulus and bone density

Reducing the Young's modulus and bone density decrease the force measured at the impact site (chin) and thus favors the occurrence of fracture by energy absorption.

c) Impact conditions

c.1) Impact velocity

Only small variations in forces at the chin and condyles were measured with increased velocity in our study. Forces were however significantly higher at the skull base with increased velocity due in part to the rebound effect. We chose the 6.7 m/s velocity as the reference velocity based on the published study of Viano et al. [10] who estimated the velocity of an uppercut strike to be close to this value.

Forces measured in our study are similar to previously published data; with peak forces varying between 458 and 3330 N. However, reported data were obtained on anthropometric Hybrid III dummy with the head, neck and torso components. We chose to study force transfer mechanism with the skull and did not take into account the potential effects of the cervical spine and rearward head motion. Forces found for strikes on the jaw and hooks are similar to values found on our model. Our measurements are included in the large bracket of published values; highlighting the difficulties of accurately assessing such values with so many individual specific parameters. Individual tested were 1.65 to 2 m tall and weighted between 50 to 130 kg. Values found can therefore be associated to a scenario where an average person hits another with the intent of arming him.

c.2) Impactor mass

Decreasing the impactor mass from 1.7 kg to 1.2 kg had little effect outside of decreasing the forces at the chin from 3000 to 2300 N. Increasing the mass to 2.2 kg resulted in the same energy transfer but with the added backlash effect most likely due to secondary motion of the head. Impactor masses used were representative to the morphology of a regular person.

Injury review

All experimental trials and simulations resulted in a mandible fracture located at the condyles and symphysis. Lowest force measured at the impact site was 2200 N. The fracture threshold value is considered to be between 1000 and 1800 N according to Allsop et al. [30]. Fracture

threshold value is between 2500 and 3100 N for face impact and in between 600 and 800 N for lateral impact according to Unnewehr et al. [35]. Thus are results are considered in agreement with previous studies.

In our study, we validated the skull model to show the energy transmission to the skull but we wanted to show by a Von Mises picture the cerebral pressure areas. Von Mises stress was distributed with an anterior to posterior orientation through the temporal lobe and ending at the brainstem level. Viano et al. [10] found that a hook strike increases stress in the temporal region and at the midbrain level. Belingardi et al. [8] developed a FEM of the head based on the experimental trials made by Nahum et al. [36] and found increased cerebral pressure with significant stress in the coronal section of the brainstem which confirms its pivot role with movement of the brain. Thus, according to our study, in a simple uppercut, residual impact energy (some is absorbed by the face with broken bones) arrives at the brainstem. But for Ommaya and al, the brainstem and mesencephalon would be the last to be affected both functionally and structurally after human head injury because they are anatomically the most protected structures from injuring strains [5, 37]. Consequently, in every case where structural damage is found in the brainstem, structural damage will also be found in the brain hemispheres [5, 37-39]. The most important consequence of Ommaya's theories was the newly introduced and controversial concept that when primary damage is found in the rostral brainstem, it is never isolated but is associated with diffuse brain damage to the hemispheres [5, 37-39]. This concept challenged the long-standing classical view that the main mechanism producing traumatic unconsciousness was an isolated "primary brainstem injury" [5, 40-43].

Head acceleration in our models was measured as 22 G. Viano et al. [10] found similar values with average acceleration of 24 G for uppercut strike.

Thus, we can suppose during the uppercut, part of the impact energy reaches to the brainstem (seen in images of the brain with Von Mises, red in the image) which is obviously supported

by the stress found at the condyles during simulations and fractures observed levels at the temporomandibular joint simulation that in both experiments. This is very interesting from a forensic point of view because the pressures observed in the brainstem could lead to a vagus nucleus stimulation, which may be involved in cardiac arrest by cardio-inhibitory reflex.

Conclusion

A validated heads' FEM allowed us to identify potential injuries induced by a mandibular impact. Although the forces propagation logically follows an anteroposterior direction (fronto-temporo-occipital), the efforts intensity distribution varies with different parameters. The forces' intensity doesn't decrease gradually depending on its propagation but its variation depends on many parameters including, first, the mandibular impact, but also the force applied and the quality of the dentition site. We found that various parameters affect how forces are transferred; with the impact site having the most effect, along with the impact configuration and dentition integrity. The Von Mises illustration pressures on the brain allows us to visualize more importants pressure areas at the occiput and the brainstem during an uppercut mandibular impact.

FEM parameters may be adapted to individual characteristics to suit realistic situation; which is a major advantage for forensic investigations and legal expertise.

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Figures and Tables

Figure 1: 3D Head model (sagittal section)

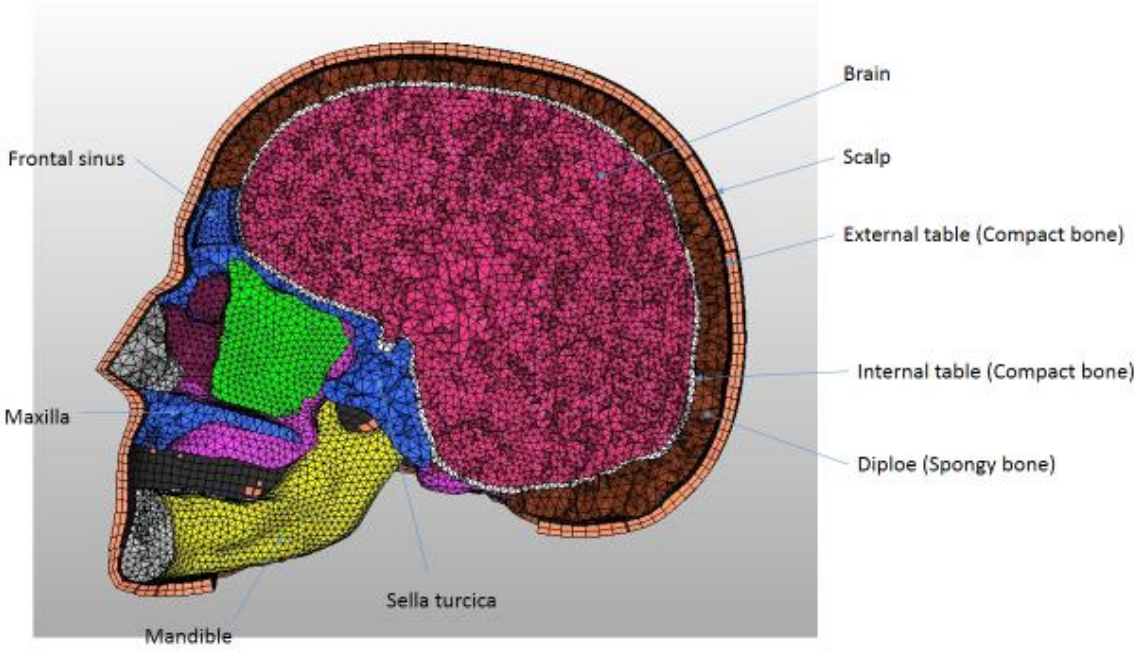

Figure 2: Mandible condylar neck fracture

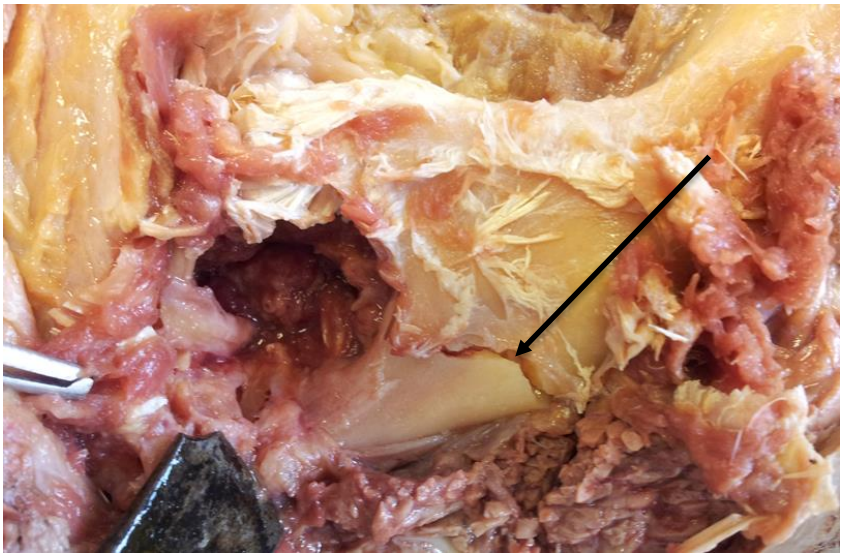

Figure 3: Uppercut simulation. Von Mises stress distribution at 0, 3, 6 and 9 ms. (T=Time)

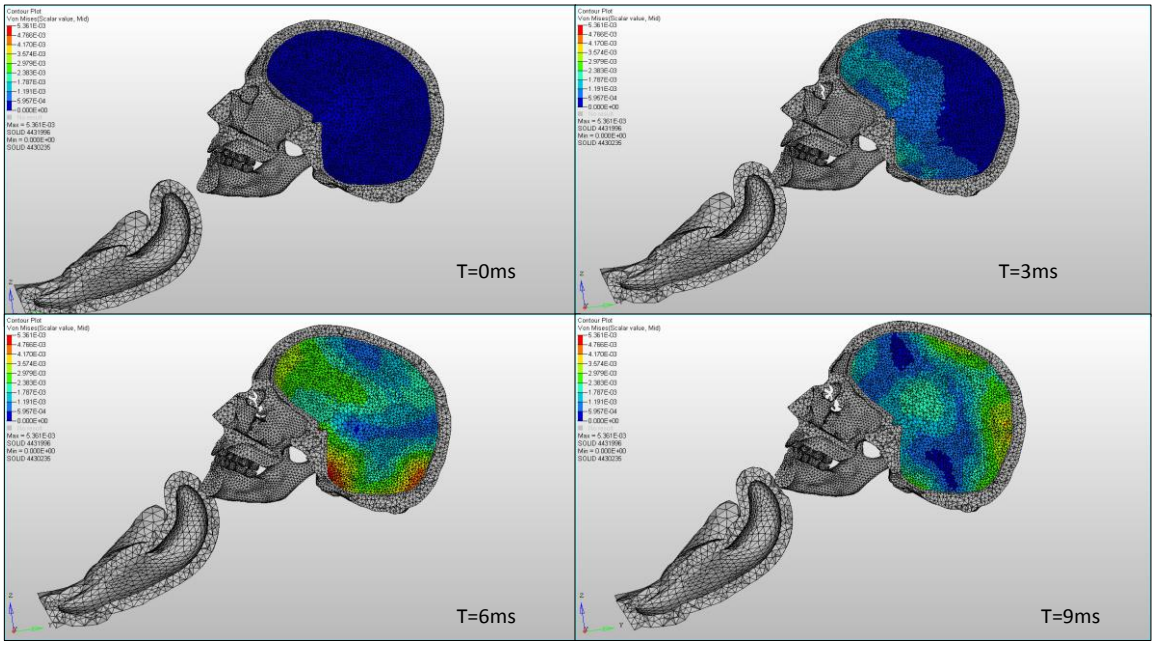

Table 1: Subject's anthropometric data

	Age (y.o.)	**Gender**	**Height (cm)**	**Weight (kg)**	**Dentition**
Subject n°1	75	Female	154	43	Complete
Subject n°2	≥ 75	Male	173	94	Incomplete
Subject n°3	≥ 75	Male	171	79	Incomplete

Table 2: Forces in Newton (N) from experimental trials tab

Trials	**Forces (N)**
Subject n°1	2608.2749
Subject n°2	2462.544
Subject n°3	4508.1568

Table 4: Validation of the finite element model compared to the results of experimental trials of Schneider and Viano.

					FEM
Authors	Impact site	Impactor mass (kg)	Impact velocity ($m.s^{-1}$)	Forces (N)	Forces (N)
Schneider et al.	anteroposterior	3,12	4,87-5,44	de 1840 à 4000	3700
	lateral	3,81	5,44-5,97	820-3400	2900
Experimental trials	uppercut	5	5	2400-4500	2500
Viano et al.	anteroposterior (jaw)	1,67	6,7	1010-4090	2200
	uppercut	1,67	6,7	458-3330	3000
	lateral (Hook)	1,67	6,7	994-9950	2300

Table 5: Forces measured for all simulations in Newton (N)

Anatomical site	Chin	condyles/ skull base	occiput
Impact velocity ($m.s^{-1}$)			
4	2700	2120	680
5	2400	1500	650
6,7	3000	1800	970
8	2900	1750	780 second peak at 1700
Cortical bone thickness (mm)			
1,5mm	3000	1800	970
3mm	2400	1900	740
5mm	2300	1880	550 second peak at 1900
Impactor mass (kg)			
1,2	2300	1680	800
1,7	3000	1800	970
2,2	3200	1400	880 second peak at 3400
Impact site			
Lateral	2300	2200	600
Frontal	2200	2200	850
uppercut	3000	1800	970
dentition			
Lower toothless	3500	3500	800
Upper toothless	2800	3200	800
Complete toothless	3500	3600	680
Complete	3000	1800	970
Young's modulus (MPa)			
10000	2500	1900	800 second peak at 2200
13700	3000	1800	970
density ($kg.m^{-3}$)			
1500	2250	1600	800
2500	3000	1800	970

Anatomical component	Property	Thickness (mm)	Density ρ (kg/m³)	Young's Modulus E (MPa)	Poisson coefficient v	σ c: Ultimate stress failure in compression	σ t: Ultimate stress failure in tension	σ max
Trabecular bone	Elastoplatic		1500	4600	0,05	35 MPa	35 MPa	
Cortical bone	Elastoplatic	1.5	1900	15000	0,21	145 MPa	90 MPa	
Mandible	Elastoplatic	1.5	2500	13000	0,3			
Teeth: molare enamel surface		0.5	1800	84100	0,23			220
Teeth's dentin/ Alveolar surface				18600	0,31			
Scalp	Elastic	5	1000	16,7	0,42			
LCR	Elastic		1040	0,12	0,49			
Face	Elastic	1 à 3	2500	5000	0,23			55
Brain	Viscoelastic		1040	0,049				
Mandible articular disc			1050	44,1	0,4			

Table 3: FEM anatomical components properties.

II

3. Etude de la transmission des forces au cerveau et tronc cérébral avec couplage tête/ cou

Dans cette seconde étude, l'objectif était d'appronfondir notre compréhension des mécanismes lésionnels mis en jeu lors d'un impact au niveau de la face plus particulièremet d'évaluer la transmission des forces au cerveau et au tronc cérébral. Suite aux morts subites secondaires à un impact mandibulaire, nous voulions étudier cette région anatomique trop peu analysée.

La transmission des forces au cerveau et à la moelle épinière à partir d'impacts mandibulaires de type coup de poing a été évaluée par la création et la validation d'un modèle par éléments finis complet de tête/ cou. Les impacts de type uppercut et jaw (antéro postérieur) sont associés à un étirement important et de fortes contraintes au niveau de la jonction du tronc cérébral et de la moelle épinière. Les impacts de type hook (crochet) entrainent une transmission directe des forces au tronc cérébral et à la moelle sans étirement de celle-ci. Les décès secondaires à ce type d'impacts sans lésions histologiques retrouvées peuvent être liés à une hyper sollicitation du tronc cérébral, lieu de passage des voies sensitivomotrices et du nerf vague et centre de régulation des grandes fonctions végétatives. Les paramètres biologiques variant selon chaque individu, la modélisation numérique permet de les moduler à l'infini (forme de mandibule, dentition…) pour une approche réaliste d'applications médico-légales.

Cet article a été accepté au Forensic Sciences International sous réserve de corrections (cf annexe 2)

Study of cerebrospinal injuries by force transmission secondary to mandibular impacts using a finite element model

Lucile Tuchtan [a,b,*], Yves Godio-Raboutet [c,d], Clémence Delteil [a,b], Georges Léonetti [a,b], Marie-Dominique Piercecchi Marti [a,b], Lionel Thollon [c,d]

a *Forensic department, APHM, Hôpital de la Timone, 13385 Marseille, France*

b *Aix Marseille Univ, CNRS, EFS, ADES, Marseille, France*

c *Aix Marseille Univ, IFSTTAR, LBA, Marseille, France*

d *iLab-Spine (International Laboratory – Spine Imaging and Biomechanics)*

* Corresponding author. Tel.: +33 491482245; fax: +33 491923331

E-mail address: lucile.tuchtan@ap-hm.fr (Lucile Tuchtan)

Abstract

Brain and cervical injuries are often described after major facial impacts but rarely after low-intensity mandibular impacts. Force transmission to the brain and spinal cord from a mandibular impact such as a punch was evaluated by the creation and validation of a complete finite element model of the head and neck. Anteroposterior uppercut impacts on the jaw were associated with considerable extension and strong stresses at the junction of the brainstem and spinal cord. Hook punch impacts transmitted forces directly to the brainstem and the spinal cord without extension of the spinal cord. Deaths after this type of blow with no observed histological lesions may be related to excessive stressing of the brainstem, through which pass the sensory-motor pathways and the vagus nerve and which is the regulatory center of the major vegetative functions. Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (jaw shape, dentition…) for a realistic approach to forensic applications.

Keywords*:* Finite element model, Cerebrospinal injury, Force transmission, Brainstem, Mandibular impact

1. Introduction

High kinetic energy impacts to the skull are often associated with hemorrhagic brain and meningeal injuries whose mechanism has been examined in several studies and for various types of cranial impacts [1–5]. Boxers are subjected to craniofacial impacts, and even low-velocity impacts may be associated with major brain injuries because of the evident anatomical proximity between the face and the brain. Inertia effects may be observed when the head is violently shaken without a direct impact on the cranium (particularly in hyperextension) [3,6–8]. Movement of the head caused by a blow to the face can in itself cause direct concussion even if no anatomical injuries can immediately be observed, because the damage is axonal [9–12]. In literature, many authors demonstrated that the axon functional role can be altered even if it is not cut [11–14]. The brainstem pivots upon facial impact and suffers alterations by the subsequent shearing mechanisms [15]. Without fracture, the skull movement at impact may still have caused a direct brain contusion. Death may thus be secondary to force transmission to the brain, either by a so-called reflex mechanism that involves nerve conduction (by vagus nerve overstimulation) [16], or by central nervous system injury (axonal damage) [9,11,17].

When injuries cannot be identified by gross examination or histologically because of rapid death [18], the forensic and also sometimes clinical problem is how to relate facial trauma to injuries that led to an altered state of consciousness or even to sudden death. This issue may arise in criminal proceedings concerning voluntary acts of violence or involuntary events such as road accidents, and when no focal cerebral hemorrhage is identified although the victim died during the violence and no other cause of death has been identified. The digital approach is a means of attempting to understand mechanisms of injury arising from head/neck dynamics and also of simulating an event described by a third party. In this study, we firstly developed a finite element model of the head and neck in order to understand facial fracture mechanisms and to observe force transmission in the unit formed by the brain, brainstem and cervical spinal cord.

Secondly, we examined some examples of mandibular facial trauma in order to study the potential mechanisms of injury.

2. Materials and methods

2.1. Characteristics of the model

The finite element model that we developed is a combination of a head model developed by the Laboratory of Applied Biomechanics (LBA) and a neck model developed in collaboration with the École Polytechnique Montréal (EPM) (Fig.1).

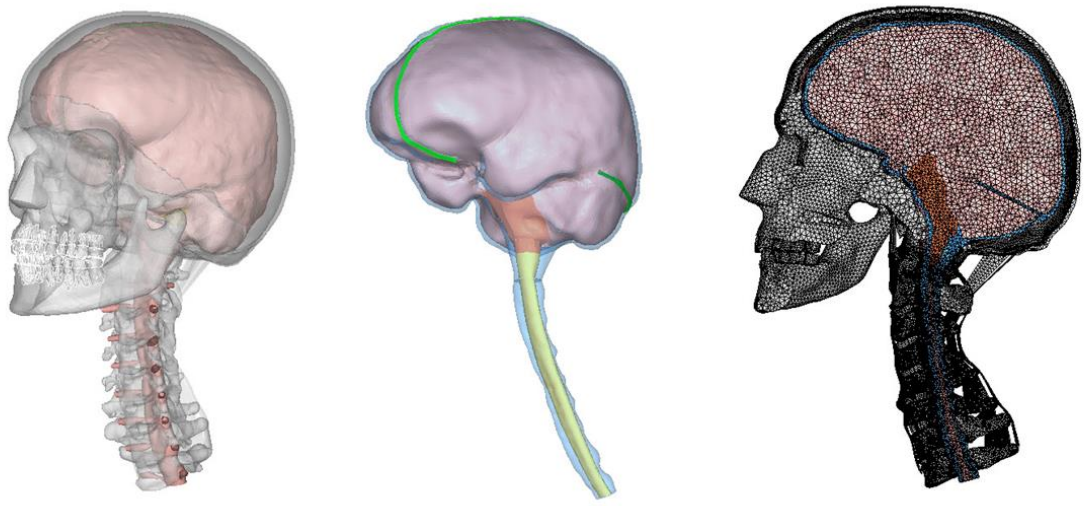

Fig 1: Finite element model of human head: a) whole model b) brain and spinal cord c) mid-saggital view.

Skull geometry was reconstructed from 1 mm Computerized Tomography (CT) scan slices of a 30-year-old man using MICMICS 12.3® software (Matérialise, Louvain, Belgium). The model was developed using Hypermesh® and Hypercrash® software (Altair Engineering, Inc., Detroit, MI, USA). The average size of the elements was 2 mm. The junction of the brainstem and the spinal cord was modeled in continuation of brainstem elements represented by tetrahedral elements. In the neck model developed by our laboratory (LBA, IFSTTAR in

collaboration with ILABSPINE) [18,19], only the elements of the vertebral bodies and the spinal cord were retained.

Because of the anatomical complexity of the brain, the meninges and the neck, each part (pia mater, dura mater, falx cerebri, tentorium cerebelli, hemispheres, cerebellum, brainstem, cervical spinal cord, cervical vertebrae and ligaments) was modeled according to its different mechanical properties. Continuous meshing was used.

The skull was reconstructed in three layers representing compact bone (external and internal tables) and cancellous bone (diploë) modeled using tetrahedral elements. The brain and subarachnoid space, comprised between the brain and the skull to simulate the cerebrospinal fluid, were also modeled using tetrahedral elements. The dura mater, falx cerebri and tentorium cerebelli were modeled with three-node shell elements. The cerebellum and brainstem were individualized. A boundary condition was applied on the C7 vertebrae (rotation and translation were fixed).

2.2. Validation of the model

The model underwent several evaluations: four different sources of validation (the studies of Nahum et al., Trosseille et al., and Viano et al. (Fig. 2) and our own experimental studies in the laboratory) based on the most relevant experiments in the literature and according to three different configurations (three mandibular impacts: uppercut (Fig. 3), hook (Fig. 4) and anteroposterior impact (Fig. 5)) in order to study the influence of point of impact at the level of the mandible.

1. Nahum et al.[2] (test 37): blow with a rigid cylindrical bar (mass 5.59 kg, impact velocity 9.94 m/s) on the frontal region of a seated post-mortem human subject (PMHS), with the torso supported. The blow was delivered in a sagittal plane and an

anteroposterior direction, with the subject's head inclined forward at 45° in the Frankfort plane (Fig. 2).

2. Trosseille et al. [20]: blow with a rigid iron bar (mass 23.4 kg, impact velocity 7 m/s) to the face of a seated PMHS in an anteroposterior direction (experiment MS 428-2) (Fig. 2). In order to evaluate the response of our model, we compared the force of impact at the level of the frontal region and acceleration of the head at the center of gravity using Nahum's tests. Pressures at the frontal and occipital regions were measured and evaluated according to the tests of Nahum and of Trosseille.
3. Viano et al. [21]: impacts on 3 mandibular areas with different impact velocities with a hand mass of 1.67 kg: jaw (9 2 m/s), hook (11 m/s) and uppercut (6.7 m/s) (Fig. 2).
4. Experiments carried out in our laboratory in 2015: blow with a rigid cylinder (mass 5 kg, impact velocity 5 m/s) on a PMHS in order to evaluate the force of the blow at the mandibular symphysis and to assess the type of fracture observed [22].

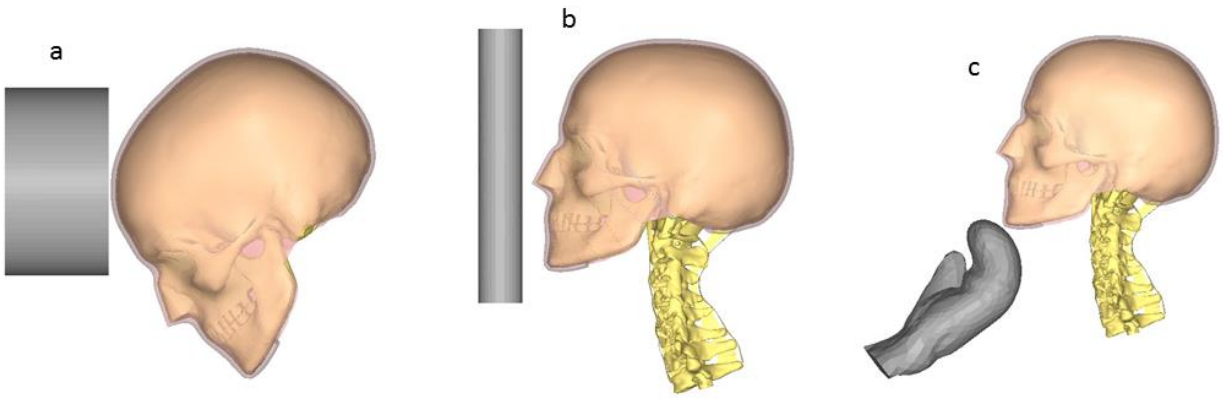

Fig 2 : Three numerical validation : Nahum et al (a), Trosseille et al (b), Viano et al (c).

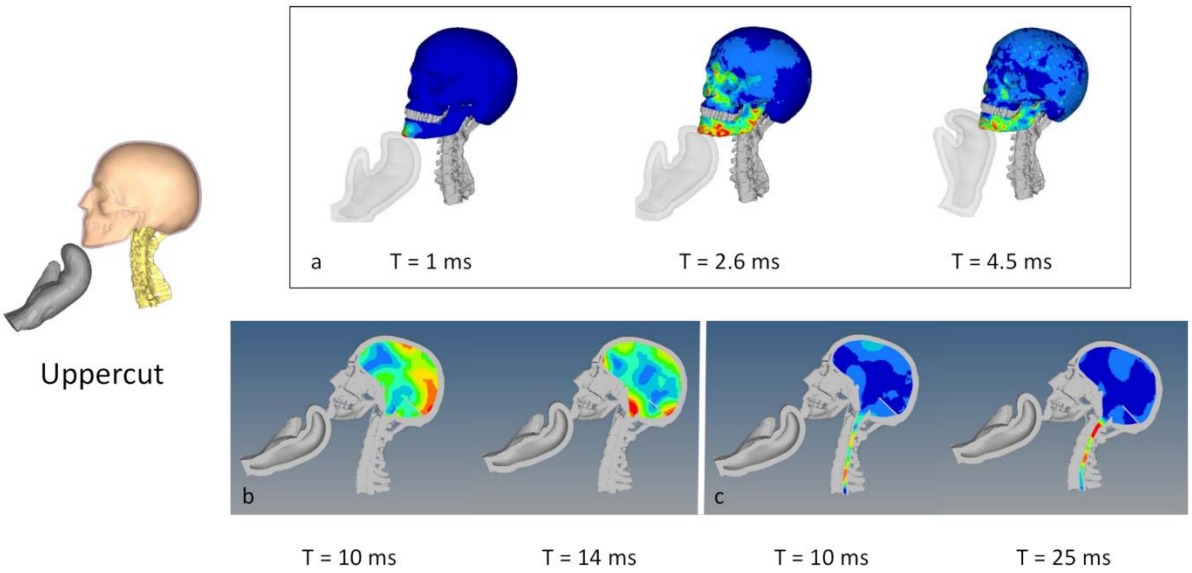

Fig 3: Stress propagation and distribution (Von Mises stress) in human head for uppercut scenario with focus on skull (a), brain (b) and cervical spinal cord (c).

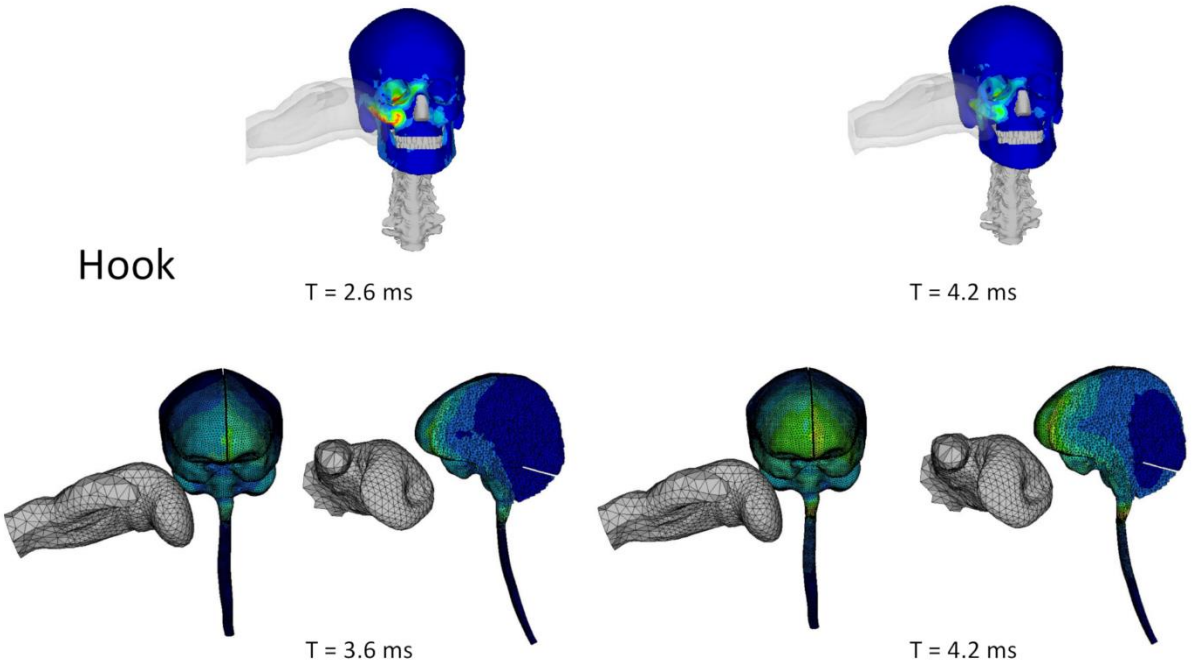

Fig 4: Stress propagation in human head for hook scenario with focus on skull and brain.

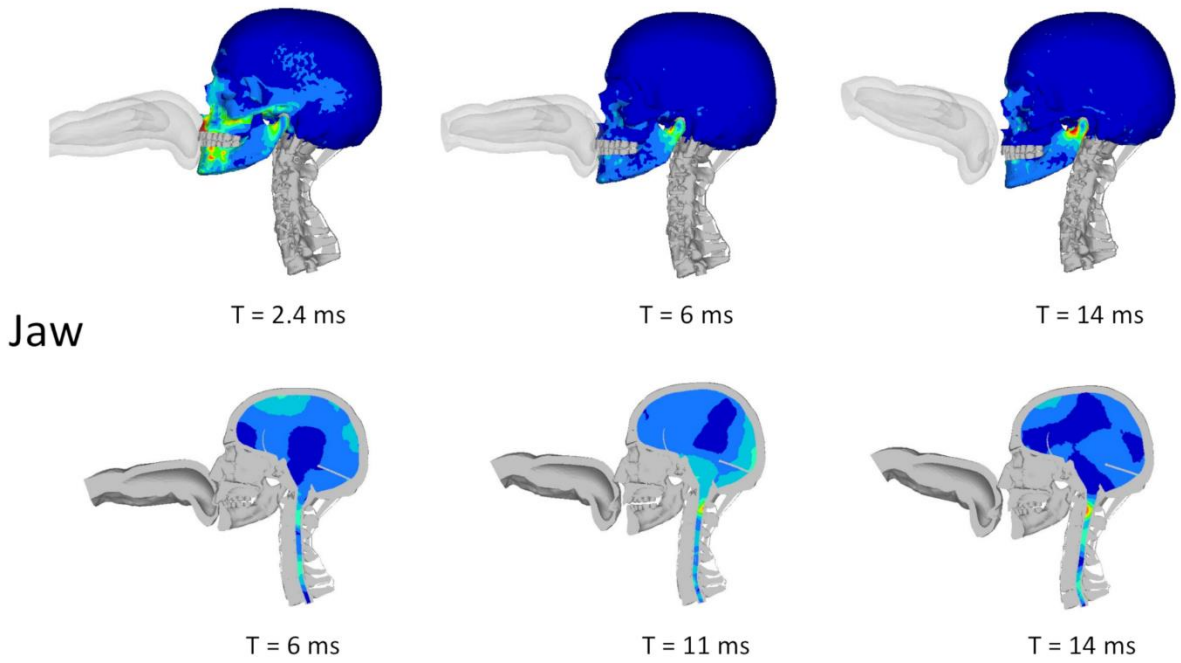

Fig 5: Stress propagation in human head for Jaw scenario with focus on skull and brain.

2.3. Resulting injuries

The injuries were examined at two levels: we observed firstly the distribution and propagation of stresses in the head, and secondly we evaluated the extension of the cervical spinal cord by measuring the space between the pons/medulla oblongata junction and C3.

The influence of impact location (uppercut, hook, face) on the development of stresses to the different parts of the brain (cerebrum, brainstem, cerebellum) and to the brainstem/spinal cord junction was also evaluated based on the tests of Viano et al. [18].

3. Results

The finite element model of the head consisted of 687,000 tetrahedral and hexahedral 3D elements and 85,000 shell elements (3 and 4 nodes). The mechanical parameters attributed to each anatomical part were based on the data of the literature (Table 1).

The finite element model of the neck consisted of 368,000 tetrahedral and hexahedral 3D elements and 122,000 shell elements (3 and 4 nodes). The head and neck together had a mass of 5580 grams.

We validated our digital model by reproducing the tests of Nahum, Trosseille and Viano. The comparative tests are summarized in the following table 2.

Anatomical components	Properties of the materials	Thickness (mm)	Density ρ (kg.m^{-3})	Young's modulus E (MPa)	Poisson coefficient v	σ c: compressive yield stress	σ t: tensile yield stress	σ max	Ref.
Cancellous bone	elastoplastic		1500	4600	0.05	35 MPa	35 MPa		[23]
Cortical bone	elastoplastic	1.5	1900	15000	0.21	145 MPa	90 MPa		[23]
Mandible	elastoplastic	1.5	2500	13000	0.3				[24]
Teeth: enamel surface crown		0.5	1800	41000	0.3			220	[24]
Dentin density/alveolar area				18600	0.31				[24]
Scalp	elastic	5	1000	16.7	0.42				[23]
CSF	elastic		1040	0.12	0.49				[23]
Face	elastic	1 to 3	2500	5000	0.23			55	[23]
Brain	viscoelastic		1200	1225					[23]
Disc of the mandibular joint			1050	44.1	0.4				[25]
Dura mater	elastic	0.5		5	0.45				[26]
Pia mater	elastic	0.1		2.3	0.45				[26]
Vertebral cortical bone	elastoplastic	0.37-0.9		3319	0.3				[19]

Table 1. Mechanical properties of the anatomical elements of the finite element model

Authors	Impact site	Impactor mass (kg)	Impact velocity (m/s)	Forces (N)		Pressure (MPa)		Acceleration (m/s^2)	
				Literature	Digital simulations	Literature	Digital simulations	Literature	Digital simulations
Nahum et al. (1977)	Anteroposterior	5.59	9.94	8000N	9800N	Frontal 0.12 MPa Posterior fossa 0.08 MPa	Frontal 0.14 MPa Posterior fossa 0.06 MPa	2034	2019
Trosseille et al. (1992)	Anteroposterior	23.4	7	X	X	Frontal 0.09 MPa Occipital 0.018 MPa	Frontal 0.12MPa Occipital 0.011 MPa	X	X
Viano et al. (2005)	Anteroposterior Jaw Uppercut Hook	1.67	9.2 6.7 11	2349 SD 962N 1546 SD 857N 4405 SD 2318N	4300N 2300N 5080N	X	X	X	X
Experimental test (LBA)	Uppercut	5	5	3150 ± 1141 N	2600N	X	X	X	X

Table 2: Conditions of evaluation of the model in relation to the literature

The results obtained (Table 2) were in agreement with the data of the literature concerning impact forces, head acceleration (Nahum's tests) and the different pressures measured in brain tissue.

3.1. Variation of impact location based on Viano's tests

- Uppercut (Fig. 3)

Forces were propagated in an anterior to posterior direction, were greatest at the junction of the brainstem and the spinal cord, and were associated with strong stresses at the occiput with a backlash effect that was visualized by pressures at the frontal level. Stresses were distributed at the level of the mandible extending to the condyles and then to the base of the skull, and were also distributed to the cheekbone. Forces spread to the frontal bone, ethmoid bone and nasal bone. The force of the impact caused considerable cervical extension that led to strong stresses at the level of the cervical spinal cord and the brainstem. Major stresses were visualized at the origin of the mandibular symphysis without condylar fracture. Extension of the cervical spinal cord was 2.6 mm.

- Hook (Fig. 4)

Simulations carried out on the finite element model revealed no fracture. The extension observed was very small (0.1 mm). Forces were propagated from the punch at the temporomandibular joint to the opposite side at the temporal level, with stresses mainly exerted at the junction of the brainstem and the spinal cord, frontal regions and at the falx cerebri.

- Anteroposterior (Fig. 5)

Simulations carried out on the finite element model in the anteroposterior direction on the jaw revealed that forces were propagated from the incisors to the maxillary arches, the septum, the ethmoid bone and then towards the palate and the sphenoid bone. These stresses were distributed in the frontal and temporal regions up to the occiput and the foramen magnum. In

brain tissue, stresses were propagated from the frontotemporal lobe to the occipital lobe, with greatest stress at the junction of the brainstem and the cervical spinal cord. A minimal fracture of the mandibular symphysis was visualized, with no penetrating or displaced fracture. Elongation of the cervical spinal cord was 2 mm, indicating cervical extension.

4. Discussion

4.1. Finite element model

Ours is one of the first complete models to include the fully modeled face and jaw, to which we added a neck model validated by the Laboratory of Applied Biomechanics in collaboration with the École Polytechnique de Montréal. Biomechanical studies have addressed mechanisms of injury either of the skull or of the spine, but not of the head and neck as a whole [18,19,23,27]. The relevance of this study is that it associates the cranial and cervical parts, which functionally are totally inseparable, in order to study their dynamics in facial impacts. Moreover, addition of the neck to our initial model allowed us to visualize the stresses exerted on the brainstem and the cervical spinal cord through three different mandibular impacts.

4.2. Force transmission and influence of impact location

Visible craniocerebral injuries (fractures, brain hemorrhages, contusions) give us little difficulty in understanding the mechanism involved in force transmission in a craniofacial impact. Our study also examines non-visible injuries, that is, non-hemorrhagic axonal injuries responsible for altered neurological functions and leading to death.

In anteroposterior facial impacts such as uppercuts, we observed hyperextension of the spinal cord/brainstem junction together with major stresses in this area, but without the cranial fractures, around the foramen magnum in particular, that have sometimes been observed in other studies [6,8,17,28]. Forces were distributed along the mandible to the base of the cranium,

with stresses passing from the frontal lobes to the occipital lobes, associated with high pressure at the brainstem and along the spinal cord. In order to keep as close as possible to real-life situations, we carried out impacts of the types of punches received in boxing.

A hook-type punch, on the other hand, did not cause hyperextension of the spinal cord through their mechanism, but considerable force was propagated without decreased intensity of the stresses measured at the impact zone of the brainstem.

Previously published head and neck models only examined brain injuries, and did not address the dynamics and stresses of brain/spinal cord tissue of the head and neck as a whole. During impacts or falls on the chin, injury by elongation or even rupture of the cervical spinal cord have in fact been described in the literature but not measured [3,6–8,29,30]. Voigt et al. reported brainstem injuries produced not only by hyperextension or anteflexion but also by torsion or other forces applied to the head [31]. Depending on the type of accident, brainstem injuries (partial severance) have been reported in vehicle drivers or passengers in high-velocity impacts where the face or forehead hit the dashboard or windscreen.

Our findings after hook-type punches were in agreement with those of Zivković et al. [32]. The location of cranial impact associated with specific cranial fractures is predictive of the presence or absence of pontomedullary injury. Lateral and frontal impacts are associated with the absence of pontomedullary injury, whereas impacts on the chin and the absence of direct cranial trauma are associated with pontomedullary injury, as we confirmed in our study. Jaw impacts cause violent movement of the head responsible for immediate craniocervical dislocations that may cause indirect brainstem injury, generally pontomedullary, because the pontomedullary junction is the thinnest, and therefore the weakest, anatomic part of the brainstem [32]. As we described in our previous publication [22], during a mandibular impact kinetic energy is transmitted from the mandible to the temporomandibular joints and then to the base of the cranium and to the brain. In our tests, we did not observe any fracture of the skull

base around the foramen magnum because of the lower impact velocities and forces used. In both situations, transmission of the impact force was decreased. The energy would thus be sufficient to produce a pontomedullary injury, but not sufficient to produce a fracture of the skull base. The development of pontomedullary injuries is dependent on impact energy and also on the position of the fracture, and less dependent on head movement. Mandibular and facial impacts cause acceleration of the head and rotational acceleration of the brain, and with sufficient impact energy, they may lead to rotation and deformation of the brain responsible for the brainstem injuries that we identified. The brainstem is not only a central sensory-motor pathway, but also a regulatory center for the major vegetative functions: vigilance, heart rate, respiratory rate, in particular at the level of the medulla oblongata. Simple contusion or compression of the medulla oblongata can thus lead to loss of consciousness and vegetative dysregulation that may result in death.

4.3. Protection of the face, fractures and anthropometrics

Although there have been controversial findings on facial fractures and brain injuries [33–36], our previous study and that of Zivković et al. [22,32] demonstrate the role of the facial bones in absorbing energy, protecting the brain and brainstem from the transmission of high kinetic energies. Mandibular fractures occur essentially at the impact point of each location, thus decreasing by half the force transmitted to the brain, as shown in our first article [22] in frontal and uppercut impacts. In lateral impacts, forces are transmitted directly to the base of the skull and so to the brain, without extension of the spinal cord but without decrease of stresses at the junction of the brainstem and the spinal cord. This mechanism of energy transmission has been described by Tse et al., by Zandi and Seyed Hoseini and by Lee et al., [5,37,38]. Tse et al. stated that the facial fractures closest to the brain are a major risk factor for underlying brain injuries [4].

In the literature, numerous comparative clinical studies have examined the number of fractures in relation to the severity of the brain injuries observed and have established a correlation, but without analyzing the dynamics of the forces applied nor their velocities.

These descriptive studies do not take into account the unique characteristics of each individual and the multiple factors that intervene in the mechanism of injury: shape of the mandible, bone density, dentition, underlying disease conditions, age… Finite element modeling offers an alternative to experimental research, enabling the digital reproduction of situations of injury and the possibility of evaluating an infinite number of conditions and injuries.

4.4. Limitations

Our modeling of a skull, brain and its spinal cord made it possible to locate stresses that had no clinical consequences. However, it could not reproduce axonal injuries and the extremely complex brain interconnections of the various sensory-motor pathways. Differentiation of gray and white matter and fluid-structure modeling of the CSF and brain vessels need to be added to improve this model.

4.5. Future prospects

Understanding the influence of the mode of impact and of the victim's characteristics on the development of brain lesions is of major importance. But the unique nature of the impact is not the only factor that has to be taken into account. So in the light of these data, we hope to study the effect on brain tissue of multiple lower-energy impacts and also to modify the environment by evaluating the stress produced when the facial impact occurs when the individual is lying on the ground or immobilized against a hard surface.

5. Conclusion

Using digital simulations based on a finite element model of the head and neck, we were able to analyze extra- and intracranial injuries following different mandibular impacts. This digital study enabled us to confirm the involvement and extension of the brainstem and cervical spinal cord during low-velocity impacts. Deaths secondary to this type of impact without identifiable histological lesions may be related to excessive stressing of the brainstem, along which pass sensory-motor pathways and the vagus nerve, and which is a regulatory center for the major vegetative functions. Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (mandible shape, dentition…) for a realistic approach to forensic applications.

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Partie III

Corrélations cliniques

Implication du tronc cérébral dans la mort subite secondaire à des impacts mandibulaires ?

Des décès dans les suites d'impacts cranio-faciaux, sans lésions anatomiques ont été pris en charge dans le service de médecine légale de Marseille. Dans trois cas, les individus décédés avant l'arrivée des secours et probablement rapidement après une perte de connaissance immédiate, présentaient des lésions tégumentaires sur la face comme uniques témoins de violence. Chez des individus jeunes et en bonne santé, meme si l'origine du décès apparait comme traumatique, il est toujours recherché un état antérieur pouvant à minima interférer avec le mécanisme létal.

Lors d'un impact facial, le segment céphalique effectue un mouvement d'hyperextension contraint et violent. Deux mécanismes physiques contemporains et simultanés sont mis en jeu : un effet de contact au niveau de l'impact crânien à l'origine de lésions locales au point d'impact et un effet d'inertie, en raison de la mise en mouvement du segment céphalique (accélération) ou de l'arrêt de ce mouvement (décélération) à l'origine de lésions diffuses et multifocales. Ainsi le mouvement de la tête peut à lui seul causer une commotion cérébrale directe même s'il n'y a pas de lésions anatomiques objectivables immédiatement et ce de façon distincte d'éventuelles lésions médullaires par « coup de fouet » cervical plus décrites chez le nourrisson.

Nos recherches sur la transmission des forces à l'extrémité céphalique par la modélisation en éléments finis, retrouvent de fortes contraintes au niveau de la jonction cérébro-spinale.

En l'absence de lésions traumatiques identifiées hormis sur le revêtement cutané et en l'absence d'autres causes de décès mises en évidence, nous nous sommes intéressés à ce mécanisme lésionnel.

Nous rapportons ici, trois décès secondaires à des impacts faciaux présentant des lésions cérébrales uniquement visualisées lors de l'analyse anatomopathologique permettant de soutenir les mécanismes lésionnels traumatiques.

Cet article a été soumis au *International Journal of Legal Medecine* (cf annexe 3)

Case report

Sudden death after facial impacts: is the brainstem involved?

Lucile Tuchtan[1,2,*] • Clémence Delteil[1,2] • Yves Godio-Raboutet[3,4] • Georges Léonetti[1,2] • Lionel Thollon[3,4] • Marie-Dominique Piercecchi-Marti[1,2]

[1] Forensic Department, APHM, Hôpital de la Timone, 264 rue Saint Pierre, 13385 Marseille, France

[2] Aix Marseille Univ, CNRS, EFS, ADES, 27 avenue Jean Moulin, 13385 Marseille, France

[3] Aix Marseille Univ, IFSTTAR, LBA, boulevard Pierre Bramard 13015 Marseille, France

[4] iLab-Spine (International Laboratory – Spine Imaging and Biomechanics), boulevard Pierre Bramard 13015 Marseille, France

* Corresponding author. Tel.: +33 491482245; fax: +33 491923331

E-mail address: lucile.tuchtan@ap-hm.fr (Lucile Tuchtan)

Lucile Tuchtan ORCID: 0000-0001-6248-2840

Clémence Delteil ORCID: 0000-0001-5171-8520

Lionel Thollon ORCID: 0000-0002-4456-0902

Abstract

Three deaths following facial impacts in the presence of witnesses and resulting in brain lesions that were visualized only on histopathological examination were studied at the forensic medicine institute of Marseille. Craniofacial impacts, even of low intensity, received during brawls may be associated with brain lesions ranging from a simple knock-out to fatal injuries. In criminal cases that are brought to court, even by autopsy it is still difficult to establish a direct link between the violence of the impact and the injuries that resulted in death. During a facial impact, the head undergoes a movement of violent forced hyperextension. Death may thus be secondary to the transmission of forces to the brain, either by a mechanism involving nerve conduction that may be termed a reflex mechanism (for example by vagal hyperstimulation) or by injury to the central nervous system (axonal damage). In such situations, autopsy does not make it possible to determine the cause of death, but only to suspect it in a context of voluntary violence in the presence of witnesses, with or without violent injury observed on external examination or on superficial incisions to determine the extent of bruises or hematoma. Only histological analysis is contributory.

Keywords: Head trauma • Brainstem • Axonal injury • Mandibular impact

Introduction

Craniofacial impacts are well known to boxers, but even low-intensity impacts received during brawls may be associated with brain injury ranging from simple loss of consciousness to fatal lesions [1–3]. In criminal cases, the judge cannot consider that a compatible chronological course of events is sufficient to affirm causality, even if several witnesses were present. The difficulty of establishing the link between the violence of the impact and the injury responsible for death is still present at autopsy, and further investigations are required, histopathology in particular.

During a facial impact, the head undergoes a movement of forced violent hyperextension. Two simultaneous and concurrent physical mechanisms come into play: a contact effect resulting in local lesions at the point of cranial impact, and an inertia effect when the head is set in motion (acceleration) or when motion ceases (deceleration), which causes diffuse and multifocal lesions. The movement of the head can in itself cause direct concussion even if there are no immediately evident anatomic lesions, and independently of possible cervical cord injury due to cervical "whiplash" injury which is more frequently described in the infant [4]. In practice, the contact effect and the inertia effect act together to produce contusion lesions at the surface of the brain and/or axonal lesions and/or suspension of nerve function with the possibility of axonal involvement causing deafferentation.

Death may thus be secondary to transmission of force to the brain either by a mechanism involving nerve conduction, known as a reflex mechanism (for example by vagal hyperstimulation) [5–8], or by central nervous system lesions (axonal damage) [9–12].

In such situations, autopsy does not make it possible to determine the cause of death, but only to suspect it in a context of voluntary violence in the presence of witnesses, with or without violent injury observed on external examination or superficial incisions to determine the extent

of bruises or hematoma. We report three cases of deaths following facial impacts where the victims presented with brain lesions that were visualized only on pathological analysis, which made it possible to support the mechanisms of traumatic injury.

Case reports

Case 1

A 32-year-old man was assaulted, in the presence of several witnesses who gave identical accounts of the event, by several persons who delivered four or five uppercut type punches while he had his back against a wall and then threw him to the ground. The victim was described as already unconscious when he received a kick in the face. According to a witness who assisted him immediately after the event, the victim appeared to be alive. The witness placed him in the lateral security position and maintained the neck and head in the axis of the trunk with his hand. Respiratory pauses occurred. When the professional emergency services took over 20 minutes later, the victim was in cardiorespiratory arrest. He was declared dead 45 minutes later in spite of transient return of circulation. At autopsy, only superficial external injuries were found in the facial and cervical region, such as abrasions of the brow bone, left eyelid, left cheek and the posterior cervical region, as well as rib fractures. No fractures of the skull, face or neck or peri-, intracerebral or spinal cord hemorrhage were observed. However, the victim had a hypertrophic heart which pointed to decompensation of a pre-existing state rather than to a direct effect of the blows received, particularly as toxicology later showed a high blood alcohol level (2.28 g/L). The pathology expert report confirmed the hypertrophic cardiomyopathy, but was not able to define its cause or affirm its role in the lethal mechanism of a previously unknown disorder. The report confirmed the absence of intracranial hemorrhagic changes. However, immunostaining with anti-beta-amyloid precursor protein (APP) antibodies revealed axonal lesions of the pedoncles, pons, medulla oblongata and the nerve roots. Lesions in this

location produce respiratory and then circulatory arrest, which appeared to be compatible with the victim's clinical state as described by the witnesses and were in support of a direct link between the acts of violence and death.

Fig. 1 Axonal lesions of the medulla oblongata (x 100) (A) and upper medulla oblongata (x 100) (B).

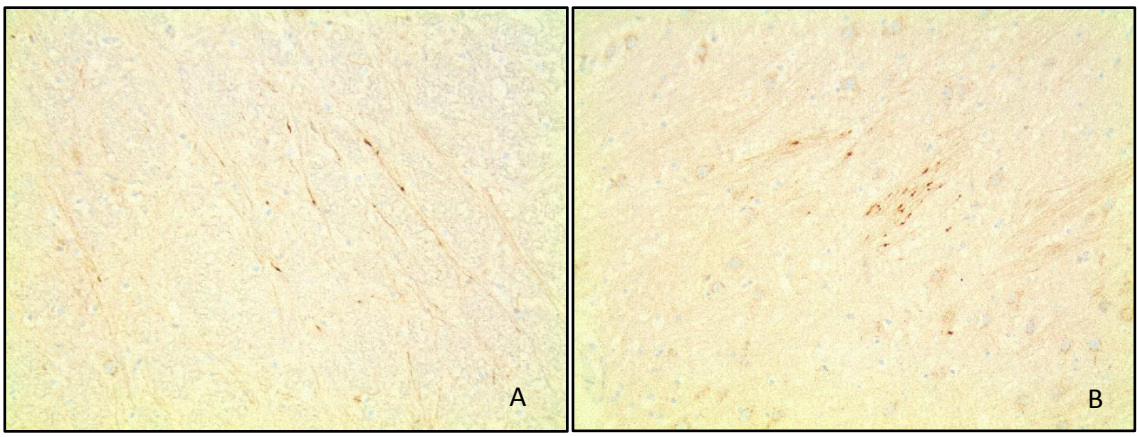
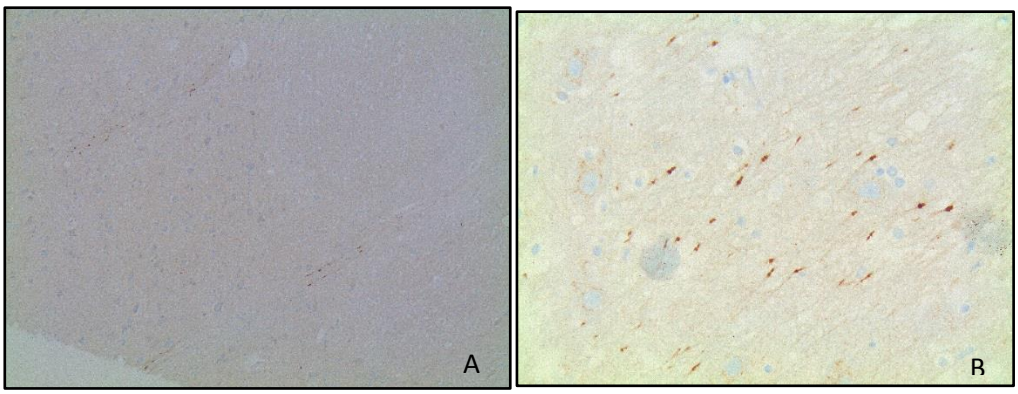


Fig. 2 Axonal lesions of the peduncles x 50 (A) and x 200 (B)



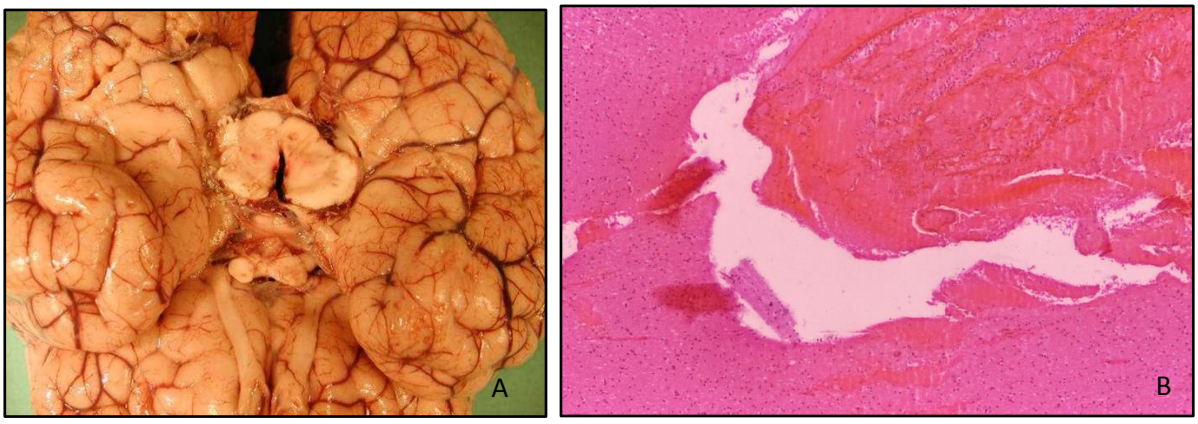
Case 2

A 14-year-old adolescent, who had recently been admitted to a young people's home, received before witnesses a kick in the face following a dispute with another adolescent. According to the witnesses, he immediately had an altered state of consciousness which was rapidly followed by cyanosis and respiratory arrest, then by a convulsive episode and urinary incontinence before

circulatory arrest. Cardiac activity was restored after 40 min of resuscitation but the adolescent progressed to brain death one day later. Organ removal was performed (liver, kidneys). Antemortem examination of his injuries revealed fracture of the nasal bones but no brain hemorrhage. The initial brain CT scan showed no abnormality of supra- or subtentorial density. No spinal bone lesions were identified. The autopsy revealed no skin lesions and in particular no facial lesions. Only cerebral edema associated with a thin hemorrhagic film next to the brainstem was observed on gross examination.

As well as anoxic-ischemic edema, pathological examination revealed marked median laceration of the brain peduncles passing through the ventricle, with hemorrhagic margins. This laceration accompanied by axonal lesions was interpreted as being of traumatic origin secondary to the violent facial impact.

Fig. 3 Inferior view of the brain showing transection of the peduncle: marked median laceration (A). View of a histological section (B).



Case 3

A 42-year-old man was found dying on the ground in front of his home by his female friend. When the emergency services arrived, he was in cardiorespiratory arrest. Resuscitation was

attempted for 40 min but was unsuccessful. At the scene, there was a blood stain under the victim's head with splashes of blood. His friend had last seen him on the night before the event during an evening of drinking at their home, where a third person was believed to have been present. As the street was equipped with security cameras, it was possible to observe the circumstances of death. An individual had delivered a series of punches to the forearms (the victim initially attempted to protect himself), the trunk and lastly to the face, with an uppercut that caused the victim's immediate collapse. At autopsy, numerous facial and cervical bruises were found, associated with underlying hemorrhagic infiltrations without associated hemorrhagic brain lesions, and numerous bruises and hematomas of the limbs. Pathological examination found hemorrhagic laceration of the cerebral peduncles and axonal lesions in the same area.

Discussion

Even by autopsy, it is still currently difficult to establish a direct link between acts of violence and death. Supplementary investigations, and pathological examination in particular, are indispensable to identify lesions that are not visible on external gross examination and so to explain the mechanism of injury. In the course of legal proceedings, the judge cannot simply accept a compatible chronological chain of events described by several "objective" witnesses present at the scene in order to affirm a definite causal link between the events and death unless he or she can rule out other causes of death. If there are no identifiable lesions, the mechanism of injury that is suggested results from well-grounded argumentation that rules out all possible diagnoses one by one, thus leaving a diagnosis of exclusion. Such arguments are not admissible by a tenacious lawyer, as witness reports are not considered as irrefutable evidence (vague description of the mechanism of injury, site of impact unclear).

The cases reported here of deaths following facial injuries, whose clinical consequences were described by impartial witnesses, are substantive evidence of the link between injury and death. The literature contains descriptions of brainstem lesions, with or without associated fractures, particularly after high-velocity impacts in road traffic accidents [7,13]. The cases we report show that punches or kicks can produce injuries that are just as severe. In these contexts of closed head and brain injury involving forces of acceleration or deceleration, lesions have been described in the central axial regions of the brain [14], including at the level of the brainstem lesions of the superior colliculi, the roof of the fourth ventricle, the medial reticular formation (including the dorsal medullary nuclei), the middle and lower cerebral peduncles, and the pons.

The hypothesis of a reflex mechanism during intense stretching of the brainstem is a probable one because of the passage of the major sensory motor pathways, the reticular formation (including the pre-Bötzinger complex), and the presence of central nuclei in this pivotal zone [15,16]. The vasomotor centers and the central pattern generator for respiration lie in the brainstem. These centers are responsible for short-term regulation of arterial blood pressure and for the nerve supply of the muscles of the thoracic cavity and the upper airways, respectively. Involvement of these centers may lead to death due to considerable slowing of the heart rate or respiratory arrest. Our subjects were young and in good health. Only one had a pre-existing heart condition which could have decompensated as a result of stress after blows to the trunk, causing cardiac arrest. This point, which initially appears of little significance, was of fundamental importance for the magistrates, in particular enabling them to bring a charge against the persons accused.

In order to prove axonal damage of traumatic origin, the pathologist must be alerted of the need to obtain samples in target areas such as the peduncles and the medial axis of the brain in general, as in suspected shaken baby syndrome [14,17]. However, anti-beta APP antibody immunostaining is required to reveal these axonal lesions. When neurons are damaged, beta-

amyloid precursor protein accumulates in the axons due to inhibition of axonal transport [18]. In the adult, axonal lesions can be observed when survival is longer than 2 to 3 hours [19–22]. Interpretation of anti-APP immunostaining must follow fundamental principles: the duration of survival after cardiocirculatory arrest, thanks to resuscitation measures, must be sufficiently long, and vascular perfusion of the brain must be adequate during survival. Conversely, survival for too long a period may result in a false negative result [23].

Furthermore, too long a survival time can lead to torsion of the peduncles because of subtentorial involvement, leading to Duret hemorrhage (transtentorial herniation) through ischemia, which may be either masked or mistaken for traumatic lesions [24].

The marrow of the cervical spine is also affected by hyperextension of the neck [25] and it is indispensable to systematically obtain samples.

In the literature, some authors such as Belingardi et al. have demonstrated that the brainstem acts as a pivot in a facial impact and that it undergoes major damage during shear stress [26]. Lesions of varying degrees of severity may result, from transient local locomotor paralysis to a range of more or less extensive parenchymal lesions. These effects of inertia may be observed when the head is violently shaken without an impact (particularly in hyperextension): for example, a driver who has a whiplash injury when his vehicle is hit from behind, a rugby player or footballer pushed from behind, a boxer who receives an uppercut, or a helmeted head that receives an impact.

Another team, that of Gennarelli et al., studied the conditions in which diffuse axonal lesions are produced, and concluded that the functional consequences following loss of consciousness were directly related to axonal destruction at the time of impact [9]. Other studies have shown that the axons could be functionally altered even if they were not transected [10,12,27,28]. The lesions due to stretching are found in areas of low axonal resistance: the transitional zones in

the brain between white and gray matter, the periventricular white matter, the corpus callosum, and extremely severely in the dorsal midbrain. For some years, biomechanical studies have been performed both experimentally on bodies donated to science and by simulation using digital image-based finite element models [29–31]. The digital approach improves our understanding of the mechanisms of injury involved and scenarios of injury can be reproduced ad infinitum.

Energy transmission to the skull following mandibular impacts has been reported to produce stresses affecting the brainstem but without associated cranial fractures, in particular following uppercut punches [32] The presence of axonal lesions confirms the stresses produced at this transitional zone.

Conclusion

Death following facial trauma with no identifiable or with only microscopic lesions is rare and this is a unique description of three cases. These anecdotal cases confirm that stretching of the brain stem can cause death by sideration and/or axonal lesions. This mechanism of injury is no longer suggested by elimination only, but becomes true evidence that can be defended in court before the magistrates.

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Partie IV

Discussion /Conclusion générale

La difficulté d'établir le lien direct entre les faits de violence et le décès au moment de l'autopsie reste encore présente de nos jours. Des décès secondaires à un impact mandibulaire, nous ont amené à nous interroger sur le mécanisme lésionnel engendré. L'apport des examens complémentaires et en particulier anatomopathologique est incontournable afin d'identifier ces lésions non visibles lors de l'examen macroscopique externe et d'expliquer ainsi le mécanisme lésionnel. Cependant l'examen anatomopathologique peut aussi être non informatif, si la mort survient très rapidement. En effet, l'apparition de lésions microscopiques axonales nécessite un délai de survie qui selon la littérature peut varier entre 1 et 3 heures [71–74]. Ainsi, l'absence de lésions visibles à l'examen histopathologique ne présage pas de l'absence de mécanisme traumatique ou de lésions axonales non encore visibles.

Sans lésions identifiables, le mécanisme lésionnel évoqué résulte d'une argumentation étayée éliminant tour à tour tous les diagnostics possibles, laissant place ainsi à un diagnostic d'élimination ce qui n'est pas reçu comme élément de preuve dans la procédure. La modélisation par éléments finis offre une alternative à la recherche expérimentale permettant une reproduction de situations lésionnelles numériques et ainsi une évaluation des conditions et des lésions à l'infini.

La transmission des forces au cerveau et à la moelle épinière est un sujet relativement peu abordé dans la littérature.

Ainsi notre premier modèle s'est tout d'abord axé sur la transmission des forces au crâne ainsi

que sur une visualisation des pressions exercées sur le cerveau. Celui-ci a permis de confirmer qu'un impact mandibulaire peut générer, par transmission des forces, des lésions mésencéphaliques visualisées selon la propagation des contraintes de Von Mises.

Jusqu'ici les études publiées sur des modèles par éléments finis (MEF) s'étaient intéressées aux coups portés sur la face (Bruyère et al, Yoganandan et al, Allsop et al, Schneider et al…) [40,41,75]et en particulier, aux impacts frontaux ou maxillaires [41,75] que l'on retrouve dans les accidents de la route (Yoganandan et al [75,76]) lors de la percussion du volant dans le cadre des études pour la prévention routière. Les rares études sur les coups de type uppercut s'attachaient à rechercher la tolérance de la mandibule à l'impact, sur ses propriétés mécaniques ou encore sur la mastication [67–69].

Grace à la modélisation numérique, nous avons pu visualiser que la propagation des forces chemine dans le sens antéro-postérieur, avec parfois un phénomène de coup-contrecoup, associée à de **fortes contraintes au niveau du tronc cérébral :**
Les impacts mandibulaires **latéraux et frontaux transmettent directement** leurs forces au niveau de la base du crâne dans ce premier modèle.

Concernant les variabilités biologiques comme la présence de dents, celles-ci permettrait d'absorber une partie de l'énergie transmise. En effet, **l'absence de dentition en bas, en haut ou complète n'atténue pas ou peu la transmission des forces**. L'absence de dents entraine la disparition de l'os alvéolaire et ainsi une diminution des surfaces s'intercalant entre l'impacteur et la base du crâne. Une étude réalisée par Walilko et al [13] sur le rôle du protège-dents bi-maxillaires montre une réduction de plus de la moitié de l'effort mesuré au niveau de la fosse mandibulaire de la base du crâne. La superposition de ces matériaux diminue la transmission de l'effort. Dans notre étude, nous avons choisi de tester deux situations extrêmes, avec ou sans dents, mais ces données doivent être modulées selon la nature des

dents restantes (altérées ou non, le nombre de dents restantes, la présence d'un matériel prothétique, etc.). Les paramètres choisis étaient liés à la présence de dents et non à l'âge qui peut aussi par diminution de la densité osseuse, en plus de la variabilité individuelle, avoir une influence sur la transmission des efforts.

De même, l'augmentation progressive de l'épaisseur corticale entraine une diminution d'absorption de l'énergie avec une transmission directe des efforts à la base du crâne.

Aussi, une diminution du module de rigidité et de densité induit une baisse de la force mesurée au point d'impact (menton), favorisant les fractures par absorption d'énergie.

L'augmentation de la vitesse à l'impact testée lors des simulations du premier modèle, n'a entrainé que de faibles variations d'efforts au niveau du menton et des condyles alors que l'énergie massivement transmise à l'arrière du crâne, crée pour la vitesse la plus haute, un effet de coup-contrecoup croissant. Nous avons choisi comme vitesse de base celle de 6.7 $m.s^1$ en référence à l'étude de Viano et al [58] (vitesse moyenne correspondant à un individu donnant un uppercut). De même, les efforts mesurés sur les percussions frontales de la mâchoire (Jaw) et latérales (Hook) sont similaires aux efforts retrouvés sur notre modèle numérique.

La diminution de la masse d'impact du gant a peu influencé la transmission des forces en dehors d'un effort mesuré au menton.

Dans cette première étude, l'observation des contraintes de Von Mises au niveau cérébral révèle une répartition des efforts d'avant en arrière, passant par les lobes temporaux et se terminant au niveau du tronc cérébral. L'équipe de Viano et al [58] **retrouvait aussi, suite à un coup latéral, tout d'abord des contraintes au niveau temporal puis secondairement, une majoration de celles-ci au niveau mésencéphalique.** De même,

l'équipe Belingardi et al [3], qui a développé un modèle par éléments finis de la tête validé par les résultats de Nahum 1977 [48], a aussi mesuré les pressions cérébrales et a relevé sur les sections coronales des contraintes importantes au niveau du tronc cérébral confirmant ainsi son rôle de pivot lors de mouvements du cerveau.

Modèle de tête et assemblage du complexe tête/cou

Les résultats obtenus par ce premier modèle, nous ont permis de nous focaliser sur la région du tronc cérébral. Ainsi nous avons amélioré notre premier modèle de tête et ajouté un modèle de cou validé par le Laboratoire de Biomécanique Appliquée en collaboration avec l'École Polytechnique de Montréal. Actuellement, les études biomécaniques s'intéressent aux mécanismes lésionnels soit de la partie crânienne soit de la colonne vertébrale mais pas de l'ensemble tête/cou [6,51,77,78]. La pertinence de cette étude est l'association de la partie crânienne et cervicale qui sont totalement indissociables fonctionnellement, afin d'en étudier la dynamique dans le cadre d'impacts faciaux. De plus, l'apport du cou à notre modèle initial, nous permet de visualiser les contraintes exercées au niveau du tronc cérébral et de la moelle épinière cervicale, à travers 3 impacts mandibulaires.

Nous nous sommes intéressés particulièrement aux lésions non visibles macroscopiquement c'est-à-dire aux lésions non hémorragiques mais axonales entrainant des altérations des fonctions neurologiques jusqu'au décès.

Lors **d'impacts faciaux antéro-postérieurs et de type uppercut**, nous observons une **hyperextension de la jonction moelle épinière/tronc cérébral associée à des contraintes importantes de cette zone**, sans fractures crâniennes objectivées en particulier au pourtour du foramen magnum décrites parfois dans d'autres études [79–82]. La propagation des forces

s'effectuent le long de la mandibule jusqu'à la base du crâne avec des contraintes se propageant des lobes frontaux jusqu'aux lobes occipitaux associée à une forte pression au niveau du tronc cérébral et le long de la moelle épinière. Afin de s'approcher au mieux de la réalité, nous avons choisi de réaliser des impacts types coups de poings.

L'impact de type crochet, en revanche, n'entraînent pas d'hyperextension de la moelle du fait de leur mécanisme mais **la propagation des forces est importante** sans diminution d'intensité des contraintes mesurées de la zone d'impact au tronc cérébral.

Lors d'impacts ou de chutes sur le menton, des lésions d'étirement voire de rupture de moelle cervicale par hyper extension sont décrits dans la littérature [79,80,83,84]. Voigt et al [85] ont rapporté des lésions du tronc cérébral associées à des fractures de la base du crâne produites non seulement par hyper extension ou ante flexion mais aussi par torsion ou autres forces appliquées sur la tête. Selon les différents modes d'accidents, les lésions du tronc cérébral (section partielle) ont été rapportées dans les cas de conducteurs ou passagers de véhicule, impliqués dans des accidents de haute vélocité, et dans les cas où la face ou le front ont tapé contre le tableau de bord ou le parebrise.

Les résultats obtenus lors de l'impact de type crochet sont en accord avec les travaux de l'équipe de Zivkovic et al[37]. En effet, la zone d'impact sur le crâne associée à des fractures crâniennes spécifiques est prédictive de la présence ou l'absence de lésions ponto-médullaires. Les impacts latéraux et frontaux sont associés à une absence de lésions ponto-médullaires. Alors que les **impacts mentonniers** et l'absence de traumatismes crâniens directs étaient associées à des **lésions ponto-médullaires**, ce que nous confirmons dans notre étude. Les impacts mandibulaires entraînent un violent mouvement de la tête, à l'origine de dislocations cranio cervicales instantanées qui peuvent être à l'origine de lésions indirectes du tronc cérébral, le plus souvent ponto médullaires, parce que la jonction ponto-médullaire est anatomiquement la

partie la plus fine du tronc cérébral et donc la plus faible [37]. Comme décrit dans notre premier article [38], lors **d'un impact mandibulaire, l'énergie cinétique est transmise de la mandibule aux articulations temporo mandibulaires puis à la base du crâne et au cerveau. Dans nos essais, nous ne visualisons pas de fractures de la base du crâne au pourtour du foramen magnum en raison de plus faibles vitesses et forces d'impacts utilisées. Dans les deux situations nous avons une décroissance de la transmission de la force d'impact**. L'énergie serait donc suffisante pour produire une lésion ponto médullaire mais insuffisante pour produire une fracture de la base du crâne. L'apparition de lésions ponto-médullaires dépend de l'énergie d'impact mais aussi de la position de la fracture et moins du mouvement de la tête. Les impacts mandibulaires et faciaux entraînent une décélération de la tête et une accélération en rotation du cerveau, et avec une énergie suffisante d'impact, ils peuvent conduire à une rotation et une déformation du cerveau jusqu'aux lésions du tronc cérébral que nous avons pu objectivées. Le tronc cérébral est un centre de passage des voies sensitives et motrices mais aussi un centre de régulation des grandes fonctions végétatives : vigilance, rythmes cardiaques et respiratoires, notamment au niveau du bulbe. Ainsi une simple contusion ou compression du bulbe peut être à l'origine d'une perte de connaissance, de dysrégulations végétatives pouvant aller jusqu'au décès.

Protection de la face, fractures et anthropométrie

Bien qu'il y ait des données controversées concernant les fractures faciales et les lésions cérébrales [34,86–88], notre étude et celle de Zivkovic, [37,38] démontrent **un rôle d'absorption d'énergie des os de la face, protégeant le cerveau et le tronc cérébral de la transmission de fortes énergies cinétiques**. Les fractures mandibulaires sont essentiellement retrouvées au point d'impact de chaque site (symphyses mandibulaire et maxillaire, condyles),

permettant une diminution de transmission des forces au cerveau de moitié comme démontré dans notre première étude [38] lors d'impacts frontaux et de type uppercut. Lors d'impacts latéraux, les forces sont transmises directement au niveau de la base du crâne et ainsi au cerveau, sans étirement de la moelle épinière mais sans diminution de contraintes au niveau de la jonction du tronc cérébral et de la moelle épinière. Ce mécanisme de transmission de l'énergie était décrit par les observations de Lee et al, Zandi and Seyed et Tse[31,89,90], mentionnant que les fractures faciales les plus proches du cerveau sont un facteur de risque important de lésions cérébrales sous-jacentes[91].

Dans la littérature, il est retrouvé de nombreuses études comparatives cliniques étudiant le nombre de fractures par rapport à la sévérité des lésions cérébrales retrouvées et établissant un lien de corrélation sans analyse de la dynamique des forces appliquées ni des vitesses.

Ces études descriptives ne prennent pas en compte le caractère unique de chaque individu et la multiplicité factorielle intervenant dans le mécanisme lésionnel : forme de mandibule, densité osseuse, dentition, pathologie sous-jacentes, âge….

Application clinique

La difficulté d'établir le lien entre la violence de l'impact et les lésions à l'origine du décès reste présente à l'autopsie nécessitant des examens complémentaires, anatomopathologiques notamment.

Lors d'un impact facial, le segment céphalique effectue un mouvement d'hyperextension contrainte et violente. Deux mécanismes physiques contemporains et simultanés sont mis en jeu : un effet de contact au niveau de l'impact crânien à l'origine de lésions locales au point d'impact et un effet d'inertie, en raison de la mise en mouvement du segment céphalique (accélération) ou de l'arrêt de ce mouvement (décélération) à l'origine de lésions diffuses et

multifocales. Ainsi le mouvement de la tête peut ainsi à lui seul causer une commotion cérébrale directe même s'il n'y a pas de lésions anatomiques objectivables immédiatement et ce de façon distincte d'éventuelles lésions médullaires par « coup de fouet » cervical plus décrites chez le nourrisson.

Dans la littérature, il est décrit des lésions du tronc cérébral associées ou non à des fractures secondairement à des impacts de haute vélocité notamment lors d'accident de la circulation[37,84], ces cas démontrent que des coups de poing ou des coups de pieds peuvent produire des lésions aussi sévères. Dans ces contextes de traumatisme cranio-encéphalique fermé avec des forces d'accélération ou décélération, il est décrit des lésions dans les régions centro-axiales de l'encéphale [92], incluant au niveau du tronc cérébral des lésions au niveau des colliculi supérieurs, le toit du 4ème ventricule, la formation réticulaire médiane (dont les noyaux dorso-bulbaires), les pédoncules moyen et inférieur cérébraux, et le pont.

L'hypothèse d'un mécanisme reflexe lors d'une extension intense du tronc cérébral, est probable en raison du passage des grandes voies sensitivomotrices, de la formation réticulaire (comprenant le complexe de pré-Bötzinger), et de la présence de noyaux centraux dans cette zone pivot [93,94]. Les centres vasomoteurs et le générateur central de la respiration sont situés dans le tronc cérébral. Ce sont ces centres qui sont respectivement responsables de la régulation à court terme de la pression artérielle et de l'innervation des muscles de la cage thoracique et des voies aériennes supérieures. Une atteinte de ces centres peut conduire à un décès par une action bradycardisante importante ou par arrêt respiratoire. Nos sujets étaient jeunes, sains et en bonne santé. Seules des lésions axonales ont été retrouvées au niveau du mésencéphale ou pédonculaire sans aucunes autres lésions associées. Le diagnostic de décès secondaires à un impact facial par lésions du tronc cérébral n'a été controversé que dans un cas où des anomalies architecturales cardiaques étaient décrites à l'examen histopathologique sans symptomatologie

connue du vivant. Toute la difficulté pour le médecin légiste réside dans l'argumentation des diagnostics possibles selon leur probabilité lors d'un décès brutal sans cause établie

[2] mais devant des circonstances traumatiques évidentes et de sa connaissance des mécanismes physiopathologiques engendrés par les traumatismes faciaux. Nos cas ont suffisamment survécu pour observer ces lésions axonales puisque un délai est nécessaire, amenant un élément de preuve lésionnelle neuronale. Cependant, l'hypothèse peut être plus difficile à soutenir lors de décès instantanés ou presque, suite un traumatisme facial sans lésions macro et microscopiquement identifiables ; compte tenu des limites techniques [71–74]. La validation de nos travaux par des publications référencées nous parait un moyen de soutenir ce mode de décès dans ce contexte.

Ces hypothèses de mécanismes lésionnels (littérature et cas cliniques présentés) mettant en jeu des contraintes dans la zone des pédoncules cérébraux, sont en accord avec nos résultats de simulations numériques par éléments finis. En effet, lors d'un uppercut, nous observons des contraintes de l'ordre de 16 KPa au niveau des pédoncules cérébelleux. (Figure 30). Ce niveau de contrainte observé, très proche de la valeur seuil de 18KPa traduisant des lésions cérébrales [95], laisse présager de probables lésions axonales.

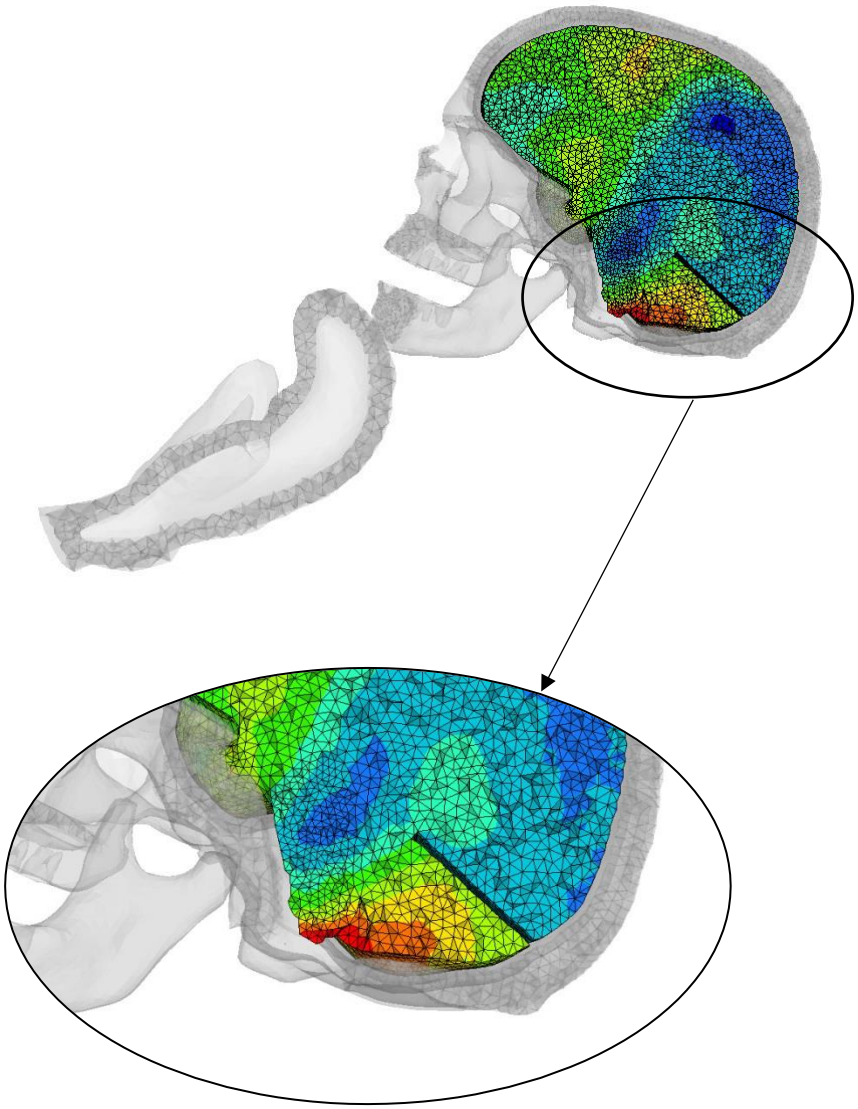

Figure 30 : Mise en évidence par notre modèle en éléments finis de contraintes de Von Mises au niveau des pédoncules cérébelleux.

Toutefois, notre modèle numérique permet de localiser uniquement des concentrations de contraintes dans cette zone mais ne peut reproduire les lésions axonales et les interconnections cérébrales des différentes voies sensitivomotrices extrêmement complexes. Une dissociation substance grise /substance blanche, ainsi qu'une modélisation fluide-structure du LCR et des vaisseaux cérébraux devront être ajoutés pour améliorer ce modèle.

Vers une modélisation des axones

L'allongement en traction des axones menant à des lésions axonales diffuses est un mécanisme lésionnel que les modèles numériques pourraient prédire. Il a été démontré que la contrainte de cisaillement est le principal facteur de lésions axonales diffuses. La réponse mécanique du tissu cérébral dépend fortement de sa neuroarchitecture sous-jacente. Le tissu de la substance blanche est très anisotrope en raison de la présence d'axones myélinisés. Incorporer cette anisotropie dans MEF de tête est un domaine qui a connu un développement considérable ces dernières années dans le but de prédire plus précisément le risque de lésions axonales diffuses.

Une méthode connue consiste à cartographier les valeurs d'anisotropie fractionnelle déterminées par les ensembles de données sur les éléments du maillage lors de l'étape de post-traitement. Ainsi afin d'étudier les lésions axonales, une modélisation des réseaux axonaux sera établie par IRM de diffusion et transposée sur le modèle par éléments finis déjà existant.

Perspectives

La compréhension de l'influence du mode d'impact et des caractéristiques de la victime sur l'apparition des lésions cérébrales est majeure. Mais le caractère unique de l'impact n'est pas la seule situation rencontrée, il est donc envisagé à la lumière de ces données, d'étudier l'action sur le tissu cérébral d'une multiplicité de chocs de moindre énergie, d'ajouter une modélisation par éléments finis des réseaux axonaux pour une meilleure évaluation des lésions, mais aussi de modifier l'environnement en évaluant la contrainte produite lorsque l'impact facial se fait alors que l'individu est allongé au sol ou bloqué contre un plan dur.

Aussi, les paramètres biologiques variant selon chaque individu, la modélisation numérique permet de les moduler à l'infini (forme de mandibule, dentition…) pour une approche réaliste d'applications médico-légales.

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Annexes

Annexe 1

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Forces transmission to the skull in case of mandibular impact

Lucile Tuchtan [a,b,*], Marie-Dominique Piercecchi-Marti [a,b], Christophe Bartoli [a,b], Dominic Boisclair [c], Pascal Adalian [b], Georges Léonetti [a,b], Michel Behr [c], Lionel Thollon [c]

[a] APHM, CHU Timone, Service de Médecine Légale et Droit de la Santé, 13385 Cedex 5 Marseille, France
[b] Aix-Marseille Université, CNRS, EFS, ADES UMR 7268, 13916 Marseille, France
[c] Aix-Marseille Université, IFSTTAR, LBA UMR_T 24, 13916 Marseille, France

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ABSTRACT

Background: Forensic investigations have been reported regarding the loss of consciousness and cardiac arrests resulting from direct mandible impact. However, the mechanisms by which the forces are transferred to the skull through direct mandible impact remain unclear. We conducted a study regarding direct mandible impact on the level of energy required to create a mandible fracture and on the energy dispersion phenomenon to the skull and to the brain.
Materials and methods: This study combines an experimental and numerical approach. Mandible strike was studied using experimental trials performed on post-mortem human subjects. A finite element model of the head and face of a male was also developed based on tomodensitometry scans. The model was validated with literature data and experimental trials. A parametric study was then performed to study the effect of diverse variables such as the dentition integrity, cortical bone thickness, etc.
Results: The forces measured on our reference model were 3000 N on the chin, 1800 N at the condyles, and 970 N in the occiput. Of all the results, we observed a decrease of approximately one-third of the efforts from the chin to the base of the skull and a lower half of the still forces at the occiput, except in the edentulous and for the lateral and frontal impact where the force is transmitted directly to the skull base area.
Conclusion: This study allowed us to create a 3D model of the mandible and face bones to better understand the force transfer mechanisms into and from the mandible. The parameters of the model may be modified to suit the individual characteristics for forensic investigations and legal matters.

1. Introduction

Establishing the relationship between the death of an individual and a violent event is a common practice in forensic science. Many cases of death by violence are relatively obvious and are the result of major blood loss or the compression of organs caused by haemorrhage. However, the process of identifying the causes of the death is sometimes solely based on the time-event continuum between the violent act and the death without confirmation of the injury mechanisms. This situation may be the case when anatomical signs of the injury mechanisms are absent, such as death by reflex reactions related to nervous conduction (death by carotid compression, thoracic impact, compression of the ocular globes, etc.) [1] or injuries to the central nervous system (axonal damage). The hypothesis that forces are internally transferred to the brain, and more specifically the brainstem, is sometimes promoted for the latter case. At the Marseille forensic service, two deaths related to uppercut impacts were recorded, with the brain histological examinations only indicating lesions of the midbrain. Genarelli et al. [2] studied the conditions for the emergence of diffuse axonal lesions via tests using apes. They concluded the functional consequences resulting in loss of consciousness are directly related to destruction of axons at impact. Others (e.g., Besenski et al. [3], Povlishock et al. [4], Sahuquillo et al. [5], and Buki et al. [6]) demonstrated that the axon functional role can be altered even if it is not cut. These stretched lesions are found in lower resistance axons areas: the brain's area transitions between grey matter and white matter, periventricular white matter, corpus callosum and, with a higher severity degree, the dorsal mesencephalon [7]. Belingardi et al. [8] demonstrated that the

* Corresponding author at: APHM, CHU Timone, Service de Médecine Légale et Droit de la Santé, 13385 Cedex 5 Marseille, France. Tel.: +33 4 91 48 22 45; fax: +33 4 91 92 33 31.
E-mail address: lucile.tuchtan@ap-hm.fr (L. Tuchtan).

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brainstem pivots upon facial impact and suffers alterations by the subsequent shearing mechanisms. Without fracture, the skull movement at impact may still have caused a direct brain contusion. Different degrees of contusion are possible, ranging from simple transient local vasomotor paralysis to various extensive parenchymal tears, which inevitably involve vascular elements. We distinguish between a "blow" versus lesions of "backlash" that the inertia causes away from the impact point. These inertial effects can be observed when the head is violently set in motion without direct impact, e.g., the driver undergoing a "whiplash" when the vehicle is buffered from behind, the rugby player or football player pushed in the back, the boxer receiving an uppercut, or even with impacts suffered by a helmeted head [9]. Our study is primarily concerned with the understanding of energy transmission upon impact of the mandibular area to the rear of the face, through the condyles. We attempt to demonstrate how during a single impact, such as an uppercut, residual energy (some is absorbed by the face with broken bones) arrives at the brainstem.

Brain injuries sustained by boxers and American football players following a mandible impact have been reported recently [10,11].

Finite element models of the head have been proposed recently [12–15] to study this issue through parametrical analysis.

As a result, we decided to investigate the effects of mandible impact resulting from fighting.

To understand this injury mechanism, we conducted an experimental and numerical study with a validated head-face finite element model (FEM). We first demonstrate that the forces are transmitted from the mandible to the skull and, in particular, to the condyles. We then illustrate the energy transfer phenomenon in which part of the residual energy spreads to the brain and brainstem. We assess how some parameters may affect this transmission, namely the impact velocity, position and mass as well as the mandible physiology and biomechanical properties (Young's modulus, cortical bone thickness, bone density, etc.).

2. Materials and methods

2.1. Experimental investigation

Three experimental trials were conducted on post-mortem human subjects (PMHS) to measure the forces involved and to validate the FEM. Two men and one woman aged above 75 years old sat in a tilting seat with a pre-set angle defined by the subjects' height. Two out of the three subjects were toothless. The subjects' anthropometric data are reported in Table 1. An uppercut strike was simulated by impacting the subjects on the mandibular symphysis with a 5-kg mass and impact velocity of 5 m/s. The impactor mass, a metallic cylinder of 76.5 mm in diameter and 458 mm in length, was mounted on a swing and dropped at a predefined height. A load cell placed on the impactor measured the forces transferred to the mandible, and an accelerometer placed on the forehead of each of the subjects measured its acceleration upon impact. The trials were filmed using a high speed camera (1000 fps) to record the impact kinematics.

The lower end of the face of each of the subjects was dissected after the impact to identify any potential fractures.

Table 1
Subject's anthropometric data.

	Age (y.o.)	Gender	Height (cm)	Weight (kg)	Dentition
Subject no. 1	75	Female	154	43	Complete
Subject no. 2	≥75	Male	173	94	Incomplete
Subject no. 3	≥75	Male	171	79	Incomplete

2.2. Numerical investigation

A previously validated 3D FEM of a human head created based on tomodensitometry scans was used in this study [12]. The model was first improved by adding the face and validated with the available literature data and our experimental trials. The effects of variations in the impact and biological parameters were then assessed.

2.2.1. FEM description

The skull geometry was created using a tomodensitometry scan with a slice thickness of 1 mm and then numerically reconstructed using MICMICS 12.3 software (Materialise, Louvain, Belgium). The main anatomical components (scalp, subarachanoid space and brain) were added in the meshing process. The meshing was produced using Hypermesh software, and the model properties were managed using Radioss (Altair Engineering, Inc., Detroit, MI, USA). The average element size was fixed at 2 mm. The skull was composed of three layers to recreate the trabecular and cortical bones. The cortical bone mesh used shell elements with three nodes, and the trabecular bone used tetrahedral elements. The brain and subarachanoid space acting as the cerebrospinal fluid situated between the brain and the skull were modelled using tetrahedral elements. The scalp was modelled using two layers of brick elements.

The facial bones were separately modelled using a parametric approach to define the size and cortical bone thickness. The sinus, teeth, mandible and articulation discs were added and sized based on literature data [16–22]. The model is presented in Fig. 1.

The FEM is composed of 700 000 volumetric elements (tetrahedral and brick) and 80 000 shell elements (3 and 4 nodes). The total mass of the model is 4.2 kg. All of the mechanical properties (Table 2) were taken from literature data [16–20,23,24].

2.2.2. FEM validation

The model was validated according to the following:

- Experimental trials conducted by Schneider et al. [25]. The resistance of the facial bones and mandible was assessed via 106 trials on 17 PMHS skulls. Two different positions were used to impact the mandibles: antero-posterior and lateral. Two Plexiglas cross members were affixed to the drop assembly and contained nylon bushings to minimise any frictional reaction with the steel guidewires. In addition, the heads were severed at the seventh cervical vertebra in the latter experiments to facilitate the placement of the skull necessary for antero–posterior mandibular impacts. The head, in all cases, was supported by wedges of soft polyurethane padding (the test conditions and limits are presented in Table 3).
- Experimental trials conducted by Viano et al. [10] where the effect of a boxer's punch was studied: eleven Olympic boxers weighing 51 kg (112 lb)–130 kg (285 lb) were included in the study. These boxers were instructed to strike the instrumented Hybrid III head with their gloved fist two times with four different punches: a straight punch to the forehead, a straight punch to the jaw, a hook, and an uppercut. Accelerometers were placed in the boxer's clenched hand (two Endevco 7264-2k accelerometers). The Hybrid III was equipped with the standard triaxial accelerometers (Endevco 7264-2k) (see Table 3).
- Experimental trials described previously in this study.

3. Results

3.1. Experimental investigation

The first PMHS trial (subject no. 1) resulted in a 2-cm-long laceration to the chin, linear fracture of the mandible symphysis,

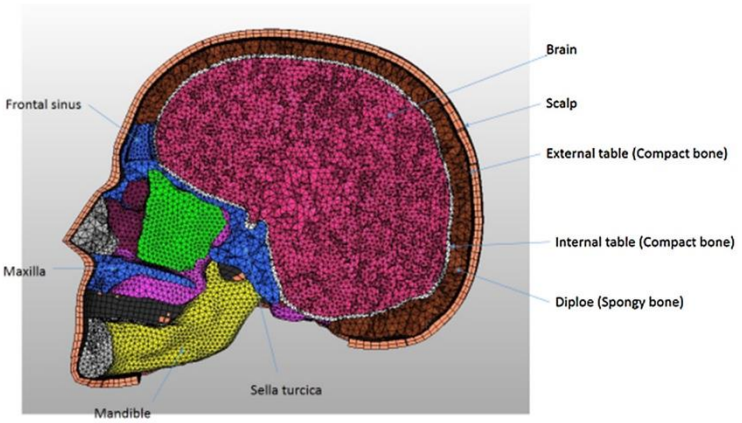

Fig. 1. 3D head model (sagittal section).

Table 2
FEM anatomical components properties.

Anatomical component	Property	Thickness (mm)	Density ρ (kg/m^3)	Young's modulus E (MPa)	Poisson coefficient ν	σ_c: ultimate stress failure in compression	σ_t: ultimate stress failure in tension	σ_{max}
Trabecular bone	Elastoplatic		1500	4600	0.05	35 MPa	35 MPa	
Cortical bone	Elastoplatic	1.5	1900	15 000	0.21	145 MPa	90 MPa	
Mandible	Elastoplatic	1.5	2500	13 000	0.3			
Teeth: molare enamel surface		0.5	1800	84 100	0.23			220
Teeth's dentine/alveolar surface				18 600	0.31			
Scalp	Elastic	5	1000	16.7	0.42			
LCR	Elastic		1040	0.12	0.49			
Face	Elastic	1–3	2500	5000	0.23			55
Brain	Viscoelastic		1040	0.049				
Mandible articular disc			1050	44.1	0.4			

Table 3
Validation of the finite element model compared to the results of experimental trials of Schneider and Viano.

Authors	Impact site	Impactor mass (kg)	Impact velocity (m/s)	Forces (N)	FEM Forces (N)
Schneider et al.	Anteroposterior	3.12	4.87–5.44	1840–4000	3700
	Lateral	3.81	5.44–5.97	820–3400	2900
Experimental trials	Uppercut	5	5	2400–4500	2500
Viano et al.	Anteroposterior (jaw)	1.67	6.7	1010–4090	2200
	Uppercut	1.67	6.7	458–3330	3000
	Lateral (hook)	1.67	6.7	994–9950	2300

and bilateral mandible neck linear fractures (Fig. 2). No fracture at the temporal regions was found. The second trial (subject no. 2) resulted in a laceration to the chin, a complete oblique linear fracture of the right side mandible and bilateral mandible neck linear fractures. The last trial (subject no. 3) resulted in a laceration to the chin and bilateral mandible neck oblique linear fractures. The average acceleration of the three tests was measured as 22 G. All of the forces measured are presented in Table 4.

3.2. Numerical investigation

3.2.1. Model validation

The validation results are presented in Table 2. Our results are comparable to the above studies; therefore, the FEM is a suitable model.

3.2.2. Variability study

A parametric study using varying impact conditions and varying biomechanical properties of the mandible was performed to assess how transmission of the forces to the skull is affected. The following parameters were used as a comparison baseline: cortical bone thickness (1.5 mm), Young's modulus (13.7 GPa), impact velocity (6.7 m/s), impactor mass (1.5 kg) and density (2500 kg/m^3).

The variations of impact conditions were:

- Impacted zone: uppercut, chin, lateral.
- Velocity: 4, 5, 6.7 and 8 m/s.
- Impactor mass (simulating hand and glove): 1.2, 1.7 and 2.2 kg.

The reference impactor mass of 1.7 kg is in accordance with the previous work of Viano et al. [10], who used a mass of 1.67 kg.

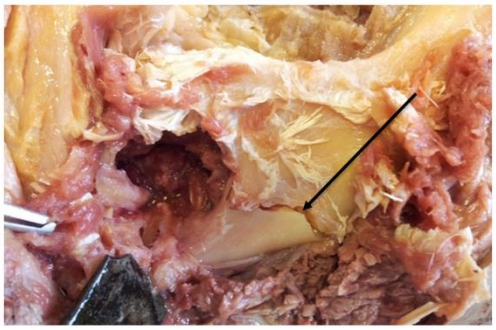

Fig. 2. Mandible condylar neck fracture.

Table 4
Forces in Newton (N) from experimental trials tab.

Trials	Forces (N)
Subject no. 1	2608.2749
Subject no. 2	2462.544
Subject no. 3	4508.1568

The biomechanical property variations of the mandible were:

- Cortical bone thickness: 1.5, 3 and 5 mm.
- Dentition: toothless, complete upper dentition, complete lower dentition, or full dentition.
- Young's modulus: 10 or 13.7 GPa.
- Bone density: 1500 or 2500 kg/m^3.

The impact forces at the mandible and the forces transferred to various face regions up to the skull were analysed for each simulation. Deceleration at the centre of the head was assessed, and visual fracture incidence was documented.

Fifteen simulations were conducted. All of the simulations were performed using Radioss software (Altair Engineering, Inc., Detroit, MI, USA). The forces were measured at three levels: the chin, a section through the mandibular condyles and the skull base, and one measurement at the occiput. The results are presented in Table 5. The force curves were filtered at 600 Hz, and the acceleration curves were filtered at 1000 Hz.

3.2.2.1. Effect of the impact site location. The forces measured at the impact site for the chin and lateral strikes and at the condyle level are similar: the forces are 2200 N and 2300 N, respectively, at the impact site and 2200 N at the condyle level. For the uppercut strike, the force at the impact site for the chin and lateral strikes was 3000 N and 1800 N, respectively, at the condyles.

3.2.2.2. Effect of biomechanical variation. The following parameters are associated with the victim:

- Cortical bone thickness: the forces measured at the chin were 3000 N for a cortical bone thickness of 1.5 mm and 2350 N for thicknesses of 3 mm and 5 mm. The forces measured at the condyles are 1850 N, regardless of the cortical bone thickness. The forces are somewhat damped with greater cortical bone thickness.
- Dentition: the forces at the condyles and at the skull base remained high (between 2800 N and 3600 N) for incomplete dentition and toothless models. The force at the condyles decreased to 1800 N for the complete dentition model.
- Young's modulus: the force at the impact site was modestly lowered with the modulus of 10 GPa compared to the reference modulus (13.5 GPa). The forces at the condyles and the occiput are similar, regardless of the modulus used, with the exception of the maximum load of 2200 N for the 10 GPa modulus.
- Bone density: the force measured at the impact site was 2250 N for the 1500 kg/m^3 density. The force increased to 3000 N with the reference bone density of 2500 kg/m^3.

Table 5
Forces measured for all simulations in Newton (N).

Anatomical site	Chin	Condyles/ skull base	Occiput
Impact velocity (m/s)			
4	2700	2120	680
5	2400	1500	650
6.7	3000	1800	970
8	2900	1750	780 second peak at 1700
Cortical bone thickness (mm)			
1.5	3000	1800	970
3	2400	1900	740
5	2300	1880	550 second peak at 1900
Impactor mass (kg)			
1.2	2300	1680	800
1.7	3000	1800	970
2.2	3200	1400	880 second peak at 3400
Impact site			
Lateral	2300	2200	600
Frontal	2200	2200	850
Uppercut	3000	1800	970
Dentition			
Lower toothless	3500	3500	800
Upper toothless	2800	3200	800
Complete toothless	3500	3600	680
Complete	3000	1800	970
Young's modulus (MPa)			
10 000	2500	1900	800 second peak at 2200
13 700	3000	1800	970
Density (kg/m^3)			
1500	2250	1600	800
2500	3000	1800	970

3.2.2.3. Impact conditions. The following parameters are associated with the striker:

- Impact velocity: the forces found at the impact zone are similar between the impact velocity and decrease progressively from the condyles to the occiput. The peak force of 780 N followed by a second peak of 1700 N was found for an impact velocity of 8 m/s.
- Impactor mass: an increase in the impactor mass increased the forces at the chin from 2300 N to 3200 N.

In our study, we aimed to assess the force dispersion not only at the mandible but also at the brain level. To do so, a visual inspection of the Von Mises stress distribution on our FEM was performed. Fig. 3 shows the stress distribution for various time steps following the impact. Significant forces are found at the base

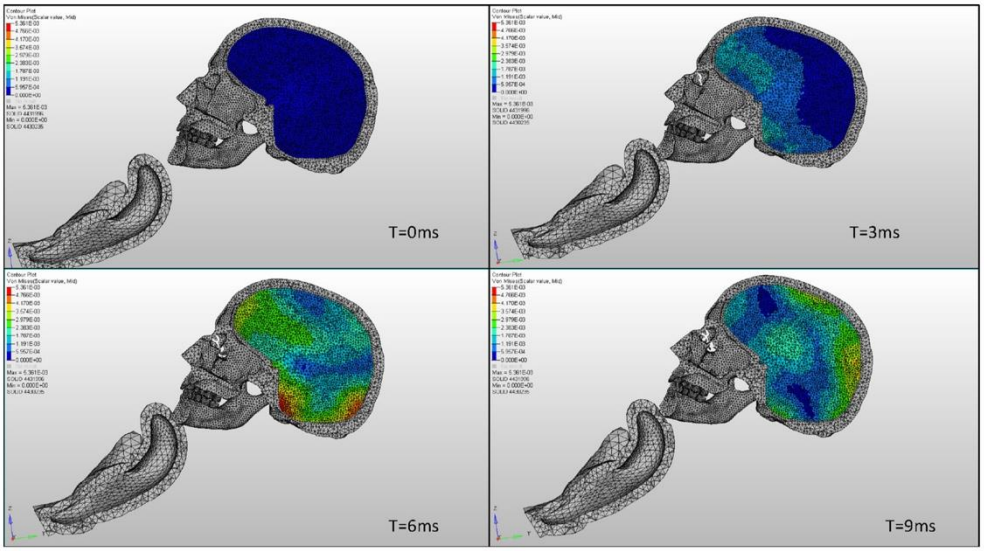

Fig. 3. Uppercut simulation. Von Mises stress distribution at 0, 3, 6 and 9 ms (*T*, time).

of the skull and specifically at the midbrain level, which is similar to the results of Viano et al. [10].

All subjects sustained a fracture at the condyles and the mandible symphysis.

4. Discussion

This study confirmed that a mandible impact causes cranial bone damage and may generate brain injuries. The forces measured in this study are similar to data reported on anthropometric surrogates and PMHS, which validates our model.

The FEM was validated with experimental trials. The FEM's parameters may be modified to suit the biomechanical variability of the victim or the striker, which is a significant benefit for forensic investigations.

Previous FEM studies investigated the impacts to the face (Bruyère et al., Yoganandan et al., Allsop et al., Schneider et al., etc.) [25–34]. Frontal or maxillary impacts resulting from road crashes (Yoganandan et al. [27,29], Hampson [28]) with contact to the driving wheel (Hodgson et al. [31]) were the focus of great attention in previous studies. Other previous studies on uppercut strikes were concerned with the mandible resistance to impact [21,35], mechanical properties [20], or chewing [22].

Evidently, from our understanding, the forces are transmitted along the anteroposterior direction (frontal-temporal-occiput). The forces at the chin, skull base and occiput are significantly decreased in the case of our model featuring a complete dentition.

4.1. Impact site effects

Our results indicate that frontal and lateral strikes transfer forces directly to the skull base and occiput, although they are slightly less for the latter. Our results at the impact site are similar to the results of Schneider et al. [25], albeit in this study, the mandible was impacted with a mass of 3.12 kg and a velocity ranging between 4.8 and 5.4 m/s. Along the anteroposterior direction, the reported forces at the impact site ranged between 1890 N and 4000 N depending on the individual. The forces at the impact site ranged between 820 N and 3400 N for an impact mass of 3.8 kg and a velocity ranging between 5.4 m/s and 5.9 m/s for lateral mandible impacts. According to Viano et al. [10], the impact force ranged between 1010 N and 4090 N for a strike at the jaw and between 994 N and 9950 N for a hook punch. Our results are within these admittedly quite large force ranges, which highlights the great variability of the impact conditions.

4.2. Biomechanical variations

4.2.1. Dentition

The forces were somewhat damped by the addition of teeth in our model. The partial dentition and toothless models yielded similar results. The forces measured on our FEM are similar to the results of our experimental trials. The toothless cases had reduced alveolar bone and therefore reduced spacing between the impactor and skull base. Walilko et al. [11] demonstrated that protective mouthpieces may reduce by half the forces measured at the mandibular fossa, with the added material increasing the amount of force damping. In our study, we chose to assess the difference between two extreme cases (complete dentition and toothless); therefore, our results must be adjusted to the condition of the remaining teeth (altered or not, number, prosthetic materials, osteoporosis, etc.). The chosen parameters were linked to the presence or lack of teeth and not to the subject's age, which may, along with individual variability, affect the force transfer mechanisms.

4.2.2. Cortical bone thickness

Similarly, increasing the cortical bone thickness decreases the force damping effects, with the forces being directly transferred to the skull base. For a cortical thickness of 5 mm, a peak appears, which is most likely due to the increased bone stiffness and decreased damping due to early bone fracture.

4.2.3. Young's modulus and bone density

Reducing the Young's modulus and the bone density decreases the force measured at the impact site (chin) and thus favours the occurrence of fracture by energy absorption.

4.3. Impact conditions

4.3.1. Impact velocity

Only small variations in forces at the chin and at the condyles were measured with increased velocity in our study. However, the forces were significantly higher at the skull base with increased velocity, due in part to the rebound effect. We chose the velocity of 6.7 m/s as the reference velocity based on the published study of Viano et al. [10] who estimated the velocity of an uppercut strike to be close to this value.

The forces measured in our study are similar to previously published data, with the peak forces varying between 458 N and 3330 N. However, the reported data were obtained on an anthropometric Hybrid III dummy with the head, neck, and torso components. We chose to study the force transfer mechanism with the skull and did not take into account the potential effects of the cervical spine and the rearward head motion. The forces found for strikes on the jaw and hooks are similar to the values found with our model. Our measurements are within the large range of published values, highlighting the difficulties of accurately assessing such values due to the large number of individual specific parameters. The individuals tested were 1.65–2 m tall and weighted between 50 and 130 kg. The values found can therefore be associated with a scenario where an average person hits another person with the intent of harming the person.

4.3.2. Impactor mass

Decreasing the impactor mass from 1.7 kg to 1.2 kg had little effect outside of decreasing the forces at the chin from 3000 N to 2300 N. Increasing the mass to 2.2 kg resulted in the same energy transfer but with the added backlash effect, most likely due to secondary motion of the head. The impactor masses used were representative of the morphology of a regular person.

4.4. Injury review

All of the experimental trials and simulations resulted in a mandible fracture located at the condyles and symphysis. The lowest force measured at the impact site was 2200 N. The fracture threshold value is considered to be between 1000 N and 1800 N, according to Allsop et al. [30]. The fracture threshold value is between 2500 N and 3100 N for face impact and between 600 N and 800 N for lateral impact according to Unnewehr et al. [35]. Thus, all of the results are considered to be in agreement with the results of previous studies.

In our study, we validated the skull model to demonstrate the energy transmission to the skull, but our focus was a Von Mises picture of the cerebral pressure areas. The Von Mises stress was distributed with an anterior to posterior orientation through the temporal lobe and ended at the brainstem level. Viano et al. [10] found that a hook strike increases the stress in the temporal region and at the midbrain level. Belingardi et al. [8] developed an FEM of the head based on the experimental trials performed by Nahum et al. [36] and found increased cerebral pressure with significant stress in the coronal section of the brainstem, which confirms its pivotal role in movement of the brain. Thus, according to our study, in a simple uppercut, the residual impact energy (some is absorbed by the face with broken bones) arrives at the brainstem. However, according to Ommaya et al., the brainstem and mesencephalon are the last to be affected, both functionally and structurally, after human head injury because they are anatomically the structures most protected from injuring strains [5,37]. Consequently, in every case where structural damage is found in the brainstem, structural damage will also be found in the brain hemispheres [5,37–39]. The most important consequence of Ommaya's theories was the following newly introduced and controversial concept: when primary damage is found in the rostral brainstem, it is never isolated but is associated with diffuse brain damage to the hemispheres [5,37–39]. This concept challenged the long-standing classical view that the main mechanism producing traumatic unconsciousness was an isolated "primary brainstem injury" [5,40–43].

Head acceleration in our models was measured as 22 G. Viano et al. [10] found similar values of head acceleration, with an average acceleration of 24 G for an uppercut strike.

Thus, we can suppose that during the uppercut, part of the impact energy reaches the brainstem (as shown in images of the brain with the Von Mises stress, red in the image), which is obviously supported by the levels of stress found at the condyles during the simulations and the fractures observed at the temporomandibular joint in both experiments. This result is very interesting from a forensic point of view because the pressures observed in the brainstem could lead to a vagus nucleus stimulation, which may be involved in cardiac arrest via the cardio-inhibitory reflex.

5. Conclusion

A validated FEM of heads allowed us to identify the potential injuries induced by a mandibular impact. Although the propagation of forces logically follows an anteroposterior direction (fronto-temporo-occipital), the effort intensity distribution varies with different parameters. The intensity of the forces does not decrease gradually depending on its propagation, but its variation depends on many parameters, primarily including the mandibular impact but also the force applied and the quality of the dentition site. We found that various parameters affect how the forces are transferred, with the impact site having the most effect, followed by the impact configuration and the dentition integrity. The Von Mises illustration of the pressures on the brain allowed us to visualise the more important pressure areas at the occiput and the brainstem during an uppercut mandibular impact.

FEM parameters may be adapted to the individual characteristics to suit a realistic situation, which is a major advantage of the use of the model for forensic investigations and legal expertise cases.

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Annexe 2

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Study of cerebrospinal injuries by force transmission secondary to mandibular impacts using a finite element model

--Manuscript Draft--

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Corresponding Author:	Iucile tuchtan, MD Assistance Publique Hopitaux de Marseille Marseille, FRANCE
First Author:	Iucile tuchtan, MD
Order of Authors:	Iucile tuchtan, MD
	Yves Godio-Raboutet
	Clémence Delteil, MD
	Georges Léonetti, MD, PhD
	Marie-Dominique Piercecchi-Marti, MD, PhD
	Lionel Thollon, PhD

Abstract:	Brain and cervical injuries are often described after major facial impacts but rarely after low-intensity mandibular impacts. Force transmission to the brain and spinal cord from a mandibular impact such as a punch was evaluated by the creation and validation of a complete finite element model of the head and neck. Anteroposterior uppercut impacts on the jaw were associated with considerable extension and strong stresses at the junction of the brainstem and spinal cord. Hook punch impacts transmitted forces directly to the brainstem and the spinal cord without extension of the spinal cord. Deaths after this type of blow with no observed histological lesions may be related to excessive stressing of the brainstem, through which pass the sensory-motor pathways and the vagus nerve and which is the regulatory center of the major vegetative functions. Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (jaw shape, dentition…) for a realistic approach to forensic applications.

Marseille, June 6, 2019

Prof. C. Jackowski
Co Editor-in-Chief
Forensic Science International

Dear Professor Jackowski,

On behalf of my co-authors, I wish to submit our manuscript, “Study of cerebrospinal injuries by force transmission secondary to mandibular impacts using a finite element model”, for consideration for publication in Forensic Science International as an original research paper.

Impacts to the mandible have received little attention in the literature, in particular low intensity impacts. A complete finite element model of the head and neck has also rarely been created and validated, as most studies have focused on the skull alone. We were interested in brainstem and spinal cord injury secondary to this type of impact because in our forensic institute we had cases of deaths following blows to the mandible which had been seen and described by several objective witnesses, whereas the lesions found were relatively minor (axonal lesions). We therefore thought it would serve a useful purpose to further examine the injury mechanism of mandibular impacts.

With thanks in advance for your consideration,

Yours sincerely,

Dr Lucile Tuchtan
Forensic Department
Assistance Publique des Hopitaux de Marseille
CHU Timone
264 rue Saint Pierre
13385 Marseille Cedex 5
France
lucile.tuchtan@ap-hm.fr
Tel: +33491482245
Fax: +33491923331

Study of cerebrospinal injuries by force transmission secondary to mandibular impacts using a finite element model

Lucile Tuchtan [a,b,*], Yves Godio-Raboutet [c,d], Clémence Delteil [a,b], Georges Léonetti [a,b], Marie-Dominique Piercecchi Marti [a,b], Lionel Thollon [c,d]

a *Forensic department, APHM, Hôpital de la Timone, 13385 Marseille, France*

b *Aix Marseille Univ, CNRS, EFS, ADES, Marseille, France*

c *Aix Marseille Univ, IFSTTAR, LBA, Marseille, France*

d *iLab-Spine (International Laboratory – Spine Imaging and Biomechanics)*

* Corresponding author. Tel.: +33 491482245; fax: +33 491923331

E-mail address: lucile.tuchtan@ap-hm.fr (Lucile Tuchtan)

Highlights

Force transmission to the brain was evaluated by a complete finite element model.

Anteroposterior uppercut impacts were associated with extension of the brainstem.

Hook punch impacts transmitted forces directly to the brainstem and the spinal cord.

Death without histological lesion may be related to excessive stressing of the brainstem.

Abstract

Brain and cervical injuries are often described after major facial impacts but rarely after low-intensity mandibular impacts. Force transmission to the brain and spinal cord from a mandibular impact such as a punch was evaluated by the creation and validation of a complete finite element model of the head and neck. Anteroposterior uppercut impacts on the jaw were associated with considerable extension and strong stresses at the junction of the brainstem and spinal cord. Hook punch impacts transmitted forces directly to the brainstem and the spinal cord without extension of the spinal cord. Deaths after this type of blow with no observed histological lesions may be related to excessive stressing of the brainstem, through which pass the sensory-motor pathways and the vagus nerve and which is the regulatory center of the major vegetative functions. Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (jaw shape, dentition…) for a realistic approach to forensic applications.

Keywords*:* Finite element model, Cerebrospinal injury, Force transmission, Brainstem, Mandibular impact

1. Introduction

High kinetic energy impacts to the skull are often associated with hemorrhagic brain and meningeal injuries whose mechanism has been examined in several studies and for various types of cranial impacts [1–5]. Boxers are subjected to craniofacial impacts, and even low-velocity impacts may be associated with major brain injuries because of the evident anatomical proximity between the face and the brain. Inertia effects may be observed when the head is violently shaken without a direct impact on the cranium (particularly in hyperextension) [3,6–8]. Movement of the head caused by a blow to the face can in itself cause direct concussion even if no anatomical injuries can immediately be observed, because the damage is axonal [9–12]. In literature, many authors demonstrated that the axon functional role can be altered even if it is not cut [11–14]. The brainstem pivots upon facial impact and suffers alterations by the subsequent shearing mechanisms [15]. Without fracture, the skull movement at impact may still have caused a direct brain contusion. Death may thus be secondary to force transmission to the brain, either by a so-called reflex mechanism that involves nerve conduction (by vagus nerve overstimulation) [16], or by central nervous system injury (axonal damage) [9,11,17].

When injuries cannot be identified by gross examination or histologically because of rapid death [18], the forensic and also sometimes clinical problem is how to relate facial trauma to injuries that led to an altered state of consciousness or even to sudden death. This issue may arise in criminal proceedings concerning voluntary acts of violence or involuntary events such as road accidents, and when no focal cerebral hemorrhage is identified although the victim died during the violence and no other cause of death has been identified. The digital approach is a means of attempting to understand mechanisms of injury arising from head/neck dynamics and also of simulating an event described by a third party. In this study, we firstly developed a finite element model of the head and neck in order to understand facial fracture mechanisms and to observe force transmission in the unit formed by the brain, brainstem and cervical spinal cord.

Secondly, we examined some examples of mandibular facial trauma in order to study the potential mechanisms of injury.

2. Materials and methods

2.1. Characteristics of the model

The finite element model that we developed is a combination of a head model developed by the Laboratory of Applied Biomechanics (LBA) and a neck model developed in collaboration with the École Polytechnique Montréal (EPM) (Fig.1).

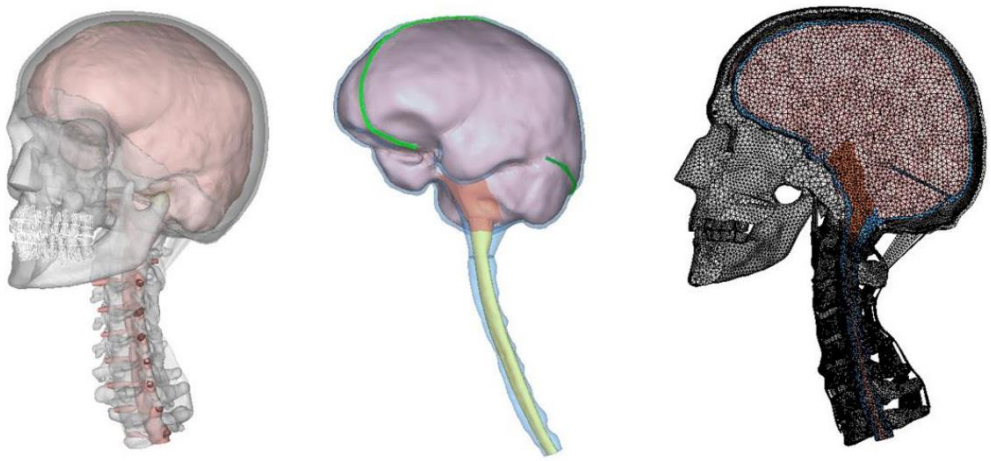

Fig 1: Finite element model of human head: a) whole model b) brain and spinal cord c) mid-saggital view.

Skull geometry was reconstructed from 1 mm Computerized Tomography (CT) scan slices of a 30-year-old man using MICMICS 12.3® software (Matérialise, Louvain, Belgium). The model was developed using Hypermesh® and Hypercrash® software (Altair Engineering, Inc., Detroit, MI, USA). The average size of the elements was 2 mm. The junction of the brainstem and the spinal cord was modeled in continuation of brainstem elements represented by tetrahedral elements. In the neck model developed by our laboratory (LBA, IFSTTAR in

collaboration with ILABSPINE) [18,19], only the elements of the vertebral bodies and the spinal cord were retained.

Because of the anatomical complexity of the brain, the meninges and the neck, each part (pia mater, dura mater, falx cerebri, tentorium cerebelli, hemispheres, cerebellum, brainstem, cervical spinal cord, cervical vertebrae and ligaments) was modeled according to its different mechanical properties. Continuous meshing was used.

The skull was reconstructed in three layers representing compact bone (external and internal tables) and cancellous bone (diploë) modeled using tetrahedral elements. The brain and subarachnoid space, comprised between the brain and the skull to simulate the cerebrospinal fluid, were also modeled using tetrahedral elements. The dura mater, falx cerebri and tentorium cerebelli were modeled with three-node shell elements. The cerebellum and brainstem were individualized. A boundary condition was applied on the C7 vertebrae (rotation and translation were fixed).

2.2. Validation of the model

The model underwent several evaluations: four different sources of validation (the studies of Nahum et al., Trosseille et al., and Viano et al. (Fig. 2) and our own experimental studies in the laboratory) based on the most relevant experiments in the literature and according to three different configurations (three mandibular impacts: uppercut (Fig. 3), hook (Fig. 4) and anteroposterior impact (Fig. 5)) in order to study the influence of point of impact at the level of the mandible.

1. Nahum et al.[2] (test 37): blow with a rigid cylindrical bar (mass 5.59 kg, impact velocity 9.94 m/s) on the frontal region of a seated post-mortem human subject (PMHS), with the torso supported. The blow was delivered in a sagittal plane and an

anteroposterior direction, with the subject's head inclined forward at 45° in the Frankfort plane (Fig. 2).

2. Trosseille et al. [20]: blow with a rigid iron bar (mass 23.4 kg, impact velocity 7 m/s) to the face of a seated PMHS in an anteroposterior direction (experiment MS 428-2) (Fig. 2). In order to evaluate the response of our model, we compared the force of impact at the level of the frontal region and acceleration of the head at the center of gravity using Nahum's tests. Pressures at the frontal and occipital regions were measured and evaluated according to the tests of Nahum and of Trosseille.
3. Viano et al. [21]: impacts on 3 mandibular areas with different impact velocities with a hand mass of 1.67 kg: jaw (9 2 m/s), hook (11 m/s) and uppercut (6.7 m/s) (Fig. 2).
4. Experiments carried out in our laboratory in 2015: blow with a rigid cylinder (mass 5 kg, impact velocity 5 m/s) on a PMHS in order to evaluate the force of the blow at the mandibular symphysis and to assess the type of fracture observed [22].

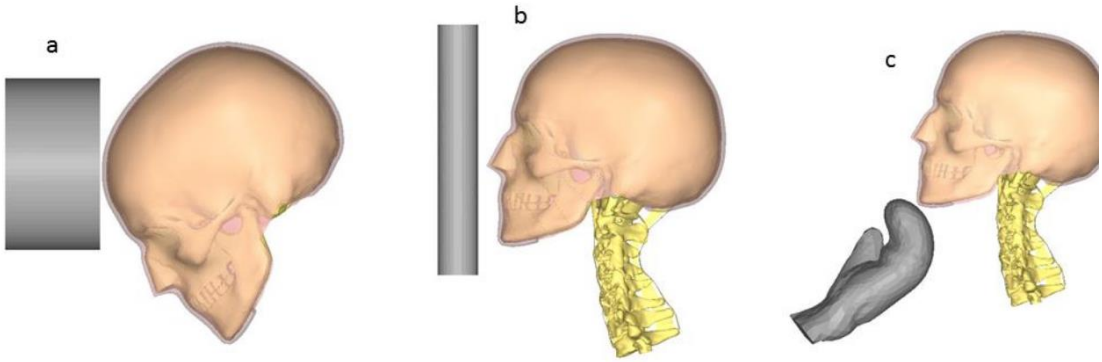

Fig 2 : Three numerical validation : Nahum et al (a), Trosseille et al (b), Viano et al (c).

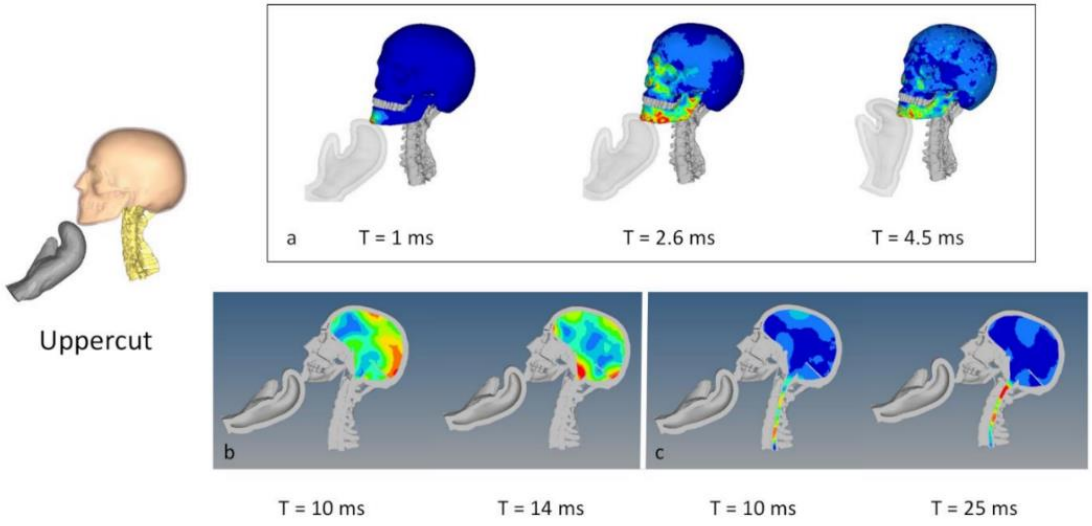

Fig 3: Stress propagation and distribution (Von Mises stress) in human head for uppercut scenario with focus on skull (a), brain (b) and cervical spinal cord (c).

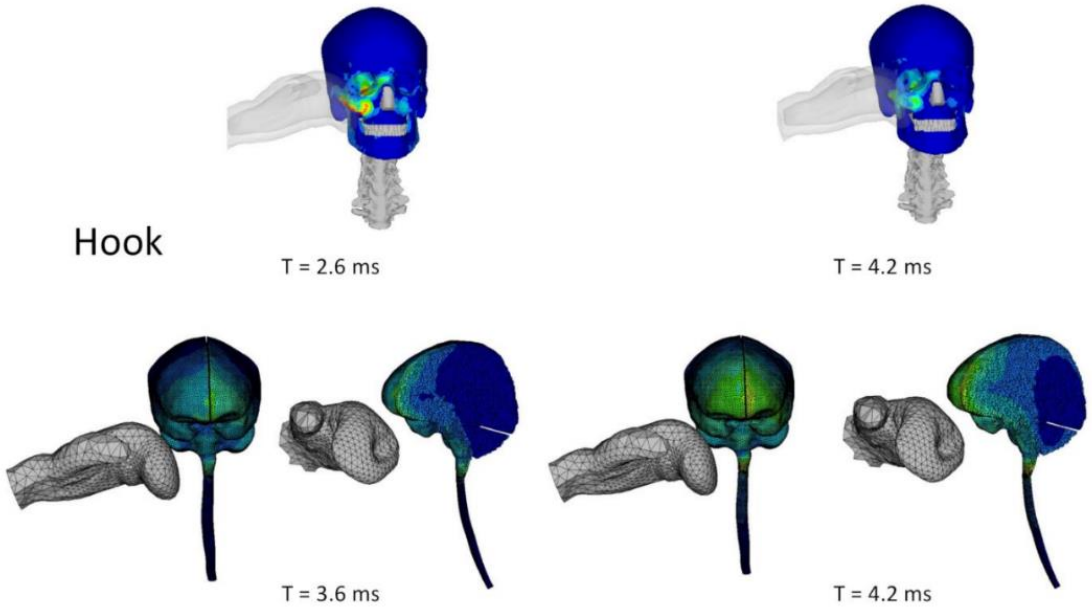

Fig 4: Stress propagation in human head for hook scenario with focus on skull and brain.

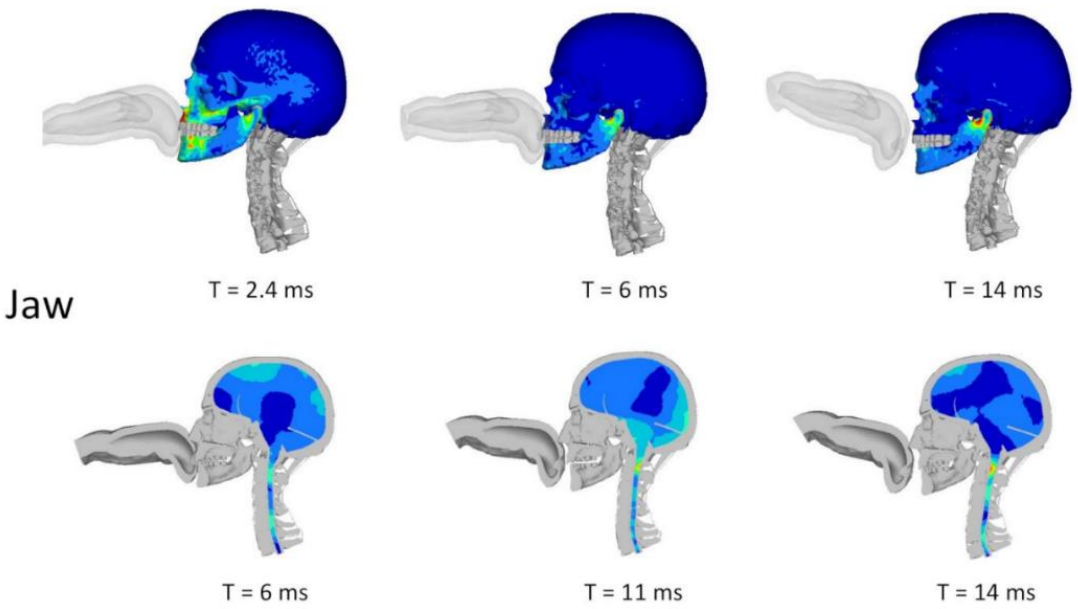

Fig 5: Stress propagation in human head for Jaw scenario with focus on skull and brain.

2.3. Resulting injuries

The injuries were examined at two levels: we observed firstly the distribution and propagation of stresses in the head, and secondly we evaluated the extension of the cervical spinal cord by measuring the space between the pons/medulla oblongata junction and C3.

The influence of impact location (uppercut, hook, face) on the development of stresses to the different parts of the brain (cerebrum, brainstem, cerebellum) and to the brainstem/spinal cord junction was also evaluated based on the tests of Viano et al. [18].

3. Results

The finite element model of the head consisted of 687,000 tetrahedral and hexahedral 3D elements and 85,000 shell elements (3 and 4 nodes). The mechanical parameters attributed to each anatomical part were based on the data of the literature (Table 1).

The finite element model of the neck consisted of 368,000 tetrahedral and hexahedral 3D elements and 122,000 shell elements (3 and 4 nodes). The head and neck together had a mass of 5580 grams.

We validated our digital model by reproducing the tests of Nahum, Trosseille and Viano. The comparative tests are summarized in the following table 2.

Anatomical components	Properties of the materials	Thickness (mm)	Density ρ (kg.m^{-3})	Young's modulus E (MPa)	Poisson coefficient v	σ c: compressive yield stress	σ t: tensile yield stress	σ max	Ref.
Cancellous bone	elastoplastic		1500	4600	0.05	35 MPa	35 MPa		[23]
Cortical bone	elastoplastic	1.5	1900	15000	0.21	145 MPa	90 MPa		[23]
Mandible	elastoplastic	1.5	2500	13000	0.3				[24]
Teeth: enamel surface crown		0.5	1800	41000	0.3			220	[24]
Dentin density/alveolar area				18600	0.31				[24]
Scalp	elastic	5	1000	16.7	0.42				[23]
CSF	elastic		1040	0.12	0.49				[23]
Face	elastic	1 to 3	2500	5000	0.23			55	[23]
Brain	viscoelastic		1200	1225					[23]
Disc of the mandibular joint			1050	44.1	0.4				[25]
Dura mater	elastic	0.5		5	0.45				[26]
Pia mater	elastic	0.1		2.3	0.45				[26]
Vertebral cortical bone	elastoplastic	0.37-0.9		3319	0.3				[19]

Table 1. Mechanical properties of the anatomical elements of the finite element model

Authors	Impact site	Impactor mass (kg)	Impact velocity (m/s)	Forces (N)		Pressure (MPa)		Acceleration (m/s^2)	
				Literature	Digital simulations	Literature	Digital simulations	Literature	Digital simulations
Nahum et al. (1977)	Anteroposterior	5.59	9.94	8000N	9800N	Frontal 0.12 MPa Posterior fossa 0.08 MPa	Frontal 0.14 MPa Posterior fossa 0.06 MPa	2034	2019
Trosseille et al. (1992)	Anteroposterior	23.4	7	X	X	Frontal 0.09 MPa Occipital 0.018 MPa	Frontal 0.12MPa Occipital 0.011 MPa	X	X
Viano et al. (2005)	Anteroposterior Jaw Uppercut Hook	1.67	9.2 6.7 11	2349 SD 962N 1546 SD 857N 4405 SD 2318N	4300N 2300N 5080N	X	X	X	X
Experimental test (LBA)	Uppercut	5	5	3150 ± 1141 N	2600N	X	X	X	X

Table 2: Conditions of evaluation of the model in relation to the literature

The results obtained (Table 2) were in agreement with the data of the literature concerning impact forces, head acceleration (Nahum's tests) and the different pressures measured in brain tissue.

3.1. Variation of impact location based on Viano's tests

- Uppercut (Fig. 3)

Forces were propagated in an anterior to posterior direction, were greatest at the junction of the brainstem and the spinal cord, and were associated with strong stresses at the occiput with a backlash effect that was visualized by pressures at the frontal level. Stresses were distributed at the level of the mandible extending to the condyles and then to the base of the skull, and were also distributed to the cheekbone. Forces spread to the frontal bone, ethmoid bone and nasal bone. The force of the impact caused considerable cervical extension that led to strong stresses at the level of the cervical spinal cord and the brainstem. Major stresses were visualized at the origin of the mandibular symphysis without condylar fracture. Extension of the cervical spinal cord was 2.6 mm.

- Hook (Fig. 4)

Simulations carried out on the finite element model revealed no fracture. The extension observed was very small (0.1 mm). Forces were propagated from the punch at the temporomandibular joint to the opposite side at the temporal level, with stresses mainly exerted at the junction of the brainstem and the spinal cord, frontal regions and at the falx cerebri.

- Anteroposterior (Fig. 5)

Simulations carried out on the finite element model in the anteroposterior direction on the jaw revealed that forces were propagated from the incisors to the maxillary arches, the septum, the ethmoid bone and then towards the palate and the sphenoid bone. These stresses were distributed in the frontal and temporal regions up to the occiput and the foramen magnum. In

brain tissue, stresses were propagated from the frontotemporal lobe to the occipital lobe, with greatest stress at the junction of the brainstem and the cervical spinal cord. A minimal fracture of the mandibular symphysis was visualized, with no penetrating or displaced fracture. Elongation of the cervical spinal cord was 2 mm, indicating cervical extension.

4. Discussion

4.1. Finite element model

Ours is one of the first complete models to include the fully modeled face and jaw, to which we added a neck model validated by the Laboratory of Applied Biomechanics in collaboration with the École Polytechnique de Montréal. Biomechanical studies have addressed mechanisms of injury either of the skull or of the spine, but not of the head and neck as a whole [18,19,23,27]. The relevance of this study is that it associates the cranial and cervical parts, which functionally are totally inseparable, in order to study their dynamics in facial impacts. Moreover, addition of the neck to our initial model allowed us to visualize the stresses exerted on the brainstem and the cervical spinal cord through three different mandibular impacts.

4.2. Force transmission and influence of impact location

Visible craniocerebral injuries (fractures, brain hemorrhages, contusions) give us little difficulty in understanding the mechanism involved in force transmission in a craniofacial impact. Our study also examines non-visible injuries, that is, non-hemorrhagic axonal injuries responsible for altered neurological functions and leading to death.

In anteroposterior facial impacts such as uppercuts, we observed hyperextension of the spinal cord/brainstem junction together with major stresses in this area, but without the cranial fractures, around the foramen magnum in particular, that have sometimes been observed in other studies [6,8,17,28]. Forces were distributed along the mandible to the base of the cranium,

with stresses passing from the frontal lobes to the occipital lobes, associated with high pressure at the brainstem and along the spinal cord. In order to keep as close as possible to real-life situations, we carried out impacts of the types of punches received in boxing.

A hook-type punch, on the other hand, did not cause hyperextension of the spinal cord through their mechanism, but considerable force was propagated without decreased intensity of the stresses measured at the impact zone of the brainstem.

Previously published head and neck models only examined brain injuries, and did not address the dynamics and stresses of brain/spinal cord tissue of the head and neck as a whole. During impacts or falls on the chin, injury by elongation or even rupture of the cervical spinal cord have in fact been described in the literature but not measured [3,6–8,29,30]. Voigt et al. reported brainstem injuries produced not only by hyperextension or anteflexion but also by torsion or other forces applied to the head [31]. Depending on the type of accident, brainstem injuries (partial severance) have been reported in vehicle drivers or passengers in high-velocity impacts where the face or forehead hit the dashboard or windscreen.

Our findings after hook-type punches were in agreement with those of Zivković et al. [32]. The location of cranial impact associated with specific cranial fractures is predictive of the presence or absence of pontomedullary injury. Lateral and frontal impacts are associated with the absence of pontomedullary injury, whereas impacts on the chin and the absence of direct cranial trauma are associated with pontomedullary injury, as we confirmed in our study. Jaw impacts cause violent movement of the head responsible for immediate craniocervical dislocations that may cause indirect brainstem injury, generally pontomedullary, because the pontomedullary junction is the thinnest, and therefore the weakest, anatomic part of the brainstem [32]. As we described in our previous publication [22], during a mandibular impact kinetic energy is transmitted from the mandible to the temporomandibular joints and then to the base of the cranium and to the brain. In our tests, we did not observe any fracture of the skull

base around the foramen magnum because of the lower impact velocities and forces used. In both situations, transmission of the impact force was decreased. The energy would thus be sufficient to produce a pontomedullary injury, but not sufficient to produce a fracture of the skull base. The development of pontomedullary injuries is dependent on impact energy and also on the position of the fracture, and less dependent on head movement. Mandibular and facial impacts cause acceleration of the head and rotational acceleration of the brain, and with sufficient impact energy, they may lead to rotation and deformation of the brain responsible for the brainstem injuries that we identified. The brainstem is not only a central sensory-motor pathway, but also a regulatory center for the major vegetative functions: vigilance, heart rate, respiratory rate, in particular at the level of the medulla oblongata. Simple contusion or compression of the medulla oblongata can thus lead to loss of consciousness and vegetative dysregulation that may result in death.

4.3. Protection of the face, fractures and anthropometrics

Although there have been controversial findings on facial fractures and brain injuries [33–36], our previous study and that of Zivković et al. [22,32] demonstrate the role of the facial bones in absorbing energy, protecting the brain and brainstem from the transmission of high kinetic energies. Mandibular fractures occur essentially at the impact point of each location, thus decreasing by half the force transmitted to the brain, as shown in our first article [22] in frontal and uppercut impacts. In lateral impacts, forces are transmitted directly to the base of the skull and so to the brain, without extension of the spinal cord but without decrease of stresses at the junction of the brainstem and the spinal cord. This mechanism of energy transmission has been described by Tse et al., by Zandi and Seyed Hoseini and by Lee et al., [5,37,38]. Tse et al. stated that the facial fractures closest to the brain are a major risk factor for underlying brain injuries [4].

In the literature, numerous comparative clinical studies have examined the number of fractures in relation to the severity of the brain injuries observed and have established a correlation, but without analyzing the dynamics of the forces applied nor their velocities.

These descriptive studies do not take into account the unique characteristics of each individual and the multiple factors that intervene in the mechanism of injury: shape of the mandible, bone density, dentition, underlying disease conditions, age… Finite element modeling offers an alternative to experimental research, enabling the digital reproduction of situations of injury and the possibility of evaluating an infinite number of conditions and injuries.

4.4. Limitations

Our modeling of a skull, brain and its spinal cord made it possible to locate stresses that had no clinical consequences. However, it could not reproduce axonal injuries and the extremely complex brain interconnections of the various sensory-motor pathways. Differentiation of gray and white matter and fluid-structure modeling of the CSF and brain vessels need to be added to improve this model.

4.5. Future prospects

Understanding the influence of the mode of impact and of the victim's characteristics on the development of brain lesions is of major importance. But the unique nature of the impact is not the only factor that has to be taken into account. So in the light of these data, we hope to study the effect on brain tissue of multiple lower-energy impacts and also to modify the environment by evaluating the stress produced when the facial impact occurs when the individual is lying on the ground or immobilized against a hard surface.

5. Conclusion

Using digital simulations based on a finite element model of the head and neck, we were able to analyze extra- and intracranial injuries following different mandibular impacts. This digital study enabled us to confirm the involvement and extension of the brainstem and cervical spinal cord during low-velocity impacts. Deaths secondary to this type of impact without identifiable histological lesions may be related to excessive stressing of the brainstem, along which pass sensory-motor pathways and the vagus nerve, and which is a regulatory center for the major vegetative functions. Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (mandible shape, dentition…) for a realistic approach to forensic applications.

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Responses to reviewers

Thank you for the reviewers' comments on our manuscript entitled "Study of cerebrospinal injuries by force transmission secondary to mandibular impacts using a finite element model". All of these comments were very helpful for revising and improving our paper. We have studied these comments carefully and have made corresponding corrections that we hope will meet with your approval. The changes in the revised manuscript are marked in red. The responses to the reviewers' comments are provided below.

We would like to express our great appreciation to you and the reviewers for the comments on our paper. If you have any further queries, please do not hesitate to contact us. Thank you very much to review this article

The relevance of this investigation should be shown by referencing literature. Literature referred to in this manuscript mainly concern higher loads (accidents, etc.). The reviewer also read the former publication [19]. There the relevance is discussed in more detail in the introduction.

We fully agree with this comment. We add this paragraph: "In literature, many authors demonstrated that the axon functional role can be altered even if it is not cut [11–14]. The brainstem pivots upon facial impact and suffers alterations by the subsequent shearing mechanisms [15]. Without fracture, the skull movement at impact may still have caused a direct brain contusion."

- References should be given concerning the material parameters and failure criteria used in this study (Table 1).

As suggested, the reference was add.

- The material properties of the glove/fist are not given. Was there any validation of the glove model?

We thank the reviewer for this comment. We did not validate the glove model, the glove used is a standard boxing glove identical to the one used by Viano during his experimental work. We used a Viano compliant glove model: a High density polyurethane foam.

- In section 3.1 force / stress propagation is described referencing Figures 3, 4 and 5. Obviously, in the figures only the stress propagation of the soft tissue (brain, brainstem, cord, etc.) is visualized. In the text, also stress propagation in bone is mentioned. Please add further figures showing van Mises stresses in bone.

- The authors should also refer to the times in Fig. 3 - 5.

We fully agree with this comment. The photos have been added, the times are different because there is a difference between the propagation of forces on the bone and in the soft tissues.

- The authors report an elongation of the cervical spinal cord of 2 mm. Please give some references concerning the injury risk of such an elongation (literature?).

We thank the reviewer for this comment. This has never been measured in the literature, but extensional lesions are described and visualized. Axonal lesions are described as well as vascular stripping in these same regions, which is why we are interested in this measurement. 2 Articles on elongation have been added. (Réf. 29 and 30).

Further remarks:

- In section 2.1 please add that individualized means CT based FE modelling / segmentation, etc.
- Section 2.3: Authors are not not given (… [18])
- Section 3: The comparative … table 2 !
- Section 3.1, Hook: Exerted instead of exercised ?`
- Section 4.2: Mandibular impacts cause .. deceleraction of the head => acceleration ?

We thank the reviewer for this remarks. These corrections were done.

CRediT author statement

Lucile Tuchtan: writing, Conceptualization, Methodology,

Yves Godio-Raboutet : Conceptualization, Methodology, software

Clémence Delteil : Visualization, Investigation,

Georges Léonetti : supervision

Marie-Dominique Piercecchi Marti : supervision, validation, rewieving

Lionel Thollon : supervision, validation, rewieving, sofrware

Annexe 3

Article soumis à l'International Journal of Legal medicine

International Journal of Legal Medicine

Sudden death after facial impacts: is the brainstem involved?

--Manuscript Draft--

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Full Title:	Sudden death after facial impacts: is the brainstem involved?
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Corresponding Author:	lucile tuchtan, M.D Assistance Publique Hopitaux de Marseille Marseille, Bouches du Rhone FRANCE
Corresponding Author Secondary Information:	
Corresponding Author's Institution:	Assistance Publique Hopitaux de Marseille
Corresponding Author's Secondary Institution:	
First Author:	lucile tuchtan, M.D
First Author Secondary Information:	
Order of Authors:	lucile tuchtan, M.D
	Clemence Delteil, MD
	yves Godio Raboutet
	Martin Kollop, MD
	Georges Léonetti, MD, PhD
	Lionel Thollon, PhD
	Marie Dominique Piercecchi-Marti, MD, PhD
Order of Authors Secondary Information:	
Funding Information:	
Abstract:	Three deaths following facial impacts in the presence of witnesses and resulting in brain lesions that were visualized only on histopathological examination were studied at the forensic medicine institute of Marseille. Craniofacial impacts, even of low intensity, received during brawls may be associated with brain lesions ranging from a simple knock-out to fatal injuries. In criminal cases that are brought to court, even by autopsy it is still difficult to establish a direct link between the violence of the impact and the injuries that resulted in death. During a facial impact, the head undergoes a movement of violent forced hyperextension. Death may thus be secondary to the transmission of forces to the brain, either by a mechanism involving nerve conduction that may be termed a reflex mechanism (for example by vagal hyperstimulation) or by injury to the central nervous system (axonal damage). In such situations, autopsy does not make it possible to determine the cause of death, but only to suspect it in a context of voluntary violence in the presence of witnesses, with or without violent injury observed on external examination or on superficial incisions to determine the extent of bruises or hematoma. Only histological analysis is contributory.
Author Comments:	
Suggested Reviewers:	Norbert Telmon, Phd telmon.n@chu-toulouse.fr
	Valery Hedouin valery.hedouin@univ-lille.fr

Case report

Sudden death after facial impacts: is the brainstem involved?

Lucile Tuchtan[1,2,*] • Clémence Delteil[1,2] • Yves Godio-Raboutet[3,4] • Martin Kollop [1] Georges Léonetti[1,2] • Lionel Thollon[3,4] • Marie-Dominique Piercecchi-Marti[1,2]

[1] Forensic Department, APHM, Hôpital de la Timone, 264 rue Saint Pierre, 13385 Marseille, France

[2] Aix Marseille Univ, CNRS, EFS, ADES, 27 avenue Jean Moulin, 13385 Marseille, France

[3] Aix Marseille Univ, IFSTTAR, LBA, boulevard Pierre Bramard 13015 Marseille, France

[4] iLab-Spine (International Laboratory – Spine Imaging and Biomechanics), boulevard Pierre Bramard 13015 Marseille, France

* Corresponding author. Tel.: +33 491482245; fax: +33 491923331

E-mail address: lucile.tuchtan@ap-hm.fr (Lucile Tuchtan)

Lucile Tuchtan ORCID: 0000-0001-6248-2840

Clémence Delteil ORCID: 0000-0001-5171-8520

Lionel Thollon ORCID: 0000-0002-4456-0902

Abstract

Three deaths following facial impacts in the presence of witnesses and resulting in brain lesions that were visualized only on histopathological examination were studied at the forensic medicine institute of Marseille. Craniofacial impacts, even of low intensity, received during brawls may be associated with brain lesions ranging from a simple knock-out to fatal injuries. In criminal cases that are brought to court, even by autopsy it is still difficult to establish a direct link between the violence of the impact and the injuries that resulted in death. During a facial impact, the head undergoes a movement of violent forced hyperextension. Death may thus be secondary to the transmission of forces to the brain, either by a mechanism involving nerve conduction that may be termed a reflex mechanism (for example by vagal hyperstimulation) or by injury to the central nervous system (axonal damage). In such situations, autopsy does not make it possible to determine the cause of death, but only to suspect it in a context of voluntary violence in the presence of witnesses, with or without violent injury observed on external examination or on superficial incisions to determine the extent of bruises or hematoma. Only histological analysis is contributory.

Keywords: Head trauma • Brainstem • Axonal injury • Mandibular impact

Introduction

Craniofacial impacts are well known to boxers, but even low-intensity impacts received during brawls may be associated with brain injury ranging from simple loss of consciousness to fatal lesions [1–3]. In criminal cases, the judge cannot consider that a compatible chronological course of events is sufficient to affirm causality, even if several witnesses were present. The difficulty of establishing the link between the violence of the impact and the injury responsible for death is still present at autopsy, and further investigations are required, histopathology in particular.

During a facial impact, the head undergoes a movement of forced violent hyperextension. Two simultaneous and concurrent physical mechanisms come into play: a contact effect resulting in local lesions at the point of cranial impact, and an inertia effect when the head is set in motion (acceleration) or when motion ceases (deceleration), which causes diffuse and multifocal lesions. The movement of the head can in itself cause direct concussion even if there are no immediately evident anatomic lesions, and independently of possible cervical cord injury due to cervical "whiplash" injury which is more frequently described in the infant [4]. In practice, the contact effect and the inertia effect act together to produce contusion lesions at the surface of the brain and/or axonal lesions and/or suspension of nerve function with the possibility of axonal involvement causing deafferentation.

Death may thus be secondary to transmission of force to the brain either by a mechanism involving nerve conduction, known as a reflex mechanism (for example by vagal hyperstimulation) [5–8], or by central nervous system lesions (axonal damage) [9–12].

In such situations, autopsy does not make it possible to determine the cause of death, but only to suspect it in a context of voluntary violence in the presence of witnesses, with or without violent injury observed on external examination or superficial incisions to determine the extent of bruises or hematoma. We report three cases of deaths following facial impacts where the victims presented with brain lesions that were visualized only on pathological analysis, which made it possible to support the mechanisms of traumatic injury.

Case reports

Case 1

A 32-year-old man was assaulted, in the presence of several witnesses who gave identical accounts of the event, by several persons who delivered four or five uppercut type punches while he had his back against a wall and then threw him to the ground. The victim was described as already unconscious when he received a kick in the face.

According to a witness who assisted him immediately after the event, the victim appeared to be alive. The witness placed him in the lateral security position and maintained the neck and head in the axis of the trunk with his hand. Respiratory pauses occurred. When the professional emergency services took over 20 minutes later, the victim was in cardiorespiratory arrest. He was declared dead 45 minutes later in spite of transient return of circulation. At autopsy, only superficial external injuries were found in the facial and cervical region, such as abrasions of the brow bone, left eyelid, left cheek and the posterior cervical region, as well as rib fractures. No fractures of the skull, face or neck or peri-, intracerebral or spinal cord hemorrhage were observed. However, the victim had a hypertrophic heart which pointed to decompensation of a pre-existing state rather than to a direct effect of the blows received, particularly as toxicology later showed a high blood alcohol level (2.28 g/L). The pathology expert report confirmed the hypertrophic cardiomyopathy, but was not able to define its cause or affirm its role in the lethal mechanism of a previously unknown disorder. The report confirmed the absence of intracranial hemorrhagic changes. However, immunostaining with anti-beta-amyloid precursor protein (APP) antibodies revealed axonal lesions of the pedoncles, pons, medulla oblongata and the nerve roots. Lesions in this location produce respiratory and then circulatory arrest, which appeared to be compatible with the victim's clinical state as described by the witnesses and were in support of a direct link between the acts of violence and death.

Fig. 1 Axonal lesions of the medulla oblongata (x 100) (A) and upper medulla oblongata (x 100) (B).

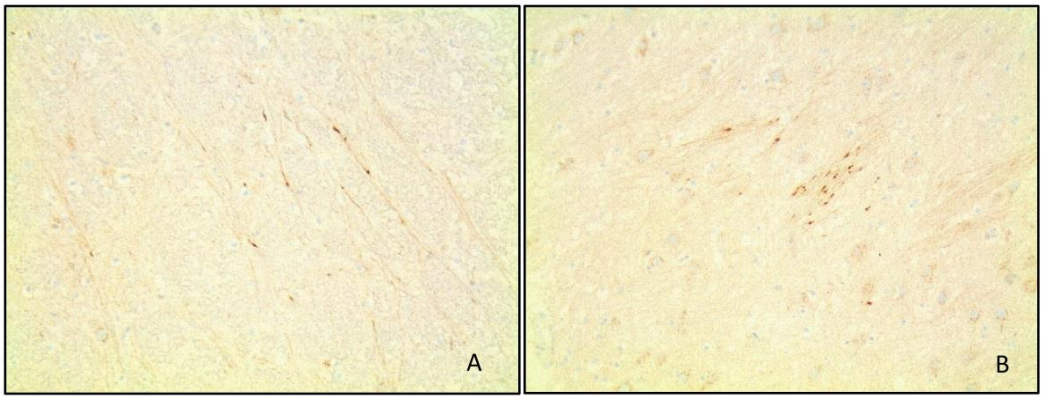
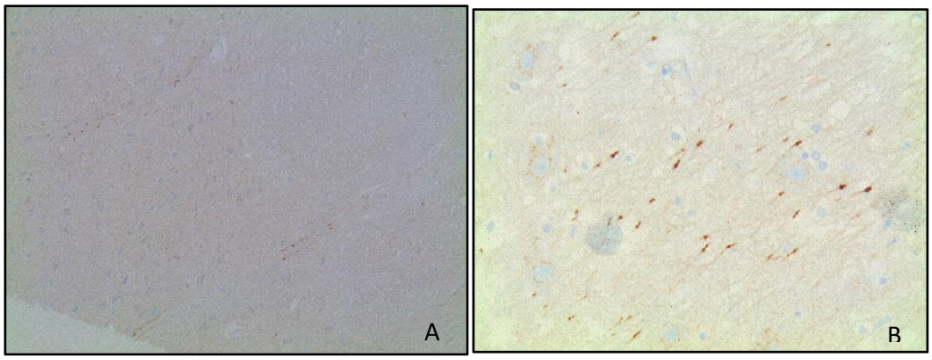


Fig. 2 Axonal lesions of the peduncles x 50 (A) and x 200 (B)

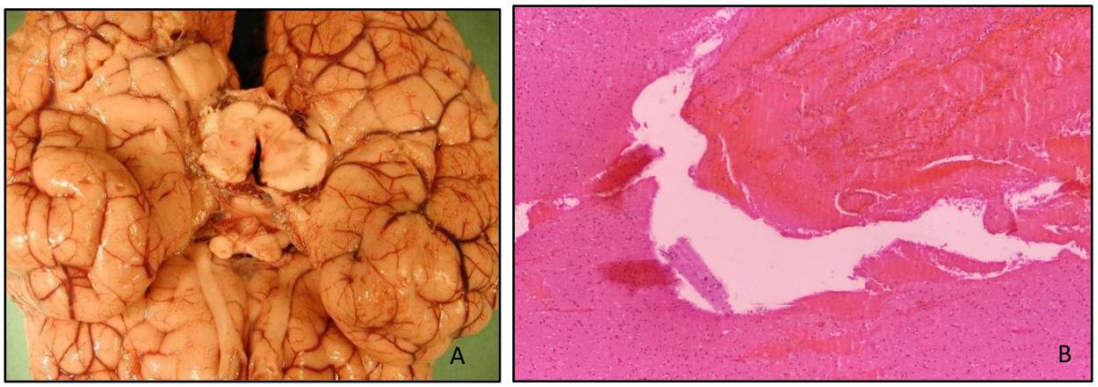


Case 2

A 14-year-old adolescent, who had recently been admitted to a young people's home, received before witnesses a kick in the face following a dispute with another adolescent. According to the witnesses, he immediately had an altered state of consciousness which was rapidly followed by cyanosis and respiratory arrest, then by a convulsive episode and urinary incontinence before circulatory arrest. Cardiac activity was restored after 40 min of resuscitation but the adolescent progressed to brain death one day later. Organ removal was performed (liver, kidneys). Antemortem examination of his injuries revealed fracture of the nasal bones but no brain hemorrhage. The initial brain CT scan showed no abnormality of supra- or subtentorial density. No spinal bone lesions were identified. The autopsy revealed no skin lesions and in particular no facial lesions. Only cerebral edema associated with a thin hemorrhagic film next to the brainstem was observed on gross examination.

As well as anoxic-ischemic edema, pathological examination revealed marked median laceration of the brain peduncles passing through the ventricle, with hemorrhagic margins. This laceration accompanied by axonal lesions was interpreted as being of traumatic origin secondary to the violent facial impact.

Fig. 3 Inferior view of the brain showing transection of the peduncle: marked median laceration (A). View of a histological section (B).



Case 3

A 42-year-old man was found dying on the ground in front of his home by his female friend. When the emergency services arrived, he was in cardiorespiratory arrest. Resuscitation was attempted for 40 min but was unsuccessful. At the scene, there was a blood stain under the victim's head with splashes of blood. His friend had last seen him on the night before the event during an evening of drinking at their home, where a third person was believed to have been present. As the street was equipped with security cameras, it was possible to observe the circumstances of death. An individual had delivered a series of punches to the forearms (the victim initially attempted to protect himself), the trunk and lastly to the face, with an uppercut that caused the victim's immediate collapse. At autopsy, numerous facial and cervical bruises were found, associated with underlying hemorrhagic infiltrations without associated hemorrhagic brain lesions, and numerous bruises and hematomas of the limbs. Pathological examination found hemorrhagic laceration of the cerebral peduncles and axonal lesions in the same area.

Discussion

Even by autopsy, it is still currently difficult to establish a direct link between acts of violence and death. Supplementary investigations, and pathological examination in particular, are indispensable to identify lesions that are not visible on external gross examination and so to explain the mechanism of injury. In the course of legal proceedings, the judge cannot simply accept a compatible chronological chain of events described by several "objective" witnesses present at the scene in order to affirm a definite causal link between the events and death unless he or she can rule out other causes of death. If there are no identifiable lesions, the mechanism of injury that is suggested results from well-grounded argumentation that rules out all possible diagnoses one by one, thus

leaving a diagnosis of exclusion. Such arguments are not admissible by a tenacious lawyer, as witness reports are not considered as irrefutable evidence (vague description of the mechanism of injury, site of impact unclear).

The cases reported here of deaths following facial injuries, whose clinical consequences were described by impartial witnesses, are substantive evidence of the link between injury and death. The literature contains descriptions of brainstem lesions, with or without associated fractures, particularly after high-velocity impacts in road traffic accidents [7,13]. The cases we report show that punches or kicks can produce injuries that are just as severe. In these contexts of closed head and brain injury involving forces of acceleration or deceleration, lesions have been described in the central axial regions of the brain [14], including at the level of the brainstem lesions of the superior colliculi, the roof of the fourth ventricle, the medial reticular formation (including the dorsal medullary nuclei), the middle and lower cerebral peduncles, and the pons.

The hypothesis of a reflex mechanism during intense stretching of the brainstem is a probable one because of the passage of the major sensory motor pathways, the reticular formation (including the pre-Bötzinger complex), and the presence of central nuclei in this pivotal zone [15,16]. The vasomotor centers and the central pattern generator for respiration lie in the brainstem. These centers are responsible for short-term regulation of arterial blood pressure and for the nerve supply of the muscles of the thoracic cavity and the upper airways, respectively. Involvement of these centers may lead to death due to considerable slowing of the heart rate or respiratory arrest. Our subjects were young and in good health. Only one had a pre-existing heart condition which could have decompensated as a result of stress after blows to the trunk, causing cardiac arrest. This point, which initially appears of little significance, was of fundamental importance for the magistrates, in particular enabling them to bring a charge against the persons accused.

In order to prove axonal damage of traumatic origin, the pathologist must be alerted of the need to obtain samples in target areas such as the peduncles and the medial axis of the brain in general, as in suspected shaken baby syndrome [14,17]. However, anti-beta APP antibody immunostaining is required to reveal these axonal lesions. When neurons are damaged, beta-amyloid precursor protein accumulates in the axons due to inhibition of axonal transport [18]. In the adult, axonal lesions can be observed when survival is longer than 2 to 3 hours [19–22]. Interpretation of anti-APP immunostaining must follow fundamental principles: the duration of survival after cardiocirculatory arrest, thanks to resuscitation measures, must be sufficiently long, and vascular perfusion of the brain must be adequate during survival. Conversely, survival for too long a period may result in a false negative result [23].

Furthermore, too long a survival time can lead to torsion of the peduncles because of subtentorial involvement, leading to Duret hemorrhage (transtentorial herniation) through ischemia, which may be either masked or mistaken for traumatic lesions [24].

The marrow of the cervical spine is also affected by hyperextension of the neck [25] and it is indispensable to systematically obtain samples.

In the literature, some authors such as Belingardi et al. have demonstrated that the brainstem acts as a pivot in a facial impact and that it undergoes major damage during shear stress [26]. Lesions of varying degrees of severity may result, from transient local locomotor paralysis to a range of more or less extensive parenchymal lesions. These effects of inertia may be observed when the head is violently shaken without an impact (particularly in hyperextension): for example, a driver who has a whiplash injury when his vehicle is hit from behind, a rugby player or footballer pushed from behind, a boxer who receives an uppercut, or a helmeted head that receives an impact.

Another team, that of Gennarelli et al., studied the conditions in which diffuse axonal lesions are produced, and concluded that the functional consequences following loss of consciousness were directly related to axonal destruction at the time of impact [9]. Other studies have shown that the axons could be functionally altered even if they were not transected [10,12,27,28]. The lesions due to stretching are found in areas of low axonal resistance: the transitional zones in the brain between white and gray matter, the periventricular white matter, the corpus callosum, and extremely severely in the dorsal midbrain. For some years, biomechanical studies have been performed both experimentally on bodies donated to science and by simulation using digital image-based finite element models [29–31]. The digital approach improves our understanding of the mechanisms of injury involved and scenarios of injury can be reproduced ad infinitum.

Energy transmission to the skull following mandibular impacts has been reported to produce stresses affecting the brainstem but without associated cranial fractures, in particular following uppercut punches [32] The presence of axonal lesions confirms the stresses produced at this transitional zone.

Conclusion

Death following facial trauma with no identifiable or with only microscopic lesions is rare and this is a unique description of three cases. These anecdotal cases confirm that stretching of the brain stem can cause death by

sideration and/or axonal lesions. This mechanism of injury is no longer suggested by elimination only, but becomes true evidence that can be defended in court before the magistrates.

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Résumé

Etablir le lien de causalité entre des violences physiques et le décès d'un individu est une problématique récurrente de la pratique médico-légale. Une grande partie des situations de violence ne pose pas de difficulté au médecin légiste. Toutefois, cette causalité n'est parfois que supposée sur un continuum temporel entre des faits de violence et le décès sans démontrer le mécanisme lésionnel. Cette situation peut provenir de l'absence de traduction anatomique de ce mécanisme lésionnel, illustrée par des cas de décès secondaires à des impacts faciaux, observés à l'institut médico legal où seules des lesions histologiques étaient objectivées.

Pour répondre à cette problématique, un premier modèle par éléments finis de tête a été réalisé initialement afin d'étudier la transmission des forces au crâne à partir d'un impact mandibulaire. Nous avons observé une diminution des transmissions des efforts à la base du crâne suite à un impact mandibulaire ainsi que des contraintes de Von Mises au niveau du tronc cérébral notamement. Ce modèle a été amélioré par l'ajout d'un cou et de la moelle épinière cervicale afin d'étudier plus précisemment les contraintes au niveau du tronc cérébral, lors d'impacts mandibulaires type uppercut ou antéro posterieur, des contraintes au niveau de la jonction cérébro spinale ainsi qu'une hyperextension de la moelle sont observées.

Ces mécanismes lésionnels mettant en jeu des contraintes dans la zone des pédoncules cérébraux, sont en accord avec nos résultats de simulations numériques par éléments finis. Ce niveau de contrainte observé, très proche de la valeur seuil traduisant des lésions cérébrales, laisse présager de possibles lésions axonales.

Les paramètres biologiques variant selon chaque individu, la modélisation numérique permet de les moduler à l'infini (forme de mandibule, dentition…) pour une approche réaliste d'applications médico-légales.

Mots clés : Modèle en éléments finis, Lésions cérébro spinales, Transmission des forces, Tronc cérébral, Impact mandibulaire.

Abstract

Establishing the relationship between the death of an individual and a violent event is a common practice in forensic science. Many cases of death by violence are relatively obvious for the medical examiner. However, the process of identifying the causes of the death is sometimes solely based on the time-event continuum between the violence act and the death without confirmation of the injury mechanisms. This situation may be the case when anatomical signs of the injury mechanisms are absent, illustrated by cases of death secondary to facial impact, observed at the Institut médico legal, where only histological lesions were objectified.

To answer this problematic, a first finite element model of head was realized initially to study the forces transmission to the skull starting from a mandibular impact. We observed a decrease of the efforts transmissions at the skull base following a mandibular impact as well as constraints of Von Mises at the level of the brainstem in particular. This model was enhanced by the addition of a neck and cervical spinal cord to more accurately study brainstem strains, uppercut or anteroposterior mandibular impact, cerebral junction and hyperextension of the cord are observed.

These lesional mechanisms involving stresses in the area of the cerebral peduncles, are in agreement with our results of numerical simulations by finite elements. This level of stress observed, very close to the threshold value reflecting brain lesions, suggests possible axonal lesions.

Biological parameters are different in each individual, and by using digital modeling they can be modulated at will (jaw shape, dentition ...) for a realistic approach of forensic applications.

Keywords: Finite element model, Cerebrospinal injury, Force transmission, Brainstem, Mandibular impact.