

No. 44337-6-II

IN THE WASHINGTON STATE COURT OF APPEALS
DIVISION II

STATE OF WASHINGTON,

Respondent,

vs.

LEANNE M. BECHTEL

Appellant.

APPEAL FROM THE SUPERIOR COURT

OF PIERCE COUNTY

Cause No. 11-1-01077-7

OPENING BRIEF OF APPELLANT

BRETT A. PURTZER
WSB #17283

HESTER LAW GROUP, INC., P.S.
Attorneys for Appellant
1008 South Yakima Avenue, Suite 302
Tacoma, Washington 98405
(253) 272-2157

Table of Contents

TABLE OF AUTHORITIES.....	2
I. ASSIGNMENTS OF ERROR.....	3
II. ISSUES PERTAINING TO ASSIGNMENTS OF ERROR.....	4
III. STATEMENT OF THE CASE.....	5
A. Procedural History.....	5
B. Facts.....	6
IV. ARGUMENT.....	14
V. CONCLUSION.....	31

TABLE OF AUTHORITIES

Cases

<u>Boeing Co. v. Heidy</u> , 147 Wn.2d 78, 51 P.3d 793 (2002).....	26
<u>Frye v. United States</u> , 293 F. 1013 (D.C. Cir. 1923).....	15, 24, 31
<u>Gazijo v. Nicholas Jern Co.</u> , 12 Wn.App. 538, 541, 530 P.2d 682, <u>affd.</u> , 86 Wn.2d 215 (1975).....	15
<u>Miller v. Likins</u> , 109 Wn.App. 140 (2001)	18
<u>State v. Allery</u> , 101 Wn.2d 591, 682 P.2d 312 (1984)	24
<u>State v. Black</u> , 109 Wn.2d 336, 745 P.2d 12 (1987)	16, 24
<u>State v. Canaday</u> , 90 Wn.2d 808, 812, 505 P.2d 1185 (1978)	16
<u>State v. Castellanos</u> , 82 Wn.App. 204, 205, 916 P.2d 893 (1996).....	29
<u>State v. Cauthron</u> , 120 Wn.2d 879, 888, 846 P.2d 502 (1993).....	16
<u>State v. Koontz</u> , 145 Wn.2d. 650, 653, 41 P.3d 475 (2002).....	28, 29, 30
<u>State v. Phillips</u> , 123 Wn.App. 761, 756 (2004)	18
<u>State v. Rogers</u> , 70 Wn.App. 626, 633-34, 855 P.2d 284 (1993).....	28
<u>State v. Stockmyer</u> , 83 Wn.App. 77, 920 P.2d 1201 (1996)	28
<u>State v. Wigley</u> , 5 Wn.App. 465, 466, 488 P.2d 766 (1971).....	15
<u>Stedman v. Stacey Cooper</u> , 172 Wn.App. 9, 292 P.3d 764 (2012)	24, 25, 26, 27
<u>Walker v. State</u> , 121 Wn.2d 214, 218, 848 P.2d 721 (1993).....	18

Statutes

4.44 RCW	4, 29, 30
----------------	-----------

Other Authorities

<i>Head & Spinal Injuries</i>	19
<i>Head Impact Reconstruction – HIC – Validation & Pedestrian Injury Risk</i>	21
<i>Injury Biomechanics for Aiding in the Diagnosis of Abusive Head Trauma</i>	23
<i>Mechanical Properties & Anthropometry of the Human Infant Head</i>	21
<i>Pediatric Biomechanics</i>	22, 23

Rules

CrR 7.5	5
ER 702.....	15, 24

Constitutional Provisions

Fourteenth Amendment.....	28
Sixth Amendment	28
Washington Constitution Art. 1 § 22	28

I. ASSIGNMENTS OF ERROR

1. The trial court erred when it allowed the biomechanical expert to testify.
2. The trial court erred when it denied appellant's motion for new trial based on jury misconduct.

II. ISSUES PERTAINING TO ASSIGNMENTS OF ERROR

1. Whether the trial court erred when it allowed the biomechanical expert to testify when the expert's opinion was not generally accepted in the scientific community? (Assignment of Error #1)

2. Whether the trial court erred when it allowed the biomechanical expert to testify when the expert's testimony would not assist the trier of fact as it was based on speculation? (Assignment of Error #1)

3. Whether the trial court erred when it denied defendant's motion for new trial based on jury misconduct when the jury separated and considered evidence in violation of RCW 4.44.300? (Assignment of Error #2)

III. STATEMENT OF THE CASE

A. Procedural History

On March 14, 2011, the State filed one count of Murder in the Second Degree for the death of A.F., a minor, that occurred on or about April 30, 2008. CP 3-4. Pre-trial, the defendant filed a motion and memorandum to exclude the State's injury biomechanic expert's testimony. CP 33-99. The state filed a response. CP 117-294.

Trial began September 26, 2012 whereupon the court heard defense's motion to exclude the state's biomechanic expert and re-enactment. The court denied the defense motion. CP 399-400. RP 108-168. On October 4, 2012, jury selection began and testimony began October 8, 2012. On October 26, 2012, the jury returned a guilty verdict and made special findings that the victim was particularly vulnerable and that Ms. Bechtel abused her position of trust. CP 401-403.

After the jury's verdict, Ms. Bechtel filed a motion and declaration for a new trial pursuant to CrR 7.5(a)(2) based on juror misconduct. CP 404-409. The state filed responsive declarations and a memorandum. CP 410-476. The court denied the motion. RP 11-15 (11/16/12).

The court sentenced Ms. Bechtel to 234 months within the Department of Corrections. CP 517-530. On December 17, 2012, Ms. Bechtel filed her notice of appeal and this appeal follows. CP 536-552.

B. Facts

Chris Franks, A.F.'s father, met Leanne Bechtel in 2006. They started dating and began residing together in February, 2007, and were engaged to be married. RP 325:11-326:17. When Mr. Franks moved in with Ms. Bechtel, she owned a pit bull, Dozer, who was a puppy, but very hyper. RP 327:2-13. Dozer was a big dog and could become very rough at times. RP 377:10-14.

In early 2008, Mr. Franks and Ms. Bechtel moved into a small one-bedroom apartment that would accept pets. RP 331:5-12. Although Mr. Franks had two young children, J.F. and A.F., from a prior relationship, no children were living with them at that time, and they had no plans for the children to move in with them when they first rented the apartment. *Id.* at 13-17. Soon after they moved into the apartment, however, Mr. Franks' children came to live with them, and Ms. Bechtel welcomed them as her own. RP 374:19-24. Because they lived in a one-bedroom apartment, Ms. Bechtel and Mr. Franks slept in the bedroom and the children slept in the living room with the dog, Dozer. RP 332:10-17.

During this time, Mr. Franks worked from 5:00 a.m. until 1:30 p.m. and, typically, would go to bed between 7:30 and 8:00 o'clock at night and leave for work by 4:30 a.m. Ms. Bechtel would get up with him while the children slept. RP 335:19-336:13; 339:3-11. Because Ms. Bechtel was not employed outside the home, RP 336:18-20, her responsibility was to maintain the household, which included meal planning, laundry, house cleaning, grocery shopping, and food preparation. RP 336:18-327:20. Ms. Bechtel was also responsible for taking care of A.F. throughout the day while J.F. was at school. RP 338:4-7. Ms. Bechtel's

relationship with the children was excellent, and A.F. referred to Ms. Bechtel as her mother. RP 342:18-343:6.

Ms. Bechtel helped nurture the children and they were thriving, particularly A.F. RP 375:11-20, 377:5-9. Ms. Bechtel helped A.F., who was not very vocal at that time, learn her alphabet, her numbers, her colors, and she would play with both children for hours. RP 375:21-376:9. In fact, when the children were with their biological mother, Amanda Nichols, or at Mr. Frank's mother's home, upon returning to the apartment, they would run to Ms. Bechtel. Ms. Bechtel and A.F. were basically inseparable. RP 376:17-23.

Although the kids adored Dozer and Dozer was extremely protective of them, he would knock the children around the room, and had even knocked over Mr. Franks in the past. RP 377:15-378:20. Dozer became particularly excitable at bath time, and he would race around the apartment, jump on the bed, lunge across the room, and jump on the couch using it as a springboard when his bath was being prepared. RP 349:9-20, 350:8-14; 378:21-379:4, 379:5-7.

Sometime before April 30, 2008, Dozer struck A.F., which caused her to hit her head against a door frame, bruising her head. RP 346:16-347:8; 347:23-348:12; 379:18-380-3. At some point, Mr. Franks and Ms. Bechtel had a discussion about Dozer being so hyper and that he was too big for the apartment. Ms. Bechtel did not want to get rid of the dog. RP 327:22-328:1, 350:24-351:8.

Lora Franks, Mr. Franks' mother, also urged them to get rid of the dog because Dozer was too big, too powerful, and too strong to be around the little kids. RP 451:10-14. Ms. Franks noted three separate occasions when Dozer had

struck A.F. and injured her, one of which was so significant that she would have taken A.F. to the doctor. RP 453:7-22; 458:22-25. Ms. Franks was concerned because A.F. was a tiny girl and Dozer weighted at least 80 pounds. RP 453:25-454:2.

On April 30, 2008, Lakewood Fire/Paramedics responded to a 911 call from the Bechtel/Franks residence involving A.F., 3 ½ years old, who had been knocked off a couch by Dozer and was unconscious. RP 192:21-193:8. Ms. Bechtel immediately called 911, and when the paramedics arrived, Ms. Bechtel, who was visibly crying and upset, showed them where A.F. was lying on the floor in front of the couch. RP 195:18-196:17; 217:9-218:1. Ex. 135. Ms. Bechtel told the paramedics that Dozer knocked A.F. off the couch. RP 198:23-25. Off to the right of the couch was a cage with Dozer inside. RP 196:22-25. A.F. was approximately three feet away from the cage, lying somewhat parallel to the couch, RP 198:12-17, which position the paramedic estimated by way of a diagram. RP 218:15-20, 219:1-4. The paramedics noted that A.F.'s pupils were fixed and dilated, which signified head trauma, and that she had a weak pulse, signifying low blood pressure. RP 202:22-203:6. The paramedics did not note any bruising to A.F.'s face or chest. RP 205:10-11. As a result, A.F. was transferred to Mary Bridge Children's Hospital. RP 214:3-6. Ms. Bechtel rode in the ambulance with them to Mary Bridge. RP 229:25-230:2. On the way to the hospital, four separate individuals provided care to A.F. and the paramedics noted that a person may become bruised as a result of that type of treatment. RP 233:7-

23. Once the paramedics arrived at Mary Bridge Children's Hospital, they transferred A.F.'s care to the trauma team. RP 216:2-6.

The emergency room physician treating A.F., noted that her pupils were abnormal, she was unresponsive and toxic appearing, and looked extremely ill. RP 278:14-279:17. Because the doctor believed she had suffered a head injury, he ordered a CT scan of her head and neck. RP 280:16-24. The head CT's revealed a depressed skull fracture which extended to the base of the skull in an area called the foramen magnum, subdural hematomas, and cerebral edema, i.e., swelling of the brain. RP 283:14-284:9; 648:18-649:15; 651:19-20. The hematomas were both acute and sub-acute denoting at least two different traumatic events. Acute being within the first few hours after the event and sub-acute being from an earlier period of time. RP 284:20-24; 315:4-7. Because of A.F.'s injuries, the emergency room doctor was concerned that A.F. was a victim of abuse or non-accidental trauma. RP 291:12-25. The doctor was also aware that falls from very low heights can cause significant skull fractures, RP 306:9-16, and that a patient's individuality may make the patient more susceptible to injury. RP 306:17-307:3.

A.F.'s care was transferred to a pediatric critical care physician who testified that A.F.'s injury likely occurred 6-12 hours before she arrived at the hospital, RP 490:17-491:10, and that her injuries were more consistent with having occurred at approximately 4:00 a.m. the morning of April 30, 2008, much earlier than reported by Ms. Bechtel. RP 492:23-493:13, 520:21-25, 534:8-11; 564:23-565:2. In a prior statement, the doctor opined that the period of time

required for the amount of swelling and cerebral edema A.F. suffered was between 12-24 hours before he saw the patient, RP 547:4-548:12, 549:20-25; 551:8-10, 552:16-553:10, 554:6-555:24, 557:22-558:15, which placed the traumatic event occurring between 8:00 and 9:00 p.m. the previous evening. RP 559:1-4.

A neurosurgeon performed a hemi-craniotomy on A.F. and noted both recent and old blood, which signified a recent and older injury. RP 696:21-701:2. The neurosurgeon testified that any type of skull fracture to the back of the head can cause brain swelling and disrupt the sagittal sinus, which he believed caused A.F.'s brain to swell. RP 705:15-21, 692:20-695:4. The doctor further opined that a short distance fall can cause the type of fracture that he found in A.F.'s case. RP 706:2-7, 708:21-24.

Lakewood Police Officer Lattimer contacted Ms. Bechtel while she was at the hospital. RP 244:20-22. Ms. Bechtel was extremely "upset, crying and had difficulty maintaining her composure." RP 245:8-10; 248:20-249:5. When questioned, Ms. Bechtel stated that A.F. had been jumping on the couch, that she had a hyper dog, that the dog had knocked A.F. off the couch, and that A.F. landed on her head. RP 245:19-22.

When Mr. Franks contacted Ms. Bechtel at the hospital, she was hysterical, in tears, and uncomprehensible. RP 363:22-364:5; 381:13-18. Ms. Bechtel told him that Dozer had knocked A.F. off the couch. RP 364:16-21. She stated that while she was preparing A.F.'s bath, A.F. was on the couch and Dozer knocked her off. RP 365:10-14. She said that when A.F.'s head hit the floor, it

sounded like a watermelon being squashed. RP 368:19-22. During the time that the two were together after A.F.'s death, Ms. Bechtel's statement never changed. RP 372:4-7. At no time did Ms. Bechtel's statement to the 911 operator or any other witness change with respect to how the dog struck A.F. and knocked her to the floor. RP 1505:1-18.

When Tacoma Police Department detectives, Eggleston and Westby, interviewed Mr. Bechtel at the hospital later that morning, she told them that on the prior day, April 29, 2009, after Mr. Franks returned home from work, the family went to Clover Park High School for swim lessons, and, upon returning home, they sent J.F. to bed early as punishment for misbehaving at school. RP 823:12-17. Later that evening, Ms. Bechtel, Mr. Franks and A.F. went into the bedroom, watched TV and then went to bed. RP 824:20-825:5.

The next morning, Mr. Franks arose about 4:00 a.m. and Ms. Bechtel said goodbye to him as he left for work. She then returned to bed and woke up around 6:30 to get J.F. ready for school. RP 825:10-19. Ms. Bechtel stated that A.F. arose at approximately 7:00 that morning whereupon they walked J.F. to the bus stop. RP 826:21-25. After returning home, she fed A.F. breakfast and then prepared A.F.'s bath. RP 828:11-829:8. While Ms. Bechtel was on her way to the bathroom, she saw A.F. standing on the couch in the living room with her upper body leaning into the back of the couch looking into a mirror, exclaiming, "look mommy, I'm a princess mommy". RP 829:24-830:5. While the bathtub was filling, Ms. Bechtel called out to A.F. to ask her if she was ready to take her bath whereupon Dozer began running around the living room. RP 834:5-9. Ms.

Bechtel explained that Dozer likes his bath and that he thought the water was for him. Dozer then jumped on the couch in between A.F. and the back of the couch and knocked her onto the floor. RP 834:14-18. Ms. Bechtel exclaimed that she saw something out of the corner of her eye falling back, flying across the room, and then saw A.F. hit the back of her head on the floor. RP 835:3-6; 836:2-11. While on her back, A.F. was unresponsive as she wouldn't look at Ms. Bechtel, her eyes moved from side to side, but she wouldn't focus. RP 836:19-837:7. Ms. Bechtel immediately called 911 and placed Dozer into his kennel per the 911 operator's instruction. RP 837:10-21. When Lakewood Police Chief Zaro arrived at the Franks/Bechtel residence, he noted bath water in the bath tub. RP 595:5-6, 606:20-21.

Both Mr. Franks and Ms. Bechtel stated that A.F. was not feeling well days before the event on April 30, 2008, and had vomited, which could be evidence of a prior subdural hematoma. RP353:25-354:1; 388:2-389:5; 837:22-838:3, 1511:12-22.

After the detectives thanked Ms. Bechtel, she approached Detective Eggleston, buried her head into his shoulder, and gave him a long hug. RP 838:11-15; 844:19-845:19.

Dr. Feldman, one of the state's child abuse experts, opined that A.F.'s injuries resulted from non-accidental trauma, but he could not rule out that her injuries were accidental. Dr. Feldman recognized that because of A.F.'s prior brain injury, she may have reacted more adversely to the new injury she sustained. RP 768:20-769:3. He acknowledged that a subdural hematoma can

result from accidental trauma, and that a skull fracture is not required before an individual will suffer a subdural hematoma. RP 769:18-19; 779:13-15. Dr. Feldman also acknowledged the difficulty of recreating the dynamics of a fall because of the variability involved, including those related to children as each is an individual and variations exist as to the amount of force necessary to cause a child to be knocked unconscious from a fall. RP 786:17-787:5, 788:17-789:13. Further, Dr. Feldman acknowledged the amount of force generated by the dog would be difficult to quantify as too many unknowns exist to determine the amount of force the dog impacted on A.F. RP 790:1-19.

Over defense objection, the State called Dr. Wilson Hayes, an injury biomechanic expert, to testify about a fall analysis and event reconstruction he prepared. RP 853:19-854:3; 876:2-25. Specifically, Dr. Hayes was asked to determine whether Ms. Bechtel's description of the fall could have caused the injuries A.F. sustained. RP 877:3-7. Dr. Hayes reviewed the police file, witness statements, medical examiner report, autopsy report, expert reports, A.F.'s medical records and the CD of Ms. Bechtel's 911 call. RP 878:5-23; Ex. 135. From this information, Dr. Hayes prepared a PowerPoint presentation of his interpretation of the mechanics of how A.F.'s fall occurred. RP 880:8-882:2. Ex. 145. Dr. Hayes opined, based upon his analysis, that Ms. Bechtel's description of the events did not produce the skull fracture or the inter-cranial injuries A.F. sustained. RP 939:1-941:16.

Dr. Colin Daly, the defense's biomechanic expert, testified and was critical of Dr. Hayes' opinion because he relied on population statistics and

studies to determine the probability of an event occurring. The inherent problem with Dr. Hayes' case specific reconstruction analysis, such as A.F.'s, is that such analysis cannot account for individual variability. Therefore, it is not appropriate to use sample statistics, which Dr. Hayes used, to determine what will occur in an individual case. RP 1396:4-11; 1407:12-25; 1408:1-21.

Dr. Daly's testimony was supported by that of Dr. Stephen Glass, a noted pediatric neurologist, who testified that a large variability exists among children on what injuries children might sustain from a fall. RP 776:3-9; 1502:3-9.

Dr. Glass testified that children have suffered complex skull fractures from short distance falls, RP 1507:12-15, and contrary to Dr. Hayes' opinion, it is impossible to rule out a short distance fall as the cause of A.F.'s skull fracture and resulting injuries. RP 1508:1-3. Dr. Glass noted that because of the different aged injuries in A.F.'s brain, her fatal injury was likely a cumulative event, resulting from multiple traumas. RP 1509:1-5. Dr. Glass' opinion, based upon his review of the evidence, was that A.F. died as a result of second impact syndrome. RP 1525:5-1528:15. Second Impact Syndrome results when you have a new brain injury superimposed over an older injury, which interferes with the brain's ability to autoregulate, which would account for the massive brain swelling that ultimately led to A.F.'s death. *Id.* RP 1525:5-1528:15.

IV. ARGUMENT

A. *The Trial Court Erred When it Allowed the State's Injury Biomechanic Expert to Testify.*

As this court is aware, witnesses are to state facts and not to express inferences or opinions, *State v. Wigley*, 5 Wn.App. 465, 466, 488 P.2d 766

(1971), because it is uniquely the function of the jury ... to draw reasonable inferences from the evidence. Gazijo v. Nicholas Jern Co., 12 Wn.App. 538, 541, 530 P.2d 682, aff'd, 86 Wn.2d 215 (1975).

Importantly, the admissibility of expert testimony is governed by ER 702:

If scientific, technical, or other specialized knowledge will assist the trier of fact to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise.

Three prongs dictate the application of ER 702: (1)

the witness must be qualified as an expert; (2) the opinion must be based on an explanatory theory generally accepted in the scientific community, and (3) the expert testimony must be helpful to the fact finder. State v. Black, 109 Wn.2d 336, 341, 745 P.2d 12 (1987); State v. Allery, 101 Wn.2d 591, 596, 682 P.2d 312 (1984).

Although the appellant conceded that Dr. Hayes was a qualified biomechanics expert, Ms. Bechtel urges that it was error to allow Dr. Hayes to testify. No evidence exists that an expert can reliably determine the amount of force a dog generates when it springs from a couch and strikes a 3 year old girl, which causes her to fall and strike her head on the ground, and from that fall determine what injuries a particular child will suffer. Additionally, no evidence exists that such opinion is based on an explanatory theory generally accepted in the scientific community. Further, Dr. Hayes' testimony did not help the jury understand the facts and evidence in this case as his opinion was based on speculation and was based on an inadequate foundation.

1. *No Evidence Exists that Dr. Hayes' Opinions on Causation are Generally Accepted in the Scientific Community.*

The second prong of ER 702 involves the so-called Frye test. Frye v. United States, 293 F. 1013 (D.C. Cir. 1923). The question for the court is

whether the scientific principle from which deductions are made is sufficiently established to have gained general acceptance in the scientific community. State v. Canaday, 90 Wn.2d 808, 812, 505 P.2d 1185 (1978). In determining whether a certain principle has gained general acceptance in the scientific community, the court may turn to literature such as law review articles, journals, and case law. State v. Cauthron, 120 Wn.2d 879, 888, 846 P.2d 502 (1993).

The court in Cauthron stated:

Under Frye, a court is to determine if the evidence in question has a valid, scientific basis. Because judges do not have the expertise required to decide whether a challenged scientific theory is correct, we defer this judgment to scientists. This inquiry turns on the level of recognition accorded to the scientific principle involved -- we look for general acceptance in the appropriate scientific community. See Jones v. United States, 548 A.2d 35, 42 (D.C.1988). If there is a significant dispute between qualified experts as to the validity of scientific evidence, it may not be admitted.

120 Wn.2d at 887. (emphasis added).

As set forth previously, the state hired Dr. Hayes to determine whether “A.F.’s fatal head injuries were caused by an accidental fall from the couch on April 30, 2008 as described by caretaker Leanne Bechtel.” The trial court allowed Dr. Hayes to testify that A.F.’s head injuries were not caused by the accidental fall. Basically, Dr. Hayes was allowed to comment on the credibility of Ms. Bechtel’s statement, i.e., she lied, which testimony is forbidden. See State v. Black, *infra*, *citing* State v. Garrison, 71 Wn.2d 312, 315, 427 P.2d 1012 (1967) (no witness may testify to his opinion as to the guilt of a defendant, whether by direct statement or inference.). State v. Black, 109 Wn.2d 336, 349, 745 P.2d 12

(1987) (state may not introduce expert testimony which purports to scientifically prove that an alleged rape victim is suffering from rape trauma syndrome).

In addition to commenting on Ms. Bechtel's credibility, the initial problem with Dr. Hayes' testimony surrounds the evidence he subjectively considered as "facts" to support his opinion. Dr. Hayes reviewed the discovery, including police reports and medical records, 911 call, Ms. Bechtel's statements to Lakewood detectives on April 30, 2008, and medical expert reports.

In Ms. Bechtel's statement, she stated that A.F. was standing on a couch leaning forward with her hands on the back of the couch as she was looking into a mirror. She said she called out to A.F. and asked her if she was ready for her bath and that she heard Dozer, her dog, running around in the living room. Ms. Bechtel stated that Dozer jumped on the couch between A.F. and the back of the couch and knocked her on the floor. When Detective Eggleston asked Ms. Bechtel whether she actually saw the dog get in between A.F. and the back of the couch, he reported her statement as follows: "She said that she didn't see it, but it must have happened that way . . . how else could A. F. been knocked back onto the floor." RP 834:18-23. Detective Eggleston asked Ms. Bechtel if she was able to see Dozer knock A.F. off the couch from her location in the bathroom. Ms. Bechtel stated that she saw something "out of the corner of her eye". RP 836:4. She said all that she saw "out of the corner of her eye . . . was something falling back" . . . "flying across the room." RP 835:10-13. Ms. Bechtel then stated that she saw A.F. hit the back of her head on the floor. RP 836:8-11.

From Ms. Bechtel's statement, and for purposes of rendering his "expert" opinion, Dr. Hayes determined "facts" based upon this discovery. In particular, he sets forth, as fact for his analysis, the average trotting speed of a dog comparable in size to Dozer, A.F.'s exact location while she's on the couch, and A.F.'s exact location where she came to rest on the floor. Ms. Bechtel, however, never provided any of this information and Dr. Hayes' "facts" are highly speculative. A.F.'s resting location was approximated by responding medical personnel as no measurements were taken of her exact location on the ground, and Dozer's speed at the time of the impact was unknown. RP 218:15-20. Rather, Dr. Hayes relies on this general information for his opinion and reconstruction.

As this court is aware, an expert's opinion must have a proper foundation and be based on facts. Walker v. State, 121 Wn.2d 214, 218, 848 P.2d 721 (1993). Expert testimony that lacks an adequate factual basis may not be admitted at trial. Miller v. Likins, 109 Wn.App. 140 (2001) ("... when ruling on somewhat speculative testimony, the court should keep in mind the danger that the jury may be overly impressed with a witness possessing the aura of an expert"). A court that admits expert testimony unsupported by an adequate foundation abuses its discretion. State v. Phillips, 123 Wn.App. 761, 756 (2004).

In Dr. Hayes' report dated July 17, 2012, he states that a reconstruction can be used to "reliably determine whether an accidental fall caused the injuries, as long as that reconstruction is governed by the law of physics, comports with the facts of the case, and produces the position of rest and the injuries actually sustained." CP 59; Ex. 102, pg. 2.

Significantly, what is missing are important “facts” of the case. As set forth above, Ms. Bechtel provided general information about the event. She did not provide A.F.’s specific location at the time of her impact with the dog or the dog’s speed when it jumped on the couch. Ms. Bechtel described A.F.’s before and after locations in general terms.

Additionally, Dr. Hayes’ opinion uses population statistics and applied such statistics to a specific event, with full knowledge that variability exists between individuals. RP 948:24-949:22. From these population statistics Dr. Hayes predicts the likelihood of an event occurring. RP 951:11-952:14.

Dr. Hayes referenced various research articles to support his position. Ex 102, pgs. 8-9. Significantly, however, many of the articles he relied upon were inconsistent with the opinion that he rendered at trial. In particular, the following was indicative of the fallacy of Dr. Hayes’ testimony with respect to injuries sustained from short distance falls:

Falls are extremely common, the severity not necessarily being directly related to the distance that the person falls. Many people die after falling from a standing position, yet others sometimes survive a fall of many metres.

...

Death can follow from a head injury, especially onto the back of the head.

...

The vexed question of head injury from falls in children is discussed in Chapter 22, but here it may be stated that, although fatal head injury from a fall usually requires a drop of a number of feet, there are well-authenticated instances of skull fractures and brain damage from trivial falls, including some medically witnessed falls from tables and settees.

RP 957:20-960:25. See Exhibit “A”. *Head & Spinal Injuries*, Chap. 5, pg. 180;

by P. Saukko.

Even though the articles Dr. Hayes referenced discuss variability in individuals, Dr. Hayes ignores such cautionary commentary and asserts such variables can be mathematically analyzed. RP 959:10-960:25.

Additionally, Dr. Hayes used a process called “scaling” which attempts to use data gleaned from experiments on a human skull and scale it down to approximate the mechanical properties associated with an infant’s skull.

Significantly, however, major limitations exist with this process, much of which Dr. Hayes simply ignores.

Despite this increase in understanding, the mechanical properties of the pediatric head are all but unknown. As an example, recent helmet standards for children have been forced to use adult criteria owing to a lack of pediatric tolerance data (Myers 1997).

One factor that makes pediatric head injury unique is that children are not miniature adults. Children have bodies with different geometric and mass proportions as compared to adults.

...

Along with the lack of data regarding the mechanical properties of the pediatric head, little documentation of detailed pediatric anthropometry exists. Data exist on the anatomical dimensions of children at various ages but no studies have investigated the location of the head center of gravity or quantified the head moments of inertia (Schneider et. al. 1986).

...

Currently, the biomechanical response of the human infant head is unknown. This paper reports the compressive and impact response using infant cadaveric specimens and derives lumped parameter estimates of infant head structural properties. We hypothesize that the properties of the human pediatric skull differ from the human adult skull and exhibit viscoelastic structural properties. The compression and impact response of a 6-month old anthropomorphic test

dummy were measured and compared to the infant cadaver results.

RP 961:1-962:7. See Exhibit “B”. *Mechanical Properties & Anthropometry of the Human Infant Head*, (2004); pgs. 1-2; by Michael Prange, Jason Luck, Alan Dibb, Chris Van Ee, Roger Nightingale & Barry Myers.

Dr. Hayes simply ignores such literature that says mechanical properties of the pediatric skull are unknown, because such unknowns don’t support his opinion or “facts” he relies upon to support his opinion. RP 962:3-9. When Dr. Hayes was presented with the limitations associated with the use of cadaver tests to infer human tolerance, which he equates as being “the gold standard for understanding the injury threshold for the human body, he simply ignores the literature. RP 962:10-14.

Prasad and Mertz acknowledge that many serious doubts are associated with inferring human tolerances from cadaver test data, but that these data are the best available for this purpose.

See Exhibit “C”. *Head Impact Reconstruction – HIC – Validation & Pedestrian Injury Risk* (1993), pg. 001757; by Thomas F. MacLaughlin, John F. Wiechel and Dennis A. Guenther.

The head injury criteria (HIC) that Dr. Hayes relies upon for his opinion was developed to predict head injuries in motor vehicle accidents, not to predict the head injuries a three year old might suffer when the child’s head strikes a floor. RP 1404:12-1405:15. Further, no article or research exists that would allow an expert to accurately determine the injuries a child might sustain under such situation.

Dr. Hayes simply ignores the literature when he renders his opinion in Ms. Bechtel's case because the literature he cites simply does not allow such analysis to be made with the scientific certainty Dr. Hayes professes.

Although efforts have been advanced using mathematical and other physical models to simulate brain injury mechanisms, to date no widely accepted specific tolerance criteria exist as a function of age.

...
These results underscore the need to experimentally obtain the mechanical properties of the various components of the human head as a function of age. In addition, although preliminary in nature, these analyses demonstrate the limitations of using simple geometric scaling for predicting head injury in the pediatric population.

See Exhibit "D". *Pediatric Biomechanics*, Chap. 21, pg. 576; by Narayan Yoganandan, Srirangam Kumaresan, Frank A. Pintar, and Thomas A. Gennarelli (emphasis added).

Compared to the adult model, the same level of sophistication has not been achieved in the pediatric models. This is because data on the material properties of the pediatric human skull, suture, and brain tissues are sparse. Furthermore, unlike the neck structure, finite element modeling of the head is still very much in its infancy.

See Exhibit "D". *Pediatric Biomechanics*, Chap. 21, pg. 567; by Narayan Yoganandan, Srirangam Kumaresan, Frank A. Pintar, and Thomas A. Gennarelli.
Id.

Simply stated, Dr. Hayes professed as gospel that which is not supported by the literature he cites as authoritative.

Finally, the following cautionary statements were pointed out to Dr. Hayes surrounding his prediction of probable injuries and inferences to a specific child's injuries.

Much of what is understood as potential for injury is based in what has been observed clinically. This knowledge base is critical for decision making but has inherent and important limitations.

Experimental studies investigating the influence of environmental factors, such as height of fall and surface type on injury potential, add important information, but also have inherent limitations. Important trends and predictions of probable injury can be studied but inference to a specific child's injuries is difficult because of unaccounted for contributing factors of injury risk. Such factors include muscle contraction, protective reflexes, and specific tissue response to trauma forces. Additional biomechanical research is needed to bridge the gap between clinical observations and experimental predictions.

See Exhibit "E". *Injury Biomechanics for Aiding in the Diagnosis of Abusive Head Trauma*, by Mary Clyde Pierce, MD., Gina E. Bertocci, Ph.D., PE, Rachel Berger, MD, MPH, Ev Vogeley, MD, JD; Neurosurgery Clinics of North America, 13 pg. 165 (2002) (emphasis added); CP 65; RP 963:13-964:6.

Perhaps most telling is that Dr. Hayes, without reservation, renders his opinion regarding pediatric biomechanics when peers in the field refuse to do so:

Human pediatric structures (e.g., vertebral column, brain, and skull) mature with age. The anatomic literature provides information on structural development. However, the biomechanical literature, as indicated, is still in its infancy particularly with regard to tolerance issues governing this developing population.

See Exhibit "D". *Pediatric Biomechanics*, Chap. 21, pg. 550; by Narayan Yoganandan, Srirangam Kumaresan, Frank A. Pintar, and Thomas A. Gennarelli.

With respect to determining the dog's speed at impact, Dr. Hayes substituted as "fact" the average trotting speed of a dog comparable to Dozer's size from a laboratory study that sought to determine the oxygen consumption of

a variety of animals, including dogs. RP 979:5-18. The dog's speed was controlled as the dogs were on a treadmill and their speed was then measured. Id. Again, Dr. Hayes attempts to equate an apple with an orange.

Much of the scientific data Dr. Hayes relied upon derived from studies designed for highway and automotive safety. See Ex. 102, 104. Nowhere within the research cited does it suggest that such studies should, or could, be used for purposes of an analysis in a case such as this.

As such, the trial court erred by not conducting a Frye hearing as Dr. Hayes' opinion is neither generally recognized nor accepted in the scientific community as it relates to injury analysis in a case such as this.

2. *Dr. Hayes' Testimony Was Not Helpful to the Trier of Fact as His Testimony was not Reliable as it was Speculative.*

The third prong under ER 702 is whether the testimony is relevant, that is, whether it will be helpful to the trier of fact. State v. Black, 109 Wn.2d 336, 745 P.2d 12 (1987); State v. Allery, 101 Wn.2d 591, 682 P.2d 312 (1984). As discussed above, Dr. Hayes testified about his analysis of the forces involved through his interpretation and inclusion of facts.

Recently, the Court of Appeals affirmed the exclusion of a biomechanical expert, comparable to Dr. Hayes, where the biomechanical expert was prepared to testify that a plaintiff was not injured in the manner in which she claimed. See Stedman v. Stacey Cooper, 172 Wn.App. 9, 292 P.3d 764 (2012). In Stedman, the defense sought to introduce the testimony of Allen Tencer, Ph.D. to testify about the severity of the force involved in a car accident. The trial court granted plaintiff's motion in limine to exclude the testimony.

On appeal, the Court of Appeals affirmed the trial court's exclusion of Dr. Tencer. The Court noted that, "to be admissible, expert witness' testimony must be relevant and helpful to the trier of fact." Stedman, 172 Wn.App. at 16. "Conclusory or speculative expert opinions lacking an adequate foundation will not be admitted." Id. "When ruling on somewhat speculative testimony, the court should keep in mind the danger that the jury may be overly impressed with a witness possessing the aura of an expert." Id.

In affirming the trial court's decision, the Court of Appeals considered decisions from other states that had concluded that biomechanical testimony was not helpful to the jury where it was based on information gathered in human volunteer crash testing. In one case the court held as follows: "the expert's testimony is speculative, is founded upon assumptions lacking a sufficient factual basis, relies upon dissimilar tests, and contains too many disregarded variables. Consequently, we hold that the testimony is unreliable as a matter of law, and, therefore, the trial court erred in admitting it." Id. at 19 (*citing* Tittsworth v. Robinson, 252 VA 151, 475 SE 2d 261, 263-64 (1996).)

Additionally, the Stedman court cited a Colorado Court of Appeals decision whereupon the court reasoned that "tests used to ascertain safety for the purposes of doing a cost benefit analysis with regard to the expense of designing the seat of a car were not applicable to prove that a particular person was unlikely to be injured in any specific accident." Id. (*citing* Schultz v. Wells, 13 P.3d 846, 851 (Colo. App. 2000).

Importantly, the Stedman court was concerned that Dr. Tencer wanted to testify that the plaintiff could not have been injured in the accident because the force of the impact was too small. Tencer's conclusion was that the forces generated by the impact were not sufficient to cause the type of injury the plaintiff was claiming. Id. at 6. Upon considering the trial court's reasons for excluding the evidence as well as decisions from other jurisdictions, the appellate court held that excluding Tencer's testimony was not an abuse of discretion.

In comparable cases, the Washington State Supreme Court has reviewed similar probability based calculations and rejected a reduction in the amount awarded for occupationally caused injuries – hearing loss. In Boeing Co. v. Heidy, 147 Wn.2d 78, 51 P.3d 793 (2002), Boeing attempted to use a "median-based" allocation method, in effect using data from hearing loss studies to find a "norm" for average hearing loss at a given age. This threshold of hearing loss was then applied by Boeing to Mr. Heidy as the percentage of his hearing loss attributable to age (and thus not included in his industrial insurance disability award).

In language directly applicable to the issues involved in Ms. Bechtel's case, the Court held:

Statistical studies showing tendencies within given age groups do not help triers of fact determine the actual extent of workers' individual work-related diseases.

147 Wn.2d at 85.

Boeing v. Heidy demonstrates the Court's rejection of the use of averages and general population tendencies to speculate about what occurred to

a specific individual in a specific event. Dr. Hayes' opinions employ these methodologies and studies and are also necessarily flawed and should have been ruled inadmissible as a result.

Here, Dr. Hayes' testimony is comparable to Dr. Tencer's testimony. Dr. Hayes opined that the amount of force generated by the dog, considering the laws of physics, and based upon his recitation of the facts giving rise to A.F.'s "fall" from the couch, could not have been sufficient to cause the traumatic injury she suffered. Further, much of the scientific evidence Dr. Hayes relied on for purposes of his opinion was from traffic safety studies, which the Stedman court criticized. In essence, Dr. Hayes' opinion is that because there is a low probability of something occurring that has already occurred, A.F. could not have been injured in the manner as described by Ms. Bechtel. Based upon the holding of Stedman, the other cases cited, and the references Dr. Hayes cited to but conveniently ignored, the trial court erred when it allowed Dr. Hayes' to testify.

3. *The Court Erred When it Allowed Dr. Hayes' Reenactment of the Incident.*

As part of Dr. Hayes' opinion, he prepared a slide show presentation seeking to recreate the events he interpreted as described by Ms. Bechtel. Ex. 145. As set forth previously, Dr. Hayes included "his" facts to support his opinion. The exact manner in which the dog struck A.F., how she moved after being struck, and how she landed are guesses by Dr. Hayes and not based upon any direct testimony nor anything that could be recreated. As such, the accident reenactment is based upon his guesses, i.e., speculation. Given the inherent

unreliability of the information he used to create his reenactment, the trial court erred by allowing his recreation into evidence.

“Demonstrative evidence may be admissible if the experiment was conducted under conditions reasonable similar to conditions existing at the actual event. Whether the similarity is sufficient is for the trial court’s discretion.” State v. Rogers, 70 Wn.App. 626, 633-34, 855 P.2d 284 (1993).

Support for excluding this testimony is set forth in State v. Stockmyer, 83 Wn.App. 77, 920 P.2d 1201 (1996). In Stockmyer, the Court of Appeals upheld the trial court’s decision excluding a videotaped re-enactment of a shooting. The Court of Appeals noted that the trial court’s “justifiable concerns over the videotaped reenactments factual inaccuracies and potential prejudicial effect” were well within the trial court’s discretion to exclude the evidence. Stockmyer 83 Wn.App. at 85.

As stated in Stockmyer, factual inaccuracies and prejudices are touchstones as to whether such re-enactment evidence is admissible. Given that the reenactment is Dr. Hayes’ interpretation of what occurred, the probative value of his interpretation is nominal at best, and the prejudice to the defendant is significant. Respectfully, the trial court erred by admitting this evidence.

B. The Trial Court Erred When it Denied Appellant’s Motion for a New Trial Based on Juror Misconduct.

“The Sixth and Fourteenth Amendments to the United States Constitution and Washington Constitution Art. 1 § 22 guarantee a defendant the right to a fair and impartial jury.” State v. Koontz, 145 Wn.2d. 650, 653, 41 P.3d 475 (2002). “The right to a fair and impartial jury is protected by the procedures contained in chapter

4.44 RCW and by court rule.” Id. “These protections govern not only the information that may be conveyed to a jury, but also the manner in which the information may be delivered.” Id. “A jury may have access to an audio taped exhibit during deliberations if, in the discretion of the court, the exhibit bears directly on the charge and is not unduly prejudicial’.” State v. Castellanos, 82 Wn.App. 204, 205, 916 P.2d 893 (1996).

In Koontz, the Supreme Court reversed a Court of Appeals decision upholding the trial court’s order that allowed the jury to replay video-taped witness testimony. The Koontz court noted that the trial court failed to apply sufficient protections against undue emphasis in the manner in which the video tape was to be replayed. Koontz, 145 Wn.2d at 657.

After the jury returned its verdict, counsel for appellant and for the State learned about an issue wherein the jury separated during its deliberation. While some of the jurors were listening to the 911 tape inside the courtroom, other jurors were in the jury room. CP 408-411. The jurors were listening to the admitted 911 tape and reviewing the transcript of the 911 tape. CP 408-09; 410-11; 466-68.

Here, the issue is not that the trial court allowed the 911 tape to be replayed, but, rather, the manner in which the jury listened to the tape. Although counsel for both the State and the defense agreed that the jury should have access to the 911 call if requested, the parties were also informed by the court that the 911 call would be played in the courtroom to all of the jurors and that the jurors would remain together. Although this occurred for a portion of the time, it is clear that the jury separated while considering this evidence, which conduct violated the court’s instructions as to

how this evidence would be reviewed. Under such circumstances, this is tantamount to the jury separating during deliberations and considering evidence that was not considered by the entire jury.

RCW 4.44.300 in part, states as follows:

Unless the members of a deliberating jury are allowed to separate, they must be kept together in a room provided for them or some other convenient place under the charge of one or more officers, until they agree upon their verdict, or are discharged by the court.

Here, while some of the jurors were considering the 911 evidence, the balance of the jurors were in the jury room. What this allowed to occur is an over emphasis of one piece of evidence by some of the jurors, i.e., the 911 call, to the exclusion of other evidence because only some of the jurors were in the courtroom considering the 911 call. The above situation is comparable to Koontz wherein the Supreme Court held that replaying video tape testimony, without appropriate safeguards, violated Koontz's right to a fair trial, thus mandating reversal. In Ms. Bechtel's case, although appropriate safeguards were established, they were not followed. As such, and based upon the aforementioned, Ms. Bechtel respectfully urges this court to grant her a new trial based on juror misconduct.

V. **CONCLUSION**

Perhaps nothing is more painful than the loss of a child. Understandably, when the specter of criminal wrongdoing exists, attorneys and judges do all within their power to make sure that justice prevails. Here, however, Ms. Bechtel was prejudiced by Dr. Hayes' testimony that failed to satisfy the Frye test and because of jury misconduct. As such, and based upon the aforementioned, Ms. Bechtel urges this Court to reverse her conviction and grant her a new trial

RESPECTFULLY SUBMITTED this 28th day of August, 2013.

HESTER LAW GROUP, INC., P.S.
Attorneys for Appellant



BRETT A. PURTZER
WSB #17283

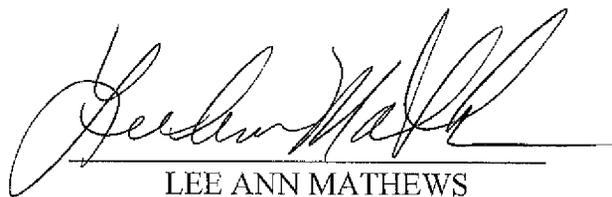
CERTIFICATE OF SERVICE

Lee Ann Mathews, hereby certifies under penalty of perjury under the laws of the State of Washington, that on the day set out below, I delivered true and correct copies of the opening brief of appellant to which this certificate is attached, by United States Mail or ABC-Legal Messengers, Inc., to the following:

Kathleen Proctor
Deputy Prosecuting Attorney
930 Tacoma Avenue South, #946
Tacoma, WA 98402

Leanne M. Bechtel
DOC #888909
Washington Corrections Center for Women
9601 Bujacich Road NW
Gig Harbor, WA 98332-8300

Signed at Tacoma, Washington, this 28th day of August, 2013.


LEE ANN MATHEWS

HESTER LAW OFFICES

August 28, 2013 - 4:39 PM

Transmittal Letter

Document Uploaded: 443376-Appellant's Brief.pdf

Case Name: State v. Bechtel

Court of Appeals Case Number: 44337-6

Is this a Personal Restraint Petition? Yes No

The document being Filed is:

Designation of Clerk's Papers Supplemental Designation of Clerk's Papers

Statement of Arrangements

Motion: ____

Answer/Reply to Motion: ____

Brief: Appellant's

Statement of Additional Authorities

Cost Bill

Objection to Cost Bill

Affidavit

Letter

Copy of Verbatim Report of Proceedings - No. of Volumes: ____

Hearing Date(s): _____

Personal Restraint Petition (PRP)

Response to Personal Restraint Petition

Reply to Response to Personal Restraint Petition

Petition for Review (PRV)

Other: _____

Comments:

No Comments were entered.

Sender Name: Leeann Mathews - Email: leeann@hesterlawgroup.com

A copy of this document has been emailed to the following addresses:

pcpatcecf@co.pierce.wa.us

CHAPTER 5

Head and spinal injuries

■ Injury to the scalp	174	■ Subarachnoid haemorrhage and alcohol	199
■ Facial injuries	178	■ The rapidity of death in subarachnoid haemorrhage	199
■ Falls	180	■ Rotational trauma to the head and upper neck: basillovertebral artery injury	200
■ Fractures of the skull	181	■ Head injuries in boxers	204
■ Forensic anatomy of the brain membranes	189	■ Cerebral injuries	204
■ Extradural haemorrhage	190	■ Histological diagnosis of early cerebral hypoxia	212
■ Heat haematoma	192	■ Secondary brainstem lesions	213
■ Subdural haemorrhage	192	■ Spinal injuries	214
■ Subarachnoid haemorrhage	196	■ References and further reading	216
■ Forensic implications of brain membrane haemorrhage	198		
■ Ruptured berry aneurysm and trauma	198		

Of all regional injuries, those of the head and neck are the most common and most important in forensic practice. Adelson (1974) gives these sound reasons for this dominance of head injuries:

- The head is the target of choice in the great majority of assaults involving blunt trauma.
- When the victim is pushed or knocked to the ground, he often strikes his head.
- The brain and its coverings are vulnerable to degrees of blunt trauma that would rarely be lethal if applied to other areas.

A sound practical understanding of the neuropathology of trauma is more essential to the forensic pathologist than any other aspect of his subject, as head injuries provide the major contribution to death in assaults, falls and transportation accidents.

INJURY TO THE SCALP

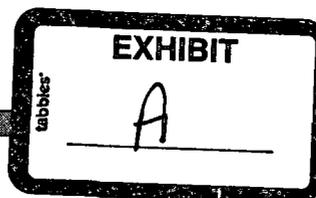
The scalp is often, though by no means invariably, damaged in trauma that causes injury to the underlying skull

and brain. The usual range of abrasions, contusions and lacerations may be inflicted, though a modifying factor is the presence of hair, which may deflect a tangential blow or partly cushion a direct impact.

When an injury is visible on the forehead, the back of the neck, the lower temple or on a bald area, the examination is no different from elsewhere on the body. In hair-covered areas, care must always be taken at autopsy to palpate the scalp in any case in which there is a possibility of injury, otherwise abrasions, swelling, bruising and even lacerations may be missed. When a lesion is found or suspected, the hair must be carefully shaved away to expose the scalp for further examination and photography.

Forensic anatomy of the scalp

Superficially, the skin carries hair follicles, sebaceous glands and sweat glands. The skin is attached to the aponeurosis (see below) by vertical strands of fibrous tissue that break up the subcutaneous layer into pockets filled with fat. The blood vessels and nerves lie in this layer, above the epicranial aponeurosis (formerly called the 'galea aponeurotica'). This is a dense sheet of fibrous tissue that lies in the deep



Cha 5
Saukko, P.

layer of the scalp over the whole cranium. It is really a flattened tendon uniting the frontal and occipital bellies of the occipitofrontalis muscle.

Deep to the aponeurosis is a thin layer of loose connective tissue that separates it from the pericranium, which is the exterior periosteum of the skull, the dura being the internal counterpart. Some veins traverse all the layers from the superficial fascia to the pericranium, and go on to penetrate the skull and communicate with the intracranial venous sinuses, thus forming a route for meningitis and sinus thrombosis from infected injuries of the scalp.

Abrasion of the scalp

Brush abrasions are less common than in other sites because of the protective effect of the hair, which also tends to prevent or blur the patterned effect of less severe impacts. Impact abrasions from a perpendicular force are imprinted as usual on to the scalp, though again the intervening hair may reduce the severity. Unless the hair is carefully removed

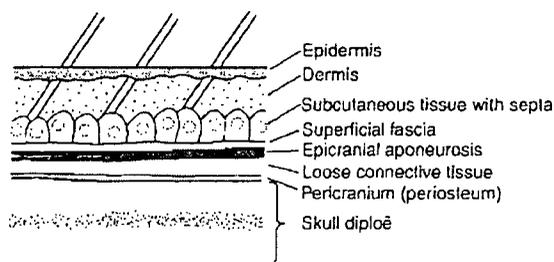


FIGURE 5.1 *Anatomy of the scalp.*

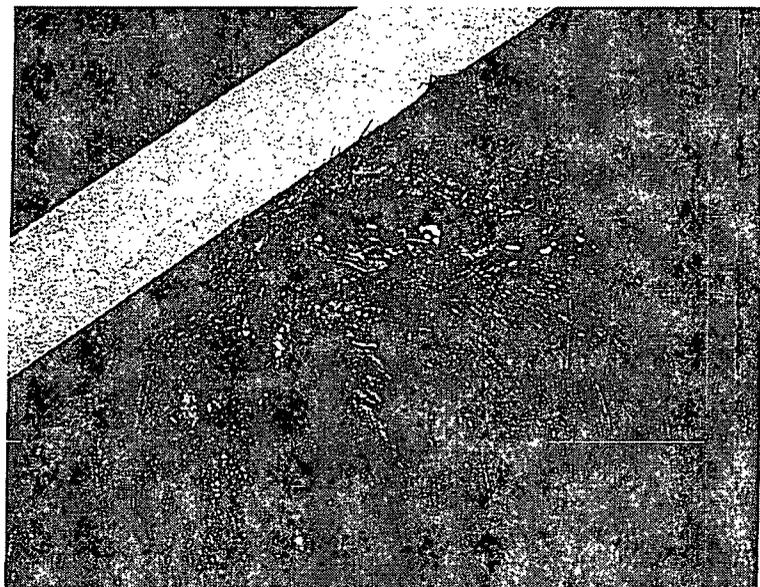


FIGURE 5.2 *A laceration of the scalp caused by a blow from an iron bar. The edges are crushed and bruised, with strands of connective tissue and hairs crossing the gap, indicating that it was not caused by a sharp-edged weapon.*

at autopsy, with a sharp scalpel or razor, and care taken not to cause artefactual cuts, lesser degrees of abrasion will inevitably be missed.

Bruising of the scalp

Bruising may be difficult to detect until the hair has been removed. Marked swelling is a common feature of extensive bruising, as the liberated blood cannot extend downward because of the rigidity of the underlying skull. However, this subsides, or at least diffuses, after death. Commonly, a severe head injury leads to a thick, swollen, indurated layer of blood beneath the scalp, which may extend over a wide area. The blood is sometimes below the aponeurosis, the tough fascial layer of the scalp, but is more often between this and the epidermis.

Blood may also be present beneath the pericranium, the periosteum that is closely applied to the outer surface of the skull. This is often seen in head injuries in infants, usually in association with skull fractures, as the source of the blood is from the fracture line itself. The close attachment of the pericranium to the suture lines in infants may sharply circumscribe the extent of the bleeding.

In addition to frank bleeding beneath the scalp, marked oedema may occur after injury and the layers of the scalp may be greatly swollen and thickened by a jelly-like infiltration of tissue fluid.

As will be discussed under 'black eye', bleeding under the scalp may be mobile, especially under gravity. Thus a bruise or haematoma under the anterior scalp may slide downwards within hours – even minutes – to appear in the orbit,



FIGURE 5.3 Lacerations of the scalp from an iron bar. The margins are bruised and the scalp tissue is extruding in places. The generally parallel direction of the five wounds indicates that the assailant probably delivered the blows in rapid succession with little change of orientation between weapon and head.



FIGURE 5.4 Stellate laceration of the scalp caused by a heavy blow with a piece of timber. The support of the underlying skull has caused the tissues to split widely. At autopsy, full clearance of the hair must be made to allow detailed examination and photography.

simulating a black eye from direct trauma. Similarly, a temporal bruise may later appear behind the ear, suggesting primary neck impact. As with bruises elsewhere, those under the scalp may be obvious immediately after infliction – or their appearance may be delayed, either during life or as a post-mortem phenomenon. They may first become evident, or much more prominent, some hours – or even a day or so – after death. This is caused either by movement of the



FIGURE 5.5 Wounds from a metal poker superficially resembling incised cuts, but having edges and tissue bridges within the wounds.

liberated blood through tissue planes or by haemolysis spreading outwards to stain the subcutaneous tissues, making it always advisable to return to examine the body a day or two following the autopsy.

The shape of an inflicting weapon or object is poorly reproduced on the scalp, again due to the padding effect of the hair. Where the scalp is free of hair, as in the upper forehead or bald areas, all traumatic lesions are similar to elsewhere on the body, with the exception that blunt impact may cause very sharply defined lacerations.

Laceration of the scalp

Lacerations of the scalp bleed profusely, and dangerous and even fatal blood loss can occur from an extensive scalp injury if it is not checked by treatment. The most gross injury is avulsion of a large area of scalp, which can be torn from the head, thereby exposing the aponeurosis or skull. This may happen if the hair becomes entangled in machinery, as was formerly not uncommon in women working in factories. A more common cause nowadays is a traffic accident, where a rotating vehicle tyre comes into contact with the head, causing a 'flaying' injury similar to that seen on limbs.

Scalp injuries may bleed profusely **even after death**, especially if the head is in a dependent position. A post-mortem injury to the head may bleed considerably if inflicted soon after death and these facts may sometimes cause confusion about the ante-mortem or post-mortem nature of the injury. There is no reliable way of resolving this difficulty.

Lacerations of the scalp may reproduce the pattern of the inflicting object, even though a random splitting is so common. Severe blows from shaped objects such as hammers or heavy tools may reproduce the profile of the weapon totally

or in part. A circular-faced hammer may punch a circle in the scalp, but more often only an arc of a circle is seen. In such cases, the position of the edge that digs in most deeply may give an indication of the angle of the blow. There may be a depressed fracture of the underlying skull of the same shape and size, though the interposition of the dense scalp may cause the skull defect to be slightly larger than the weapon. A depressed fracture in these circumstances is not inevitable, however, and one or more linear fractures may radiate from the impact site.



FIGURE 5.6 *Deep linear incised wounds due to a heavy, sharp cleaver. The depth of penetration varies, the large wound overlying extensive skull fractures. The wounds on the neck are due to light contact with the edge of the same blade.*

A major problem in scalp injuries is the differentiation between incised wounds and lacerations from blunt injury.

The scalp is the best example of a surface tissue lying over an unyielding bony support. Violent compression will crush the scalp against the underlying skull, so a blow from a blunt rod-like weapon may split the skin and underlying tissues in a sharply demarcated fashion, which may appear remarkably like a slash from a sharp instrument. Close examination, using a lens if necessary, will show that this blunt laceration has:

- bruised margins, even though this zone may be narrow
- head hairs crossing the wound, which have not been cut
- fascial strands, hair bulbs and perhaps small nerves and vessels in the depths of the wound.

Scalp injuries from falls

It is vital for the pathologist to appreciate that falls on to a flat surface, or a blow from a wide, flat object such as a plank or paving stone, may sometimes leave no external mark whatsoever on the exterior of the head, but commonly such an injury will cause a ragged split which may be linear, stellate or quite irregular.

Such injuries on the back point of the head are commonly caused by falling, especially in inebriated victims. Falls backwards against a ridge, such as a wall or pavement kerb, may cause a transverse laceration, which may be undercut and partly detached from the underlying bone so that a flap of scalp is loosened from the skull.

Falls usually injure the occipital protuberance, the forehead or the parietotemporal areas. Injuries on the vertex



FIGURE 5.7 *Sliced incised wound of the scalp, from a large knife. The wound is markedly undercut, turning a wide flap of scalp. The clean edges, with a lack of any abrasion or bruising, indicate the sharpness of the weapon.*

should always raise the suspicion of assault, as it is unusual to fall upon the top of the head, even from a considerable height. Occasionally, a fall backwards that just happens to reach a vertical surface, such as a wall or piece of furniture, can cause damage to the top of the head, but there is then usually an obvious grazing component to the lesion.

FACIAL INJURIES

Damage to the face is common, but unless gross, with skeleral damage, is rarely fatal in itself unless it leads to bleeding into the air passages. It is often ancillary to fatal cranial damage, or it may be the route by which severe trauma reaches the brain.

The usual range of injuries may be present externally, but all degrees of underlying damage may also occur in the facial skeleton. Because of the complex contours of the face, the various prominences of chin, nose, cheekbones, eyebrows, ears and lips may intercept impacts, with consequent characteristic damage. The eyebrow is particularly vulnerable, being exposed during falls and blows. A blunt impact on the brow often splits the skin and may cause an underlying frontal fracture that can involve the orbital margin.

The distal part of the nose is flexible and often escapes serious damage, though abrasion is common. The bony bridge of the nose is often fractured, which may be detected by movement and crepitus during external palpation – and by dissection at autopsy. Bleeding in the nose is more important than structural damage, as profuse haemorrhage in an unconscious victim may pass back through the posterior nares into the throat and cause fatal airway obstruction.

The maxillae and mandible may be fractured by direct blows and again cause dangerous intra-oral bleeding from associated soft-tissue damage. A heavy blow or kick to one side of the jaw can cause ipsilateral, bilateral or even contralateral fractures. Gross injury to the face, seen in kicking and some transport accidents, may actually detach the facial skeleton from the base of the skull. The lower part of the maxilla, carrying the palate and upper teeth, may be completely separated from the rest of the skull. At autopsy, the best view of the facial skeleton may be gained by dissecting the whole facial skin upwards from the neck incision and reflecting it as far as the orbits, if necessary. Good restoration can be achieved as long as the skin is not penetrated by the knife.

Injuries to the mouth and lips are very common in 'bearing-up' incidents, including child abuse. The lips may be bruised or lacerated, much of the damage arising from compression of the lips against the teeth or bony gums. Lacerations on the gingival aspect of the lips may often be exactly matched with the edges of teeth and, as discussed in



FIGURE 5.8 Facial injuries caused by kicking. The upper lip is split and the maxilla fractured on that side. The face and orbital region is grossly swollen and bruised. Death was caused by blockage of the air passages by blood.

Chapter 22, rupture of the frenulum inside the upper lip of a child is virtually pathognomonic of a sideswipe across the mouth, if damage from clumsy and forceful attempts at introducing a feeding bottle, dummy or airway can be excluded.

Kicking

Kicking of the face is regrettably common and again the prominences suffer most. Bruising, laceration and fractures may result from kicking under the side of the jaw: similar lesions occur on the maxillary area and the eyebrows. Patterned abrasions from boot soles may be seen or crescentic marks from roecaps. Brush abrasions from glancing kicks may be present on the cheeks or forehead as the sole of the shoe scrapes across the skin. It is uncommon to suffer a 'pure' black eye from a kick without other facial injuries, such as scuffed abrasion on the cheekbones, or marks on the brows or bridge of the nose. Teeth may be loosened, broken or detached by both kicks and heavy punches and black eyes and fractures of the nose are common. A kick on the side of the jaw may cause bilateral jaw fractures or even a single contralateral fracture.

It may be difficult or impossible to differentiate in every case, between injuries (especially to the face) caused by kicks



FIGURE 5.9 Black eye from a direct impact into the orbit from a punch. This is the third mechanism of production of a periorbital haematoma, the others being a fracture of the anterior base of the skull and a frontal scalp wound.



FIGURE 5.10 Black eye as a result of gravitational seepage of blood downwards from a forehead injury. The woman was struck with a rock on the frontal area and eyebrow, blood then descending during the few hours of coma before death occurred.

and those from blows from a blunt object. Toecap marks are not all that common, especially since the more flexible rubber 'trainer' shoes have become almost universal footwear.

In stamping, there is the chance that the sole pattern may leave an imprint, but a swing from a toe may leave a non-specific abrasion, bruise or laceration. It may be that the severity of the injury, including underlying bone damage, may be a better indication of a kick than the shape of the injuries, as the force delivered by a swinging foot at the end of a muscular leg is greater than that from a fist.

Black eyes

The usual periorbital haematoma or 'black eye' is usually caused by a direct punch or kick into the eye-socket, but the pathologist must always consider the several alternative explanations. A black eye may be the result of:

- ❑ direct violence, which may or may not be associated with abrasion or laceration on the upper cheek, eyebrow, nose or other part of the face
- ❑ gravitational seepage of blood beneath the scalp from a bruise or laceration on or above the eyebrow. Survival

and at least a partially upright posture of the head must have been maintained for at least some minutes, usually longer, between the time of injury and death. When the scalp lesion is high up on the frontal region, this time will probably be measured in hours

- ❑ percolation of blood into the orbit from a fracture of the anterior fossa of the skull. This is often from a contrecoup injury caused by a fall on to the back of the head, leading to secondary fracture of the paper-thin bone of the orbital roof. It is invariably associated with contrecoup contusion of the frontal lobes of the brain, as described later in this chapter.

A simple fall onto the face on a flat surface does not usually cause a black eye, as the prominences of the eyebrow, cheekbone and nose prevent damage to the orbit.

Damage to the ear

The external ear often suffers from blows to the head and is an obvious target in child abuse. Bruising and laceration of



FIGURE 5.11 Bilateral black eyes caused by leakage of blood into the orbits through comminuted fractures in the floor of the anterior fossa. This homicide victim was struck on the head with a shovel and survived for some days. Brain tissue is escaping from the nostrils through basal skull fractures.

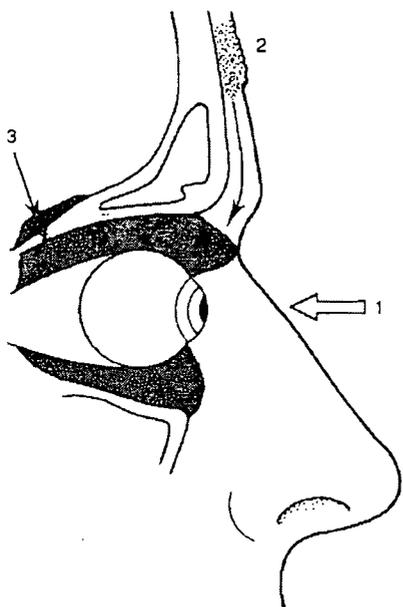


FIGURE 5.12 The production of a black eye: (1) A direct blow into the orbit. (2) An injury to the front of the scalp, draining down over the supraorbital ridge. (3) A fracture of the base of the skull (direct or contrecoup) allowing meningeal haemorrhage to escape through the orbital roof.

the pinna is obvious on examination and – in severe trauma – the root of the ear may be detached from the head, usually by a tear at the posterior margin where the ear joins the head. Where gross damage is present, especially with partial avulsion of the pinna, kicking must be considered.

The ear may be bitten and even partly detached, a fate which occasionally is suffered by the nose. In such cases the advice of a forensic odontologist may be invaluable, as teeth marks may form vital evidence.

FALLS

Falls are extremely common, the severity not necessarily being directly related to the distance that the person falls. Many people die after falling from a standing position, yet others sometimes survive a fall of many metres.

Falls from a standing position can occur if a person is drunk, from an assault, during illness (such as a fit or faint) and for many other reasons. Death can follow from a head injury, especially onto the back of the head. An occipital scalp laceration or a fracture of the skull is not necessary for cerebral damage (often frontal contrecoup) to occur. There may also be a subdural or (less often) an extradural haemorrhage, the latter more common from a fall on to the side of the head.

The vexed question of head injury from falls in children is discussed in Chapter 22, but here it may be stated that, although fatal head injury from a fall usually requires a drop of a number of feet, there are well-authenticated instances of skull fractures and brain damage from trivial falls, including some medically witnessed falls from tables and settees. The experimental work of Weber (see Chapter 22) showed that the skulls of small infants could be fractured against a variety of floor surfaces from passive falls of only 34 inches. It is thus invalid for medical witnesses to claim it cannot happen, as even one authenticated case creates a precedent. In adults, fractures have certainly occurred from falls onto very hard surfaces from only a foot or so. One such case was a drunk lying on concrete; equally drunken friends attempted to lift him but allowed his head and shoulders to fall back from about half-sitting position, causing occipital fracture.

Falls in old people very frequently cause fractures of the post-cranial skeleton – especially the neck of femur – though ribs, arms and pelvis may also suffer. Osteoporosis is the major reason for the large number of such injuries from falls. More than 47 000 fractured femora occur each year in Britain, with a 25 per cent mortality rate, mainly from subsequent pulmonary embolism or bronchopneumonia.

Falls from a height

Falls from a considerable height, usually from a building, are common in suicide and in some accidents, especially to children. Occasionally deaths from a high fall may be homicidal, again especially in children.

When a person falls or jumps from a height, the trajectory is downwards and outwards, and the distance that the body strikes the ground from the jumping point is variable. Goonetilleke (1980) has published some research on how far from the wall a body is likely to land. Much depends on whether the victim fell passively from near the wall or projected themselves outwards at the top. The body may fall whilst maintaining the same orientation to the ground, but usually turns and twists in an unpredictable manner, the amount of alteration of posture partly depending upon the height of the fall and so the time available for turning. This means that the body may strike the ground in a number of different attitudes – and may also strike some obstruction part of the way down, making interpretation of injuries difficult.

The primary impact is usually the site of the most severe injury, but this is not always the case, as it may strike two areas simultaneously, such as the head and shoulder – or it may bounce or ricochet so that two or more major impacts occur in quick succession. The amount of kinetic energy acquired during the fall has to be fully expended by the time the body comes to rest so that, if only one impact occurs, it is likely to be more damaging than a series of lesser impacts, such as a bouncing, rolling strike.

If the body falls on to the head, there is likely to be a massive fracture, often (but not always) a scalp laceration and possibly extrusion of brain. Both vault and base can fracture and sometimes the base is driven down over the cervical spine, the latter projecting into the posterior fossa. The latter injury is, however, more common with high falls onto the feet when the impact is transmitted up the spinal column and the upper vertebrae – together with a ring of bone around the foramen magnum – are intruded into the skull, causing the classic 'ring fracture' of the occipital bone. Where the fall is onto the feet, the deceleration stress can break the axial skeleton at a number of points. The legs can be broken at any point, at tibial or femoral level, often bilaterally. The femoral necks can be snapped off, the hip joints can dislocate and over-ride, or the pelvis may fracture. The latter is often through the sacroiliac joints, the upward force (or more accurately, downward force of the body on to the legs) driving the sacrum down as a wedge into the pelvis.

If the lower limbs and pelvic girdle remain intact, the transmitted force may then fracture the spine, often at mid- or upper thoracic level.

Where the fall occurs onto the side of the body, any combination of injuries can occur. Multiple rib fractures,

shoulder girdle or arm fractures, lacerations of back, buttocks or limbs and severe abdominal injuries can occur, with consequent internal lesions, such as rupture of the liver, lungs, heart or spleen.

In falls from high-rise buildings, the injuries can be extreme, as in a suicide from the twentieth floor seen by the author (BK), where the victim fell onto a fence and was completely transected at waist level.

It must also be remembered that biological and circumstantial variability allows for some remarkable escapes from falls; some persons, including children, have fallen from great heights yet have virtually walked away unscathed. Once again, it is very unwise to over-interpret the relationship between observed injuries and the likely length of the fall.

FRACTURES OF THE SKULL

Forensic anatomy

The cranium and facial bones are laid down from membrane in fetal life. The anterior fontanelle closes functionally between 9 and 26 weeks after birth, though is not tightly sealed until about 18 months. The posterior fontanelle closes between birth and 8 weeks of age. Suture lines close by interdigitation during childhood and osseous fusion occurs irregularly at variable dates during adult life.

The adult cranium consists of two parallel tables of compact bone called the 'diploë', the outer being about twice the thickness of the inner. They are separated by a central zone of soft cancellous bone, which is often misnamed the diploë. This zone is interrupted at suture lines and vanishes where the bone becomes particularly thin, especially in the floor of the skull.

The cranium varies in thickness in adults and varies from place to place, thin plates being reinforced by stronger buttresses, such as the petrous temporal, the greater wing of the sphenoid, the sagittal ridge, the occipital protuberance and the glabella. This tensile architecture of the skull has been well described and illustrated by Rowbotham (1964). The more vulnerable thin areas lie in the parietotemporal, lateral frontal and lateral occipital zones.

The average frontal and parietal thickness in a young male is between 6 and 10 mm. The thinnest area is in the temporal bone, where it may be only 4 mm, while in the occipital bone in the midline it may be 15 mm or even more. The thickness of the skull is sometimes an issue in courtroom propositions about the special vulnerability of a victim but, unless it is abnormally thin, such theorizing has little forensic relevance as it is well known that fatal brain damage can often occur with an intact skull. It has even been claimed that a thin skull is less likely to fracture,

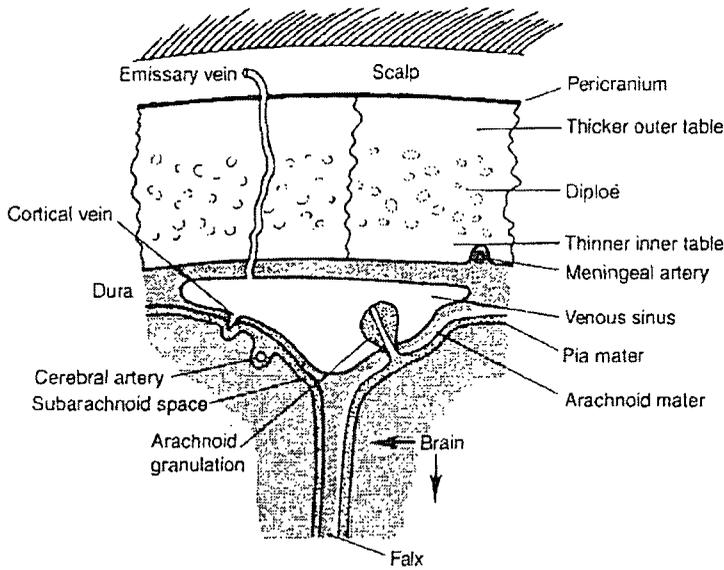


FIGURE 5.13 Forensic anatomy of skull and meninges.

as it is more flexible and can return to normal after distortion without cracking; this is certainly true of the skulls of infants. This may be irrelevant when assessing intracranial damage, however, which is the major issue in head injuries.

It is rarely the skull fracture itself that is a danger to life, but the concomitant effect of transmitted force upon the cranial contents. The presence of a skull fracture is, however, an indication of the severity of the force applied to the head and it is uncommon for a head injury that is sufficiently severe to crack the skull not to cause some intracranial effect, even if it is only transient concussion, though, once again, there are many remarkable exceptions to this generalization.

In skeletal material, fractures of the skull are often seen and the differentiation between ante-mortem injury and post-mortem damage can sometimes be difficult or even impossible in the absence of any soft tissues. Artefactual damage to a skull may be caused during recovery or exhumation, and even stones in the soil can cause erosion and even cracking of the softened, degenerate bone after long burial. Zuo and Zhu (1991) have described scanning electron microscopic details of microfractures and collagen damage, which can differentiate ante- from post-mortem injury, but these are unlikely to assist in old decayed material.

The mechanics of skull fracture

This subject has been extensively studied in living animals, isolated human heads and dried skulls. For details, the writings of Gurdjian, Webster and Lissner (1949, 1950), Weber (1984) and Rowbotham (1964) should be consulted;

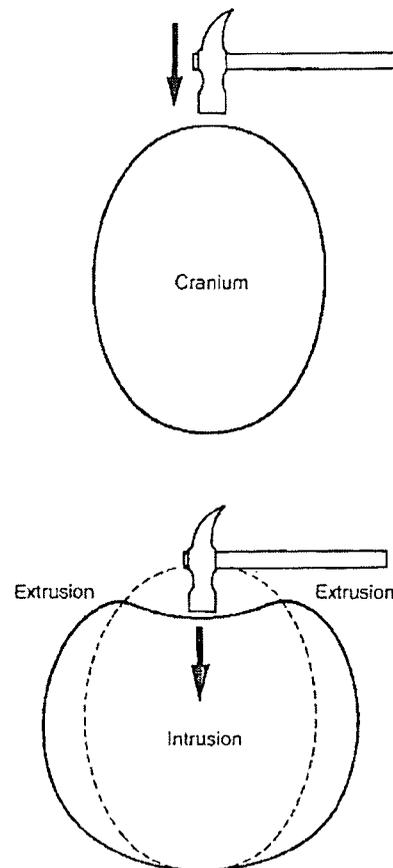


FIGURE 5.14 The 'struck hoop' illustration of impact on the skull. There is a momentary deformation, with the area of impact bending inwards and compensatory bulging elsewhere. This sets up stresses in the inner and outer tables.

a concise summary has also been provided by Shapiro *et al.* (1988). These and other workers have shown that:

■ When the skull receives a focal impact there is momentary distortion of the shape of the cranium, the extent of which may be surprisingly large, though transient. Infant skulls, which are more pliable and have flexible junctions at suture lines, may distort much more than the more rigid skulls of adults. The area under the point of impact bends inwards and, as the contents of the skull are virtually incompressible, there must consequently be a compensatory distortion or bulging of other areas – the well-known ‘struck hoop’ analogy.

Both these intruded and extruded areas can be the site of fracturing if the distortion of the bone exceeds the limits of its elasticity.

■ When the skull is deformed, compression occurs on the concavity of the curved bone and tension (tearing) forces on the convexity. If the latter exceed the elastic threshold, then fracturing takes place. Thus the inner

table will fracture where the skull is indented and the outer table will fracture at the margins of the deformed area. If the forces are great enough, a depressed comminuted fracture will occur.

■ In the more common circumstance of a wider impact from a blunt injury, the deformation of the skull is less localized but, where the force is sufficient, fractures can still occur from the same mechanism of exceeding the elastic limits. The fractures may be remote from the area of impact, following lines of structural weakness – or may extend from the area of impact, or even commence at a distance and run back to the impact site.

By using skulls coated with a brittle varnish, Gurdjian showed that stress lines developed in the cranium when it was struck and that these corresponded with the fractures that occurred with heavier impacts.

Blows in certain areas of the skull constantly give rise to fractures in specific localities – for instance, impact on the upper temporal or parietotemporal areas cause fissured fractures running obliquely downwards across the temporal area. If heavier, another fracture line tends to run obliquely contralaterally across the vault of the skull.

A heavy impact on the side or top of the head often leads to the vault fracture running into the base of the skull, usually across the floor of the middle cranial fossa along the anterior margin of the petrous temporal bone, to enter the pituitary fossa. In major injuries, this fracture line may often cross the floor of the skull completely to form a ‘hinge fracture’, separating the base of the skull into two halves. These fractures do not start at the point of impact unless there is also local

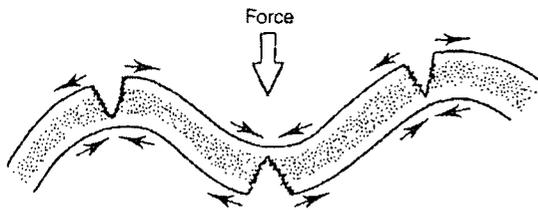


FIGURE 5.15 The skull is more susceptible to traction forces than compression, so that convexities tend to fracture during the distortion of the ‘struck hoop’.



FIGURE 5.16 Linear fracture of the posterior fossa, due to a fall on the occiput. The fracture typically crosses the thinner bone, avoiding the central buttress and ends near the foramen magnum.

depressed fracturing; they are initiated at a distance due to the compensatory deformation, but usually run back towards the impact site.

When the frontal area is struck, the usual course for a linear fracture is vertically down the forehead, turning around the orbital margin to run backwards across the floor of the anterior fossa, perhaps into the cribriform plate or air sinuses, or both. A blow or fall onto the occiput may produce a fracture that typically passes vertically or obliquely downwards just to the side of the midline of the posterior fossa, commonly reaching the foramen magnum. In addition, the contrecoup element of an occipital fall may cause fractures of the orbital plates in the anterior fossa, as a result of transmitted force through the brain itself, though the mechanism is not fully understood.

- ☒ When severe local impact causes focal and general deformation, a combination of depressed fractures and radial fracture lines may form a 'spider's-web' pattern.
- ☒ When the focal impact is severe, the depressed fracture may follow the actual shape of the impacting object, such as a hammer-head. The shape may follow only that part of the object that drives into the skull – for example, the circular head of the hammer may strike at an acute angle, so only a semicircle of bone will be punched inwards, the opposite edge sloping downwards from an irregular crack.

The deepest part of the depression will indicate where the weapon first struck; there may be 'terracing' of the margins. The author (BK) has seen a recent instance where a victim was struck with a hammer through a thin plastic bag enveloping the head. Parallel terracing cracks had opened for a fraction of a second during the blow, sufficient to trap lines of plastic into the multiple defects in the outer table. Where the impact is from a narrow edge or ring, only the outer table may be fractured, being punched into the softer centre without depression of the inner table.

- ☒ The presence of hair and scalp markedly cushions the effects of a blow, so that a far heavier impact is required to cause the same damage, compared to a bare skull. The pattern and nature of the skull fractures are, however, the same. It should be noted that the interposition of scalp and hair may slightly alter the dimensions of the skull lesion from a focal blow. For example, the padding effect of the scalp may add a few millimetres in diameter to the depressed fracture caused by a hammer, compared with the actual measured diameter of the hammerhead.
- ☒ Where two or more separate fractures occur from successive impacts and meet each other, the sequence of injuries may be determined by 'Puppe's rule', which is

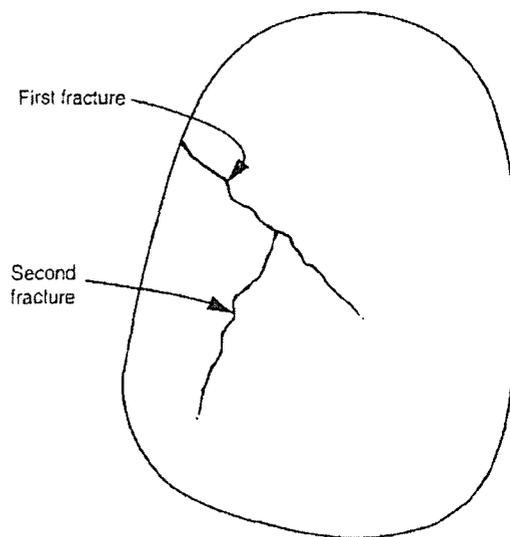


FIGURE 5.17 Diagram to illustrate Puppe's rule for the sequence of fractures. The course of a later fracture will be interrupted by an earlier pre-existing fracture line.

really eponymous common sense. The later fracture will terminate at (that is, not cross) the earlier fracture line, which naturally interrupts the cranial distortion which precedes fracturing (Figure 5.17).

Types of skull fracture

Arising from the mechanisms described above, it is conventional to classify skull fractures as follows.

LINEAR FRACTURES

These are straight or curved fracture lines, often of considerable length. They either radiate out from a depressed zone, or arise under or at a distance from the impact area, from bulging deformation. They may involve the inner or outer table, but commonly traverse both.

Depending on the stress contours of that part of the skull and the localization of the impact, they may occur anywhere in the skull, but are especially common in the weak unsupported plates. The temporal, frontal, parietal and occipital plates may all carry single or multiple linear fractures. They may extend downwards into the foramen magnum, across the supraorbital ridges, or into the floor of the skull. A common basal linear fracture is one that passes across the floor of the middle fossa, often following the petrous temporal or greater wing of the sphenoid bone into the pituitary fossa. This frequently continues symmetrically across the other middle fossa separating the base of the

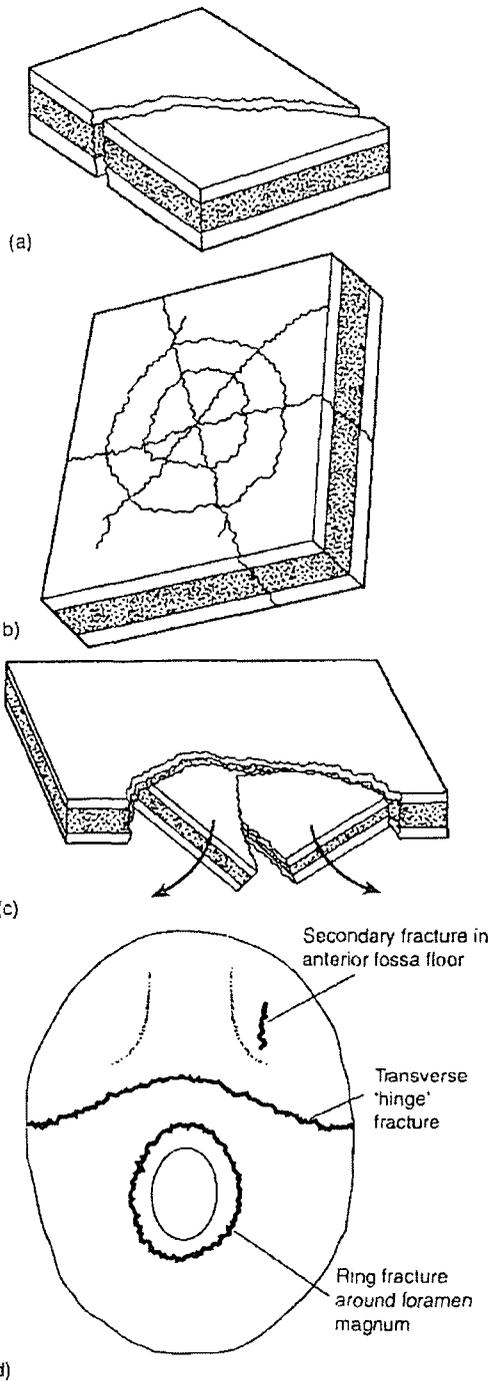


FIGURE 5.18 Types of skull fracture: (a) linear fracture; (b) 'spider-web' fracture; (c) depressed fracture; and (d) base fractures.

skull into two halves, usually being caused by a heavy blow on the side of the head; this lesion was sometimes called the 'motorcyclist's fracture' for obvious reasons. Linear fractures may follow a horizontal course around the skull, usually

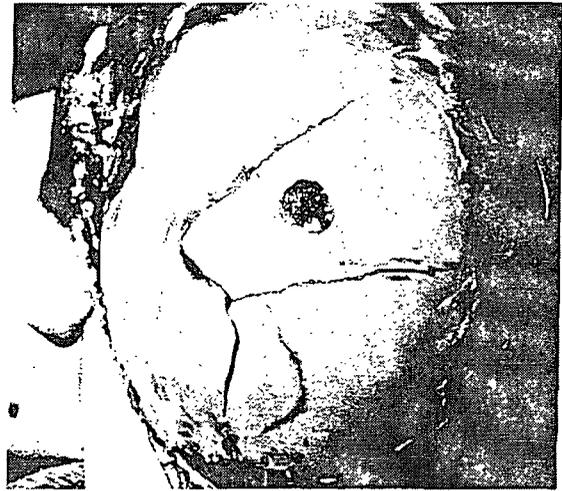


FIGURE 5.19 Linear fractures of the skull in child abuse. Blunt impact near the vertex has caused a double fracture to run down the parietal bone and continue into the temporal area, the course of the fracture being determined by lines of stress and weakness in the skull. The central burr-hole was for the surgical evacuation of a subdural haemorrhage.

from one temporal area to the other via the occiput rather than around the front.

In children and young adults, a linear fracture may pass into a suture line and cause a 'diastasis' or opening of the weaker seam between the bones. This is most often seen in the sagittal suture between the two parietal bones, but the interfrontal line of weakness left by the earlier fusion of the metopic suture can also reopen under mechanical stress. In infants, especially in the child abuse syndrome, a linear fracture of a parietal bone may reach the sagittal suture and continue across it into the opposite plate. The continuation may be direct or may be 'stepped', so that the two fractures are not in line. This appearance usually means a blow or fall onto the vertex, and the two fractures may be simultaneous but not continuous, explaining the 'stepping'.

RING FRACTURES

These occur in the posterior fossa around the foramen magnum and are most often caused by a fall from a height onto the feet. If the kinetic energy of the fall is not absorbed by fractures of the legs, pelvis or spine, the impact is transmitted up the cervical spine. This may be rammed into the skull, carrying a circle of occipital bone with it.

POND FRACTURES

This is merely a descriptive term for a shallow depressed fracture forming a concave 'pond'. It is more common in

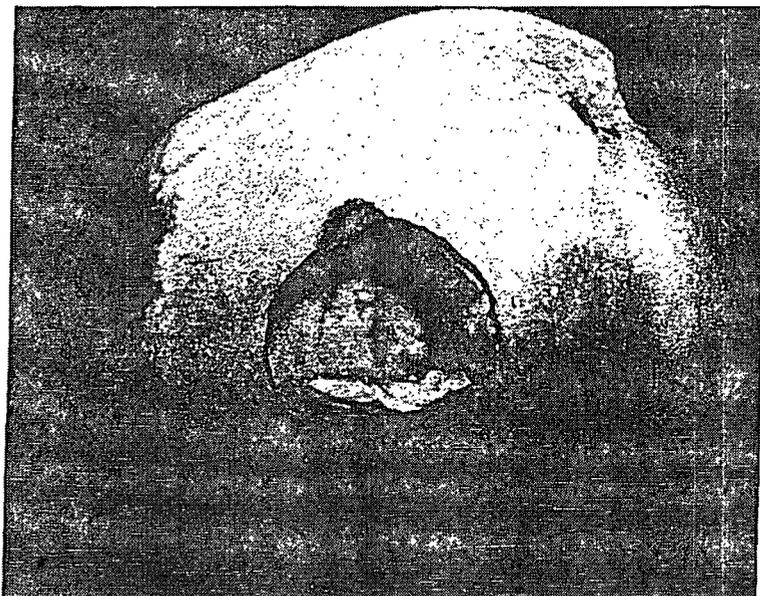


FIGURE 5.20 *Pond fracture.*

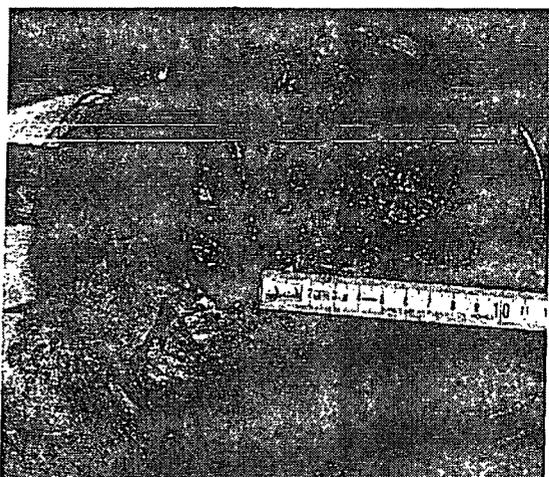


FIGURE 5.21 *Comminuted skull fracture with depression of the central area. This could also be termed a 'pond fracture' in that there is an ovoid depressed zone with radiating linear fractures. The head was struck with a heavy piece of wood along the line of the depressed area.*

the more pliable bones of infants and, indeed, a depression can occur in the absence of a fracture akin to the distortion produced by squeezing a table-tennis ball.

MOSAIC OR SPIDER'S-WEB FRACTURES

As already described, a comminuted depressed fracture may also have fissures radiating from it, forming a spider's-web or mosaic pattern. The degree of actual depression may be minimal or even absent.

DEPRESSED FRACTURES

Focal impact causes the outer table to be driven inwards and, unless absorbed in the diploë, the inner table will also usually be intruded into the cranial cavity with all the dangers of direct damage to the contents. Even sharp-edged weapons, such as heavy knives and axes, which may cause a clean-cut defect externally, usually split and deflect flaps downward from the inner table.

With axes and heavy cutting weapons such as swords, there is a characteristic lesion in the bones, whether skull or elsewhere. The initial impact slices cleanly through the bone on one edge, often burnishing the bone to an ivory-like gloss. The rebound removal of the weapon is at a slightly different angle, either from deliberate intent, or from relative movement between bone and blade. This cracks off an irregular fragment of bone of the opposite face so that the residual defect has one smooth and one rough edge. It is often seen in historical and archaeological material from battles or massacres.

The force required to cause fractures of the skull

Unlike less reliable subjective estimates of the force required to cause other injuries, objective quantitative measurements have obtained for adult skull fractures. Again, the publications of Gurdjian, Webster and Lissner should be consulted for detailed information. The following useful facts arise from their investigations:

- ☒ The tensile strength of the adult skull is of the order of 100–150 p.s.i., the compressive strength varying from



FIGURE 5.22 A depressed fracture of the skull from a blow with a heavy club hammer. The defect is wedge shaped with a curved anterior border caused by the hammer striking at an angle. The depression is concentrically terraced, with the lowest fragments lacerating the surface of the brain.

5000 to 31 000 p.s.i. (Gurdjian *et al.* 1949). A simple fissured fracture of the skull can be sustained by walking into a fixed obstruction (Kerr 1954). This requires a force of about 5 foot-pounds (73 N). Fast running into an obstruction produces about 70 ft-lb (1020 N). Falling to the ground from an erect posture also develops at least 60 ft-lb (873 N) and can easily produce skull fractures. A small stone or golf ball weighing about 100 g (4 oz) may also cause a linear fracture when thrown with moderate force against the temporal region (Graham and Lantos 2002).

- The adult head weighs between 3 and 6 kg (7–14 lb), averaging 4–5 kg (10 lb). When falling through about one metre (3 ft), so that the frontal area strikes a hard surface, impact energy of about 35 ft-lb (510 N) develops. This can cause one or two linear fractures or a mosaic fracture (Gurdjian *et al.* 1950).
- Only a small additional amount of energy above that needed to cause a single crack is required to produce multiple fractures.

In spite of these experimental data, it must never be forgotten that, like all biological phenomena, great variation is encountered and skull fractures, though they may be caused by as little as 5 ft-lb (73 N), may be absent when the impact exceeds 90 ft-lb (1314 N). The area of the skull struck, the thickness of the skull, scalp and hair, the direction of the impact and other imponderables, all affect the outcome.

As further discussed in Chapter 22, Weber carried out experiments in which he dropped the bodies of dead infants from a fixed height of only about 85 cm (34 inches) onto various hard and soft surfaces. A high proportion sustained skull fractures, both of the parietal and occipital areas, some of them crossing suture lines.



FIGURE 5.23 Springing of the sagittal suture of the skull of an infant. Though there was no skull fracture, the suture line has widened after a fall that caused a subdural haemorrhage.



FIGURE 5.24 Diastasis or 'springing' of the sagittal suture of a young adult who fell from a height onto the vertex of his head. The opening of the interparietal suture has extended anteriorly along the line of weakness of the fused metopic suture between the two halves of the frontal bone.



FIGURE 5.25 Post-mortem diastasis of the sutures as an artefact due to freeze-up of the brain simulating intra-vital skull fracture.



FIGURE 5.26 A 'hinge' fracture of the base of the skull where the fracture line runs from side to side across the floor of the middle cranial fossa, passing through the pituitary fossa in the midline, following the course of least structural resistance. The victim was a young pedestrian who was hit by a car and then sustained secondary injuries by striking the left side of the head on the ground. This injury is common in road accidents and is sometimes called the 'motorcyclist's fracture'.

Dangers of fractures of the skull

It has been emphasized that, in the majority of cases, the significance of a fractured skull is an indicator of a substantial insult to the head, with possible injury to the vital contents, rather than the fracture itself being a danger to life.

There are occasions, however, when the fracture itself has dangerous sequelae. The most common is when the crack passes through an embedded meningeal artery, causing a meningeal haemorrhage, which is considered later. A depressed fracture may impinge upon the brain and its membranes, and bone fragments may lacerate or penetrate the brain tissue.

TRAUMATIC EPILEPSY

A late effect of a depressed skull fracture may be 'traumatic epilepsy'. This is of great medico-legal significance, especially in the field of civil litigation where an accident or assault may result in lifelong neurological disability for which very large monetary compensation may be awarded.

Traumatic epilepsy usually manifests as tonic and clonic fits, which may be difficult to differentiate from idiopathic epilepsy, if the injury occurred in early life. When fits begin within weeks, or a year or two of a major head injury in a mature person who had never had fits before, the diagnosis is easier, but all cases need expert neurological examination. There must have been a substantial head injury, usually with a depressed fracture impinging on the underlying cortex, often in the parietotemporal area. The epilepsy appears usually within a range of a few weeks to up to 2 years. It is more common in open head injuries when infection has occurred, or when a spicule of bone has penetrated the meninges, as the mesodermal scarring of the cortex that results is more likely to irritate the brain than the astrocytic reaction found in closed head injuries.

INFECTION FOLLOWING SKULL FRACTURE

Other complications may occur, even in the absence of depression or comminution. The most common is infection of the meninges, or the development of a brain abscess, or both. Infection can gain access via skull fractures:

- ☞ by direct spread through a compound fracture, especially where there is a contaminated scalp injury;
- ☞ by spread from the nasal cavity when a fracture of the cribriform plate has allowed communication with the anterior fossa. This fracture is not uncommon as part of 'contrecoup' lesions, described later. Sometimes the cribriform plate is ruptured by objects entering a nostril and carrying infected material into the cranium: the author (BK) has seen two such fatalities, one from an umbrella ferrule and another from a dirty bamboo garden cane. Another case was seen by PS where a suicide attempt was made with a small calibre pistol. The bullet penetrated the right temple and went through the skull just above the anterior cranial fossa, which was fractured and caused a leakage of the cerebrospinal fluid through the nose. The victim recovered well but, in spite of prophylactic treatment with antibiotics, died one year later from purulent meningitis.
- ☞ by spread from fractures that involve a paranasal sinus, such as the frontal or ethmoid, or from the mastoid air cells or middle ear cavity. Basal fractures may allow these spaces, which communicate with the infected outside environment, to reach the meninges, especially when the latter are torn by the traumatic event. A history of leakage of cerebrospinal fluid from the nose or ear must alert both clinician and pathologist to the possibility of communicating basal fractures. In any autopsy on a suspected head injury, however slight, care must be taken to remove the dura from the interior

of the vault and base so that a close inspection for fracture lines can be made.

INTRACRANIAL INJURIES

The contents of the skull are the most fragile of the vital organs, necessitating their enclosure in the strong bony box of the cranium. Damage may occur either to the neural tissues or to the rich vasculature that surrounds and penetrates those tissues.

FORENSIC ANATOMY OF THE BRAIN MEMBRANES

The pachymeninges consist of the dura mater and the leptomeninges, the arachnoid and the pia mater (see Figure 5.13). The **dura** is formed of two layers of tough collagenous tissue, the outer of which is firmly attached to the skull and acts as its internal periosteum. The inner layer merges with the arachnoid, so that in reality there is no true subdural space, only a potential cleavage plane.

The dura forms the falx and tentorium, and the cranial venous sinuses run within it. Branches of the meningeal arteries course over and through its substance. The dura is penetrated by bridging veins, especially along the vertex and at the tips of the temporal lobes, also to a lesser extent at the frontal and occipital poles, as well as by random vessels elsewhere. Polypoid invaginations of the dura penetrate the inner walls of the venous sinuses, especially the sagittal sinus, to form the 'arachnoid granulations'.

The **arachnoid** is a thin, vascular meshwork that is intimately applied to the inner surface of the dura by means of the 'boundary layer' so that no subdural space exists in normal conditions, though their junction is so tenuous that they are easily split apart. Sheaths of arachnoid follow vessels into the brain as they penetrate into the neural surface. These vessels and thin strands of connective tissue anchor the brain within the subarachnoid space. This is filled with cerebrospinal fluid and the width of the space varies from less than a millimetre in the young to a centimetre or more in the old, in whom cerebral atrophy has developed. This means that the anchoring strands and the bridging vessels are longer and more vulnerable to shearing and rotatory stresses. Even though anatomically in the subarachnoid space, rupture of these bridging vessels often manifests itself in the subdural space.

The **pia** is not a true membrane, but a surface feltwork of glial fibres that are inseparable from the underlying brain.

EXTRADURAL HAEMORRHAGE

Also known as 'epidural haemorrhage', bleeding between the inner surface of the skull and the dura mater is the least common of the three types of brain membrane haemorrhage. According to Rowbotham (1964), only about 3 per cent of head injuries have an epidural haemorrhage large enough to be of surgical significance; a similar figure of between 1 and 3 per cent was recorded by Tomlinson (1970). Of the 635 fatal head injuries investigated by Adams (see References and further reading) in Glasgow, 10 per cent had extradural haemorrhages. The mortality rate, even with surgical intervention, averages about 11 per cent under the age of 20 years, rising to between 18 and 40 per cent in later life.

The dura is closely applied to the interior of the skull, forming the endocranium or periosteum. It is so tightly applied to the base of the skull that, except in the posterior fossa, extradural bleeding does not occur over the skull floor. In the vault there is a potential space between the dura and the bone, which can be separated by both arterial and – less often – venous leakage. Most extradural haemorrhages are associated with fractures of the skull, but about 15 per cent occur in intact skulls (McKissock 1960). According to Harwood-Nash *et al.* (1971), the incidence in children without a fracture is only 1 per cent, though Adams found that half the children with extradural bleeds in his series of over 600 fatal head injuries had no fracture. About 10 per cent of extradural haemorrhages are associated with subdural haemorrhages. Bilateral epidural haemorrhages are rare, but have been recorded.

The usual site is unilateral in the parietotemporal area, caused by rupture of a branch of the middle meningeal artery where the latter is transected by a fracture line. The posterior branch of this vessel is most commonly involved as it courses diagonally backwards across the squamous temporal bone on the lateral wall of the cranium. The anterior (frontal) branch is rarely the source of bleeding, occurring only twice in Rowbotham's series of 33 cases. The vessel usually lies in a deep osseous groove in the first part of its course. It has been claimed that almost all ruptures occur where the artery is completely roofed over in a bony tunnel so that it is unable to escape damage from a fracture, but observation does not confirm this contention.

Leakage of the high-pressure arterial blood strips back the underlying dura with progressive accumulation of a haematoma, which can reach a volume of several hundred millilitres and cover an appreciable part of the hemispheric. Adams suggests that a minimum volume of 35 ml is needed before clinical signs are apparent, though other writers suggest 100 ml is usually the minimum associated with fatalities.

In the less common occipital and frontal sites, smaller branches of meningeal arteries may be involved or the bleeding may be from torn venous sinuses, in which case there need not be a fracture. When bleeding is venous, the haematomas rarely reach a large size as the pressure is insufficient to tear back much of the dura.

The clinical signs of an epidural haemorrhage are classically those of a 'lucid' or 'latent' interval, as there may be recovery from the initial phase of concussion (see below) before sufficient blood accumulates to cause raised intracranial pressure and consequent relapse into unconsciousness. This classic picture is so frequently absent, however, that no diagnostic reliance can be placed upon it. The coma from the increasing space-occupying lesion formed by the bleed may follow the period of concussion without a break so that there is no temporary phase of recovery. Only 27 per cent of McKissock's series showed the classic history.

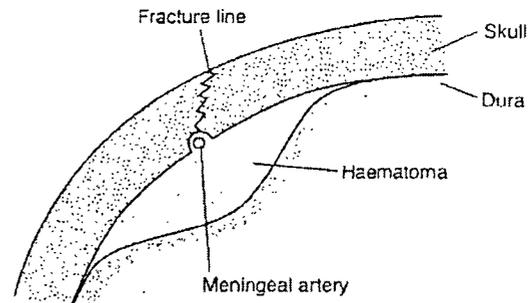


FIGURE 5.27 Formation of an extradural haemorrhage.



FIGURE 5.28 A large temporoparietal extradural haemorrhage. The under surface of the scalp on the right shows bruising at the points of impact. There was a linear fracture of the skull passing through the right middle meningeal artery.

Extradural haemorrhage

The latent interval may be of variable duration and, as just noted, may not occur if either concussion is prolonged or other brain damage coexists. Half an hour may be enough for the formation of a significant arterial haematoma, though some slow bleeds have taken more than a day to become clinically apparent. In Rowbotham's series, the range was from 2 hours to 7 days, but most were apparent after 4 hours.

More recent investigations using computed tomography, quoted by Adams, suggest that the old concept of worsening clinical symptoms being caused by the progressive accumulation of blood is incorrect, as computed tomograms show that the major volume of blood may appear soon after the injury and that, as with many space-occupying lesions, clinical signs result from other factors, such as cerebral oedema or diffuse neuronal injury.

Medico-legal considerations of extradural haemorrhage

A medico-legal danger is that a victim may be discharged from the care of a doctor or hospital casualty department when he recovers from his transient concussion only to deteriorate and perhaps die at home; negligence may then be alleged against the unsuspecting doctor. Unfortunately, even when the diagnosis is made, the results of surgical intervention are not good, there being a fatal outcome in more than half the cases operated on. Part of the reason for this poor prognosis is that many victims of extradural haemorrhage also have other damage such as cerebral contusion.

When a victim of a head injury recovers from concussion and then lapses into coma within the first 24 hours, the differential diagnosis is between an extradural or subdural haemorrhage, or cerebral fat embolism, especially if there is other skeletal damage. At autopsy the lesion is self-evident, as most haematomas lie in the temporal or parietal areas, and are opened when the usual saw-cut is made to remove

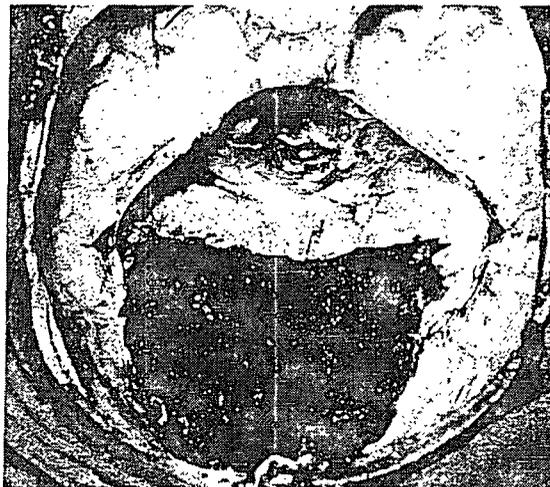


FIGURE 5.29 An extradural haemorrhage in the posterior fossa. This is an unusual site for this type of lesion, as most occur in the temporal or parietal area as a result of tearing of the middle meningeal artery. In this case a fracture line passed down the posterior fossa towards the foramen magnum, but no bleeding point could be identified.

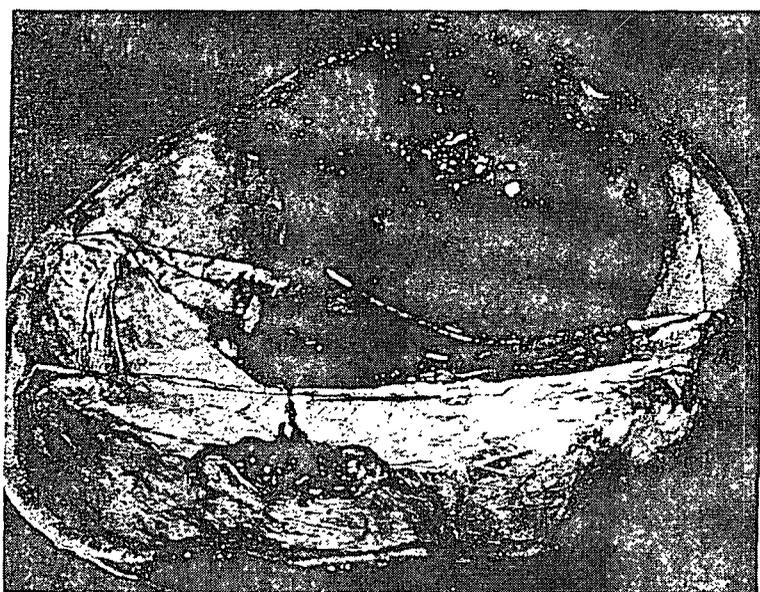


FIGURE 5.30 A fresh extradural haemorrhage in the temporoparietal area from a fracture crossing the middle meningeal artery. About 85 per cent of such haemorrhages are associated with skull fractures.

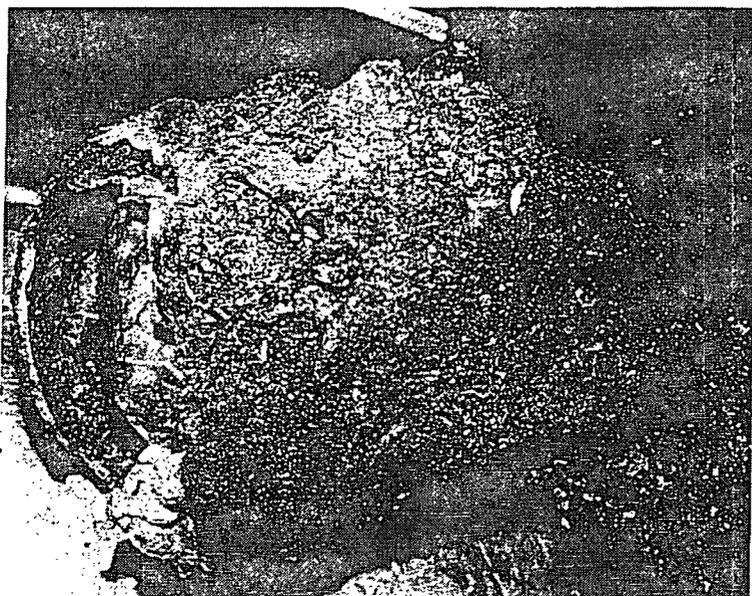


FIGURE 5.31 *Fire victim with extensive defect of the carbonized skull showing reddish-brown 'heat haematoma' on the inner surface of the skull and shrunken brain with brain tissue oozing out through a split in the dura.*

the calvarium. If in the posterior fossa, the dark blood is seen through the raised dura. Wherever it is situated the dimensions and approximate volume should be measured and an estimate made of the inward projection. The brain surface will be flattened or otherwise distorted if the haematoma is of significant size and may have given rise to the usual appearances of raised intracranial pressure, described later. The midline of the brain may be shifted laterally if the bleed is large.

Extradural haemorrhage is never a 'contrecoup' injury, this being purely a cerebral tissue lesion.

HEAT HAEMATOMA

One well-known artefact mimics an extradural haemorrhage. When a head has been exposed to severe external heat sufficient to burn the scalp and perhaps the skull, blood may be extruded from the diploë and venous sinuses into the extradural space to produce a 'heat haematoma'.

The mechanism is obscure, but may be the result of blood being 'boiled' from the diploic layer of bone through emissary veins, or shrinkage of the brain may aspirate blood from the skull. The false haematoma is brown and friable, and the adjacent brain shows hardening and discoloration from the heat.

The importance of the artefact is that it may be mistaken for a true epidural bleed from a head injury, and may mislead the pathologist and investigators into thinking that the fire was started criminally to cover up a fatal assault. As most instances are seen in conflagrations in buildings,

there is often a significant level of carboxyhaemoglobin in the body if the death occurred when the fire was in progress. This should be of the same concentration in the heat haematoma as in the peripheral blood; if the victim suffered a head injury before the fire started, then there should be little or no carboxyhaemoglobin in the haematoma.

SUBDURAL HAEMORRHAGE

Bleeding beneath the dura is much more common than extradural haemorrhage. It is also proportionately less often associated with a fractured skull, but in absolute numbers far more fractured skulls cover subdural than extradural haemorrhages. The Glasgow series of 635 fatal head injuries described by Adams included 18 per cent of subdural haematomata.

The lesion is traditionally classified into three types: the acute, the subacute and the chronic. It is unhelpful to subdivide the acute type, however, and only acute and chronic haemorrhage need be considered.

Subdural haemorrhage can occur at any age, but is common at both extremes of life. It is one of the major causes of fatal child abuse and the rediscovery of that syndrome by Caffey (1946) consisted of an association of subdural haemorrhage with long bone fractures. In old people they commonly exist in a chronic form and can be mistaken either for 'strokes' or for senile dementia. The condition is always due to trauma and there is probably no such entity as 'spontaneous subdural haematoma'. Even in states of vascular fragility, such as in senility and in bleeding diatheses,

some minimal trauma must precipitate the bleeding even if it was too trivial to be recorded in the history. It is almost certain that minor subdural bleeds, insufficient to give rise to any neurological or clinical symptoms or signs other than a transient headache, occur with the trivial knocks of everyday life. Only when the bleeding is extensive enough to become either a cortical irritant or a space-occupying lesion (probably between 35 and 100 ml) does it become clinically apparent. Of course, many subdural haemorrhages exist in combination with both subarachnoid bleeding and with cerebral damage, making its contribution to the overall symptomatology impossible to assess.

As with the extradural haematoma, the position of a subdural can never be interpreted as a 'contrecoup' lesion and is thus of no use in differentiating a blow from a fall.

Acute subdural haemorrhage

This is a common sequel to any substantial head injury, and the presence or absence of a fracture is immaterial except as an indicator of trauma to the head. Unlike extradural bleeding, a fracture plays no part in the pathogenesis of the haemorrhage, which arises from torn communicating veins that traverse the subdural space between the cortical vessels and the dural sinuses. Less often the sinuses themselves give rise to the haemorrhage.

Naturally in an open head injury or when comminuted fractures penetrate the membranes and perhaps the brain itself, subdural bleeding is merely part of a complex that includes subarachnoid bleeding, and cerebral laceration and contusion.

The lesion is often pure, however, being associated with a closed head injury where the only other signs may be scalp bruising – or even nothing at all, as blunt impacts may leave no signs in the scalp, externally or internally, and no skull fracture.

The latter situation is probably the explanation in most of the cases formerly attributed to shaking of the infant. Many paediatricians and pathologists have enthusiastically adopted the shaking aetiology when there was no overt sign of impact (or sometimes even where there was such evidence!) to such an extent that it is frequently proffered as the favoured diagnosis. However, the concept of the shaken-baby subdural has been strongly challenged recently, as it has been shown that the shearing force (required to rupture subdural vessels) is of the order of 50 times less in shaking than in impact (Duhaime *et al.* 1987). Thus it is very probable that perhaps the majority of allegedly shaken babies have, in fact, had an occult head impact, which has not left any signs on the scalp, subscalp tissues or skull.

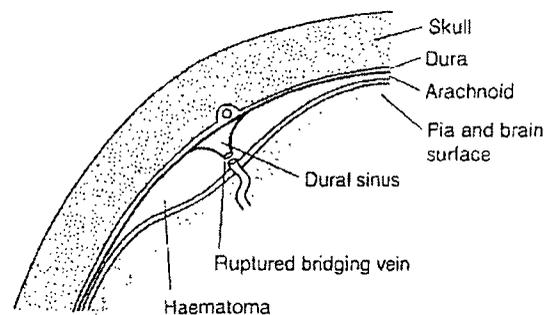


FIGURE 5.32 Formation of a subdural haematoma.

Subdural bleeding arises from shear stresses in the upper layers of the cerebrum, which moves the communicating veins laterally sufficiently to rupture their junctions at either the cortical veins or the sinus surfaces. It is very rarely possible to identify the bleeding points. Subdural bleeding is most often over the lateral surface of a cerebral hemisphere, high up in the parasagittal area.

As with most intracranial damage, the mechanical cause is a change of velocity of the head, either acceleration or deceleration, almost always with a rotational component. Where a blunt impact strikes the skull, the subdural bleed need not be situated directly under the impact area – it need not even be on the same side of the head. It is sometimes tempting to attribute a localized subdural to either a 'coup' or to a 'contrecoup' effect (see below), but this is an unsafe interpretation. In addition, a subdural haemorrhage – unlike an epidural – is quite mobile. Lesions that have obviously originated high on the parietal area commonly drain down under gravity and cover the whole hemisphere, with a large accumulation in the middle and anterior fossas, and even through the tentorial opening into the posterior fossa.

The haemorrhage may remain fluid or may clot into a firm mass: both modes are commonly present. If the bleeding is relatively slight, then a 'thin-film' subdural may be found. If the thickness of blood is less than a few millimetres, it cannot be claimed to be a space-occupying lesion, even if the area covered is quite large, as the cerebrospinal fluid in the adjacent subarachnoid compartment can be displaced sufficiently to accommodate an equivalent volume of blood.

Again, Adams suggests that, as in any intracranial space-occupying lesion, a minimum volume of about 35 ml is required to cause neurological signs, though other writers prefer a larger volume, such as 100 ml.

Whether this film of fresh blood is a sufficient irritant to cortical activity – as it undoubtedly is in a subarachnoid haemorrhage – to be a danger to life, is uncertain. It is difficult to claim that a thin layer of blood in the subdural

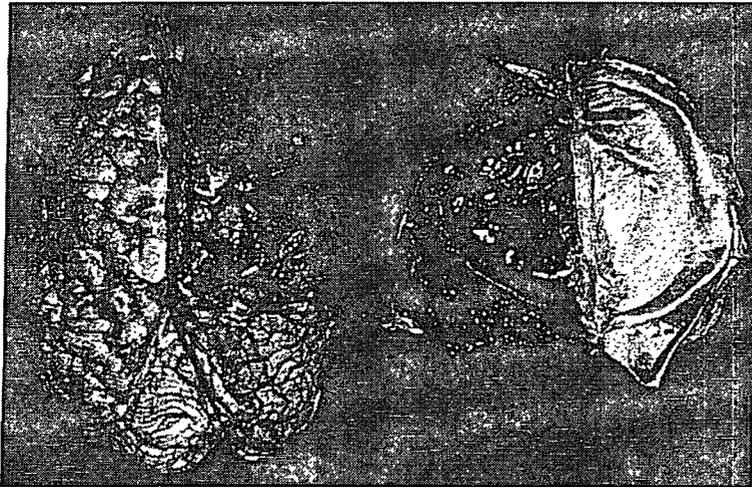


FIGURE 5.33 *Chronic subdural haemorrhage in an old person. Brown liquid escaped from the encapsulated lesion adherent to the meninges leaving a gelatinous outer membrane, as seen. The surface of the right cerebral hemisphere is stained brown from old altered blood and there is some compression of the hemisphere with a midline shift to the left. There was no history of a head injury and no significant neurological deficit.*

space is the sole cause of death, but almost inevitably the force that caused the bleeding will also have had a deleterious effect on the brain tissue, even if this is macroscopically occult such as diffuse axonal damage.

As with the extradural haemorrhage, there may be a latent interval before clinical signs and symptoms appear. After the almost inevitable concussion, which may be very brief, the victim may recover, then relapse into deepening stupor and coma when intracranial pressure rises as the subdural bleeding proceeds. Associated brain damage may, however, cause uninterrupted coma from the time of injury.

When there is a lucid interval, this may be longer than the average 4 hours of the faster arterial bleeding of the epidural haemorrhage. In fact, there is no upper limit to this interval, as the acute subdural haemorrhage merges into the chronic condition, which may recur after weeks or even months. In severe acute bleeds, as are commonly encountered in criminal cases, such intervals tend to be short or non-existent.

Chronic subdural haematoma

This lesion is most often found in old people, frequently as an incidental finding at autopsy where death was caused by some unrelated condition.

The gross appearance varies with age; recent lesions up to several weeks old are tan or red-brown with a gelatinous membrane covering the surface. The contents are thick but liquid and may have areas of redder, more recent bleeding. An older haematoma, up to months or even a year old, is firmer, with a tough membrane around both surfaces, resembling a rubber hot water bottle filled with jelly or oil. The contents are liquid and may be brown or even straw coloured. Sometimes the interior may be much firmer and

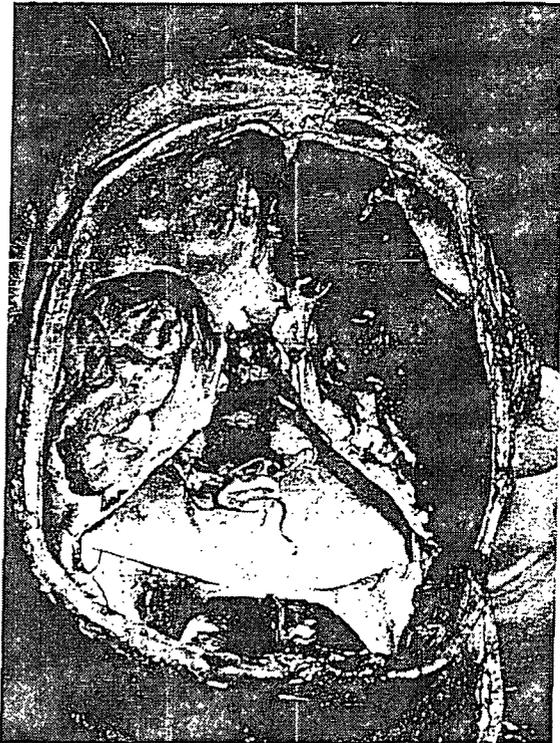


FIGURE 5.34 *A chronic subdural haemorrhage discovered as an incidental finding at an autopsy on an old person dying of an unrelated cause. The blood was brown and gelatinous, but there was no membrane.*

variegated in colour due to bleeds of different ages. Loculation is common, with different coloured fluids or ooze in each locule. The underlying brain will be depressed if the haematoma is large (more than 50–100 ml), and is often stained brown or yellow from altered haemoglobin.

Many such subdural bleeds in old people are small and obviously gave rise to no neurological abnormality; the mere finding of such a small lesion at autopsy should not be used to provide a cause of death, which should be sought elsewhere in the body. Some subdurals are substantial, however, reaching a volume of 100–150 ml. They may still have been asymptomatic, but others give rise to neurological symptoms that may have been ascribed to some other pathological cause. Examples are 'strokes', when unilateral signs were thought to be caused by a cerebral thrombosis or haemorrhage; disordered behaviour of the old person may have been blamed upon senile confusion or dementia, whereas in fact a space-occupying haematoma was really responsible.

The chronic haematoma may become large and press down on the cerebral hemisphere sufficiently to dent and distort the surface. This may progress, as does the large acute haemorrhage, to cause signs of hippocampal and cerebellar tonsillar herniation and all the attendant dangers to the vital centres in the brainstem. The chronic haematoma arises from the acute lesion, which, after an interval, becomes sheathed in a capsule of connective tissue. The haematoma may eventually absorb, it may remain dormant at the same size, or it may enlarge at any later date.

The mechanism of the enlargement is controversial. One common explanation, which seems the most reasonable, is that it occurs from repeated further bleeding, perhaps from new blood vessels that penetrate the mass as part of the healing process. The other theory involves osmosis, said to operate because the centre of the haemorrhage commonly liquefies, forming a haemorrhagic fluid that osmotically attracts into it the cerebrospinal fluid from outside the capsule, which acts as a semipermeable membrane.

The first mechanism seems more likely, as areas of fresh bleeding are often found inside a substantial haematoma but, whatever the cause, the final effect is a worsening of the space-occupying effect.

The dating of a subdural haemorrhage

An estimation of the date of onset of a subdural haematoma may have considerable forensic significance, especially if the lesion is obviously mature. There may have been one or more episodes of trauma on record, any of which may have criminal or civil connotations and the opinion of the pathologist will be sought to relate or to eliminate the lesion found from the potential causative event.

For example, in one of the author's cases an old lady was struck on the head by intruders and died several weeks later. At autopsy, a large chronic subdural haematoma was the main finding, but the defence sought to show that the haematoma must have been present before the assault by

claiming that the woman's confused behaviour over the previous year was caused by the pre-existing intracerebral lesion. Furthermore, in accidents of any type, there may be doubt about whether the subdural haematoma arose as a result of the injury, or whether a pre-existing lesion caused unsteadiness that may have precipitated the accident.

Unfortunately, in spite of several claims to reliable methods of dating subdural haematoma, such estimations are of doubtful value, partly because repetitive bleeding results in varying ages within the same haematoma.

A subdural haematoma gradually changes from dark red to a brownish colour, first being apparent not before 5 days and sometimes not obvious for 10–12 days (Crompton 1971, 1985).

Reaction to subdural bleeding begins within a few hours of onset, when cellular infiltration begins from the dural surface. A delicate 'neomembrane', histologically composed of thin-walled capillaries and fibroblastic granulation tissue, grows from the periphery to cover the outer (dural) surface of the clot during the next few days and weeks. If no further enlargement occurs, this capsule becomes more and more fibrous, though rarely does it completely absorb the haematoma by fusing with the outer capsule. According to Crompton, the presence of a membrane firm enough to be picked off with forceps makes the subdural haemorrhage at least 12 days old.

Although claims have been made for accurate dating of subdural haematomas by histological criteria (Munro and Merritt 1936), these cannot be depended upon, especially after a few months, as there is considerable personal variation in healing rates. Also, because of the frequency of repeated subsequent fresh bleeding, attempts at estimating the date of the original bleed are unrealistic. As they are of some use as an approximate guide to age, Munro and Merritt's histological criteria are summarized briefly here:

- ☒ Fibroblasts appear at the margin of the clot within 36 hours and in 4 days the neomembrane adjacent to the dura is a few cells thick. From 5 to 8 days, the membrane becomes well established and fibroblasts migrate from it into the clot.
- ☒ By 8 days the membrane is 12–14 cells thick and is visible to the naked eye. From a few days after onset, there is progressive red-cell lysis and after 5 days haemosiderin-laden phagocytes are present, which may be stained by Perl's reaction.
- ☒ From 11 days the clot is subdivided by strands of fibroblasts. By 15 days a membrane is also present on the under surface of the clot and the outer neomembrane is half to one-third the thickness of the dura itself. By day 26 it equals the thickness of the dura, but the inner membrane is still only half as thick.

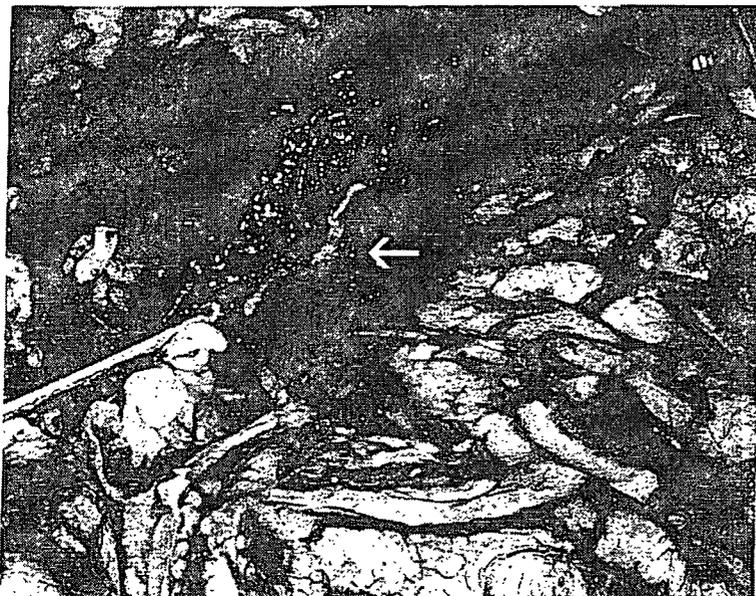


FIGURE 5.35 Subarachnoid haemorrhage from a tiny berry aneurysm (arrow) of the right middle cerebral artery.

Between 1 and 3 months the membrane has lost many fibroblast nuclei and is becoming hyaline. By 6–12 months the membrane becomes thick and fibrous, resembling the dura itself.

A number of later writers have pointed to marked variations in this chronological scheme. Within the first couple of months large sinusoidal vessels appear in the newly formed connective tissue.

From gross appearances, a rough guide is that brown colour changes occur between the first and second week, when a discrete surface membrane becomes obvious. After a month or so a firm capsule develops, forming a cystic cavity containing dark brown, watery fluid. According to Munro, liquefaction of the contents does not occur in less than 3 weeks. Some haematomas remain solid with an organizing blood clot within, often with areas of fresher haemorrhage of different ages.

When a substantial subdural bleed has occurred, the underlying cerebral cortex may be infarcted, caused by either a natural infarct having bled through the arachnoid into the adjacent subdural space or, more often when there has been a head injury, the pressure of the haematoma on cortical blood vessels (Crompton 1971, 1985).

SUBARACHNOID HAEMORRHAGE

The third type of brain membrane bleeding is even more common than subdural haemorrhage, but has a mixed aetiology. Whenever there is damage to the cortex, there will be some degree of subarachnoid bleeding, so all penetrating injuries of the brain, as well as many blunt injuries that

give rise to extradural or subdural haemorrhage, will be associated with traumatic subarachnoid bleeding.

It is not common, though not unknown, for traumatic subarachnoid bleeding to occur as a pure lesion where there is no cortical contusion, no neck injury, no deep brain lesion and no other membrane haemorrhage. Slight subarachnoid bleeding probably occurs very frequently after a moderate impact upon the head (as with slight subdural bleeding), but as the vast majority of such victims survive, no autopsy evidence is forthcoming. The increased sensitivity of medical imaging techniques, especially nuclear magnetic resonance, can demonstrate such minor bleeds which hitherto went undetected.

The other complicating issue with subarachnoid bleeding is that it frequently occurs as a result of natural disease, especially rupture of vascular malformations of several types. When trauma is also present, the complex association of either the trauma precipitating the rupture or a rupture causing a fall or other accident leading to the trauma, has to be considered. This is discussed in a later paragraph.

Although the pathology of subarachnoid haemorrhage is of prime forensic importance, because of the relationship to ruptured berry aneurysm, much of the description is offered in the chapter on sudden natural death (Chapter 25), and only the interaction between trauma and this type of bleeding is discussed here.

Appearances and mechanism of formation

The appearance of subarachnoid haemorrhage caused by trauma varies greatly according to the nature and extent of the



FIGURE 5.36 An extensive basal subarachnoid haemorrhage arising from a traumatic tear of a basilovertbral artery caused by a blow on the side of the neck.

injury. Where it is secondary to laceration of the brain or extensive cortical contusion, then its localization and severity depend upon the primary injury. Where it arises from a blunt impact, with or without other membrane bleeding or cortical bruising, its position is not a good localizing sign. Once again, its position cannot be interpreted in the same way as contrecoup contusion of the cortex, though some pathologists use its position to claim that a head injury was sustained either from a fall or from a blow to a mobile head; this is unjustified. Although where the circumstances are known, the meningeal bleeding may be seen in the correct location, this is fortuitous and is equally often sited elsewhere.

Blood in the subarachnoid space mixes with the cerebrospinal fluid, which dilutes it, makes it less ready to clot and allows more mobility. Thus bleeding high over the cerebral hemispheres readily slides down to cover the brain and enter the basal skull fossae, but usually not in a concentration sufficient to form a thick clot. The sulci tend to collect more blood, especially in the insula. Unlike those with subdural bleeding, survivors from subarachnoid haemorrhages rapidly dispose of the blood. Haemolysis turns the cerebrospinal fluid a xanthochromic yellow and, within weeks, the blood is gone. There may be some residual brown or yellow staining of the pia or arachnoid, similar to that seen when a slight dural bleed leaves its signature on the inner surface of the dura. A positive Perl's reaction for haemosiderin can develop within 36 hours and may remain for

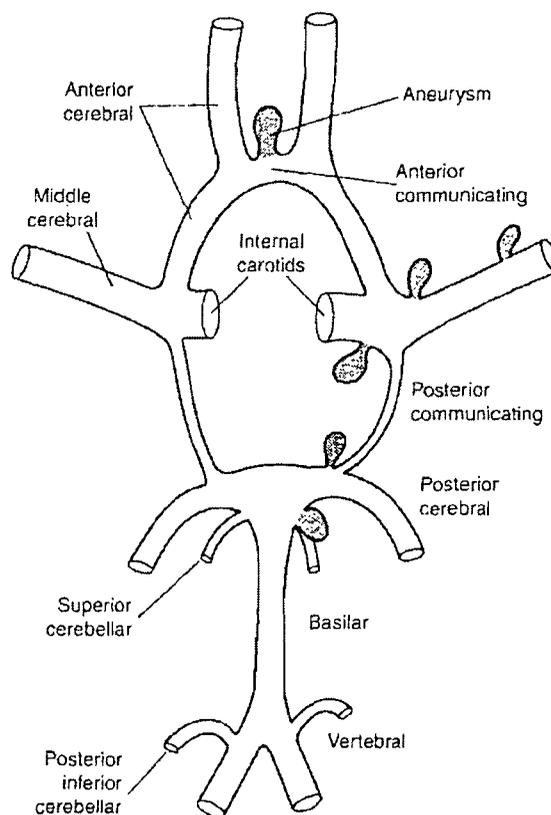


FIGURE 5.37 Cerebral arteries ('circle of Willis') and common sites for aneurysms.

months or even years, even after macroscopic discoloration of the membranes has vanished.

This forms a trap for those attempting to date both subdural and subarachnoid haemorrhage in infants, as the common bleeding arising from head-moulding during childbirth may leave a positive haemosiderin reaction for at least a year. Thus if a suspected child abuse head injury occurs in the first months of life, histological attempts to date the bleeding by Perl's reaction may be affected by residual haemosiderin from parturition.

Bleeding in the subarachnoid space is caused by the same mechanism as that in the subdural space, as shear stresses and rotational movements of the brain, described in a later section, shear or rupture the bridging veins that leave the cortex and penetrate the arachnoid en route for the large draining veins and sinuses that lie in the dura. In addition, small cortical arteries may contribute much of the leakage. Where laceration, contusion or infarction of the cortex is present, bleeding will come from cortical veins and small arteries directly into the subarachnoid space. It may also arise from intracerebral bleeding breaking out through the cortex. The subarachnoid haemorrhage allegedly caused by

damage to the vertebral arteries near the foramen magnum will be discussed under spinal injuries.

The relevance of a subarachnoid haemorrhage in causing death may be difficult to decide, though this is of more importance in the natural variety caused by a ruptured aneurysm.

In head injuries, death is far more likely to be the result of the other concomitant associated injuries to the brain substance than to a moderate amount of blood in the subarachnoid space.

In the uncommon instances when a substantial subarachnoid haemorrhage seems to be the only sequel of a fatal head injury it must be accepted as the cause of death, though cerebral oedema and microscopic diffuse axonal injury should be excluded as competitors even though this exclusion may be difficult or impossible when death follows rapidly after injury. It is indisputable, however, that sudden death can occur from massive natural subarachnoid haemorrhage in the absence of any traumatic lesions so, by analogy, the pure traumatic bleed – if severe enough – cannot be excluded as a sole cause.

Death can be remarkably rapid when a profuse haemorrhage occurs into the subarachnoid space. The mechanism is not understood, but seems to be confined to those cases where the brainstem is suddenly exposed to a large volume of blood bathing the brainstem in the posterior fossa, usually from a ruptured aneurysm or from a torn basilar or vertebral artery. Numerous instances of victims virtually dropping dead are on record, though many more exhibit the familiar signs of headache, neck stiffness, vomiting and progressive failure of consciousness, before dying at a variable period after the haemorrhage.

FORENSIC IMPLICATIONS OF BRAIN MEMBRANE HAEMORRHAGE

Because of its common occurrence, certain medico-legal problems associated with bleeding into the brain membranes must be considered.

First, the latent or 'lucid' interval in both extradural and subdural haemorrhage may lead to civil suits for negligence where a doctor – usually in a hospital emergency department – discharges an apparently well patient who later dies at home or is readmitted *in extremis*. The clinical aspects cannot be considered here, but suffice to say that it is highly unlikely that such patients would not have suffered concussion, albeit transient. Radiology of the skull is not mandatory if the clinical indications are not present, but failure to X-ray can be a legal impediment for the defence. A subdural

haemorrhage is commonly present with an intact skull, though this is uncommon with extradural bleeding.

As already mentioned, the dating of subdural haematomas may be crucial in both criminal and civil cases when it is being either maintained or denied that a particular episode of head injury causes the haematoma. Dogmatic adherence to any accurate histological dating regimen is unjustified, though broad distinctions in terms of days, weeks or months may be justified.

The possibility of a previous bleed being the precipitating factor in some later accidental injury, which then caused further bleeding, must be kept in mind.

Using a membrane haemorrhage to localize an impact can be subject to over-interpretation. An extradural haematoma is almost invariably on the same side as the blow, but because of the distortion of the skull described earlier, the fracture that traverses the meningeal artery can be some distance from the point of impact. Using Gurdjian's work, however, a reasonable estimate of the zone can be offered.

Where subdural or subarachnoid bleeding is concerned, it can be misleading to offer dogmatic opinions about the nature and position of the impact, unless there is scalp or skull damage. Though sometimes it is clear from the presence of a scalp injury that the haemorrhage is either 'coup' or 'contrecoup', there are so many exceptions when the bleeding is unrelated to the site of the blow, that a firm opinion is not justified.

RUPTURED BERRY ANEURYSM AND TRAUMA

A major cause of medico-legal problems is the association of trauma and subarachnoid haemorrhage from a ruptured 'berry' aneurysm. The pathology of the latter is described in another chapter, but here we are concerned with the allegations that an injury led to rupture of a pre-existing aneurysm.

Several variations of this scenario exist. Most commonly, an assault is rapidly followed by the signs of subarachnoid bleeding and subsequently death. Occasionally death may be extremely rapid, as previously described. The question of causation then arises, the prosecution claiming that the head injury mechanically ruptured the aneurysm. Whether this is actually so is virtually beyond absolute proof. Aneurysms of the circle of Willis are remote from the skull surface and lie deeply protected under the buffer provided by the mass of the brain. However, it is hard to deny that a heavy blow to the head, jaw or neck could not rupture, split or weaken the fragile wall of a large, thin-walled aneurysm, but when the bleeding comes from a tiny sessile bulge the evidence is not so convincing.

The complicating factor is that most assaults occur in 'fight or flight' conditions in which both aggressor and victim are physically and emotionally active, so that the adrenal response is likely to be present. Muscle tone, heart rate and blood pressure are increased by catecholamines, and it is likely that raised internal blood pressure in a weak aneurysm is a far more potent reason for rupture than a blow on the head.

A second possibility is that a person with an already spontaneously leaking aneurysm may have a rapidly developing neurological or even behavioural abnormality that leads him into conflict with another person, or into a dangerous physical position, such as a fall or traffic accident. If all this occurs over a short period of time, autopsy may not be able to distinguish the sequence of events and the aneurysmal rupture may be blamed on the trauma instead of the reverse. This may have profound civil as well as criminal legal consequences.

SUBARACHNOID HAEMORRHAGE AND ALCOHOL

A factor that is often said to increase the chance of rupture is alcohol, though there is no objective proof of this claim. A high blood alcohol is said to facilitate bursting of an aneurysm because it dilates cerebral blood vessels, increases cerebral blood flow and raises blood pressure.

The latter is not true because alcohol does not raise systolic pressure, though the pulse pressure – the difference between systolic and diastolic – may widen. The fibrous wall of an aneurysm is incapable of dilating, neither can the major basal arteries do so to any appreciable degree, as they possess little muscle in their walls. The pharmacological evidence that alcohol has any significant effect on the cerebral circulation is extremely weak. There is no evidence that alcohol is associated with completely natural subarachnoid haemorrhage from a ruptured aneurysm, though intense physical activity, such as sport or coitus, certainly does predispose to rupture.

Where alcohol and ruptured berry aneurysm are concerned, a more likely explanation is that the association is coincidental – as most altercations resulting in 'fight, fight and fistcuffs' are catalysed by alcohol. Many of the cases of ruptured aneurysm and violence occur within and on the pavements outside bars and clubs, where a high blood alcohol is virtually inevitable – but not necessarily causative.

The other aspect of alcohol is that it may cause or contribute to unsteadiness, a fall or some other traumatic event, which itself might lead to rupture of a fragile aneurysm. Ataxia and hypotonia are a feature of acute

drunkenness and, together with the aggressive behaviour and alcoholic environment of most physical violence, seem sufficient to account for their circumstantial association.

THE RAPIDITY OF DEATH IN SUBARACHNOID HAEMORRHAGE

The immediacy of death is sometimes surprising, especially in association with trauma. It is general clinical experience that the victim of a spontaneous rupture of a berry aneurysm suffers a severe headache, neck stiffness and vomiting, which may resolve or which may progress over hours or days to coma and death. Sudden death is certainly the exception in these clinical cases – but these are from a hospital population, which, by definition, excludes the rapid deaths that reach the mortuary rather than the wards.

In forensic practice, much more rapid demise is not uncommon. For example, the author (BK) recollects that as two men involved in a violent argument in a public house emerged through the outer doors, one struck the other a heavy blow on the head. The victim fell to the ground and never moved again, being certified dead a few minutes later. At autopsy, a typical fresh subarachnoid bleed from a ruptured aneurysm was displayed.

The amount of blood present in most subarachnoid haemorrhages, from whatever cause, often seems insufficient to constitute a space-occupying lesion, especially as, unlike subdural bleeding, it is able to diffuse more widely over the brain and displace cerebrospinal fluid into the spinal theca. The total volume may be considerable, however, and where survival for some hours has occurred, then the typical appearances of raised intracranial pressure may be seen at autopsy, though some of this may be contributed by progressive cerebral oedema.

Most rapid deaths exhibit substantial bleeding into the basal cisterns at autopsy, the brainstem and cranial nerve roots being bathed in a thick layer of blood and clot. As blood in the subarachnoid space seems irritant even in small quantities, it seems possible that such sudden irrigation of the medulla may lead to a rapid cardiorespiratory failure. Where the haemorrhage appears less extensive, the mechanism of death is more obscure.

Crompton points out the sensitivity of cerebral arterioles during surgical operations, when a slight touch upon the cortex leads to blanching spasm of the vessels. Subarachnoid bleeding is undoubtedly irritant and a possible reason for the sudden collapse and death is a widespread vascular spasm, which may have an effect on vital centres in the brainstem.

ROTATIONAL TRAUMA TO THE HEAD AND UPPER NECK: BASILOVERTEBRAL ARTERY INJURY

During the last few years it has been recognized that blows to the side of the neck and/or head can give rise to fatal subarachnoid bleeding. This has been attributed to tearing or dissection of a vertebral artery, allowing blood to track along the upper part of the vessel and enter the cranial cavity where the artery penetrates the dural membrane at the foramen magnum.

The evolution of this concept of traumatic subarachnoid bleeding is interesting. Originally, it was thought that most cases had a fracture of the transverse process of the first cervical vertebra, thus damaging the artery contained in the tunnel-like foramen in this bone – indeed, it was often called the 'CV-One syndrome'.

The hypothesis was that a blow to the side of the neck caused deep injury, usually manifested by skin bruising and bleeding into the muscles deep in the upper neck. This same injury was considered to fracture the transverse process and lead to tearing or dissection of the wall of the contained vertebral artery; blood then tracked upwards and medially to penetrate the dura and emerge inside the subarachnoid space of the posterior cranial fossa to cause a fatal haemorrhage.

Soon cases were being described with no such atlas vertebra fracture, but it was still accepted that mechanical forces, especially of a tilting and rotational nature, could damage the vertebral artery within the foramina of the upper cervical spine and lead to the same dissecting lesion, and hence to fatal subarachnoid haemorrhage. Until very recently, this was the theory – which is still strongly held by many pathologists – that satisfied the aetiological needs of this injury.

However, though it is likely that this classical mechanism does account for some of the deaths, there are several problems with universal acceptance of the hypothesis:

- ☒ Many pathologists – including the authors – find it hard to believe that a tiny dissection of a small artery like the vertebral artery – a lesion which often needs serial microscopic sections to confirm – could allow the torrent of blood necessary to enter the cranial cavity and produce a massive haemorrhage (sometimes well in excess of 100 ml) which may cause death within minutes.
- ☒ Several instances of a damaged vertebral vein have been reported in association with subarachnoid haemorrhage. Because of the very much lower intravascular pressure in a vein compared with an artery, the possibility of this causing a massive intracranial bleed by percolation

through the dural fenestration is even less credible than in the case of the artery.

- ☒ Increasing numbers of cases are being described with positive evidence of a blow on the side of the neck or head and a large fatal subarachnoid haemorrhage, but with completely intact vertebral arteries.

Thus, if sudden tilting and rotational forces acting on the upper spine and head can lead to subarachnoid haemorrhage without damage to the vertebral vessels, then the causative link is destroyed. Even where vertebral damage is demonstrable, this may be merely a **concomitant** event, the mechanical forces which primarily led to subarachnoid haemorrhage by direct injury to intracranial vessels also having caused the vertebral artery damage. In other words, the two lesions may in some cases – or for all we know, every case – be a **parallel phenomenon**, not a **cause-and-effect** situation. There may well be a further concomitant effect, in that direct occult brain damage, manifested later by diffuse axonal injury, is the major cause of cerebral dysfunction, both the subarachnoid haemorrhage and the vertebral artery lesion (if there is one) being merely markers of a heavy impact.

In these deaths – and certainly where no lesion at all is discernible in the upper spinal vessels – we need to look elsewhere for an explanation of the subarachnoid bleeding.

Occasionally, this is quite obvious and is situated in the intracranial vertebral vessels. For example, the author (BK) has seen a wide split in the wall of a vertebral artery **within** the posterior cranial fossa, and another where the vertebral artery was totally avulsed and transected at the internal side of the dural perforation. Both these cases arose as a result of trauma to the side of the neck, and both had a massive subarachnoid haemorrhage with no damage to vessels outside the cranium.

Bostrom, Helander and Lindgren (1992) published details of two cases of rupture of the posterior inferior cerebellar artery due to blunt basal head trauma, and proposed that the term 'traumatic subarachnoid haemorrhage' be abandoned and replaced by the nature and localization of the bleeding site.

The demonstration of intracranial bleeding from the vertebral system is difficult, as the very process of opening the skull at autopsy and removing the brain, however carefully performed, inevitably causes vascular damage. These artefacts cannot be distinguished from original bleeding points due to ante-mortem trauma.

The use of post-mortem angiography by the injection of radio-opaque contrast medium into the lower vertebral and carotid arteries has not lived up to its original claims. There is almost always diffuse leakage of contrast medium from apparently artefactual defects, and localization of true

bleeding points is often obscured by a haze of contrast opacity. However, occasionally the method reveals a specific leak, usually within the posterior cranial fossa.

The original descriptions of and claims for vertebral artery damage as the cause of traumatic subarachnoid haemorrhage were published by Cameron and Mant (1972), Coast and Gee (1984), Contostavlos (1971, 1995), Simonsen (1967, 1976) and others. Leadbeater (1994) drew attention to the problems of a universal cause-and-effect relationship; Contostavlos (1995) refuted these claims, but did not satisfactorily dispose of the concomitant hypothesis. Pathologists, both from their dissection experience and reading of the literature must make up their own minds on the matter, but should make a critical evaluation of the practicality of a massive subarachnoid bleed appearing, sometimes within minutes, from a tiny dissection of a small artery outside the dural membrane.

As many pathologists still adhere to the original concept of a subcranial vertebral artery causation for traumatic subarachnoid haemorrhage (and in a small number of instances, the evidence is persuasive), the topic is further pursued here, with the over-riding qualification that most traumatic massive meningeal bleeds are due to intracranial causes consequent upon a tilting, rotatory impact upon the head and/or neck, with or without concomitant damage to a vertebral vessel.

Forensic anatomy

The two vertebral arteries arise from each subclavian artery in the region behind the sternoclavicular joints. Each artery ascends behind the common carotid to reach the transverse process of the sixth cervical vertebra. It enters the foramen in that process and passes upwards through each similar foramen until it emerges from the upper edge of the second (axis) vertebrae. The artery then bends laterally and enters the final foramen in the atlas vertebrae. Emerging on the upper surface, the artery bends back and medially around the superior articular process, and penetrates the posterolateral aspect of the adanto-occipital membrane and the underlying spinal dura and arachnoid, emerging on the lateral side of the spinal canal just below the foramen magnum. Both arteries then ascend and converge on the ventral surface of the medulla and pons to fuse in the midline to become the basilar artery. Each vertebral artery is often of markedly different size.

Autopsy appearances

With the caveats expressed earlier, the possibility of vertebral artery trauma, concomitant or otherwise, should always be borne in mind when an external bruise is seen on the

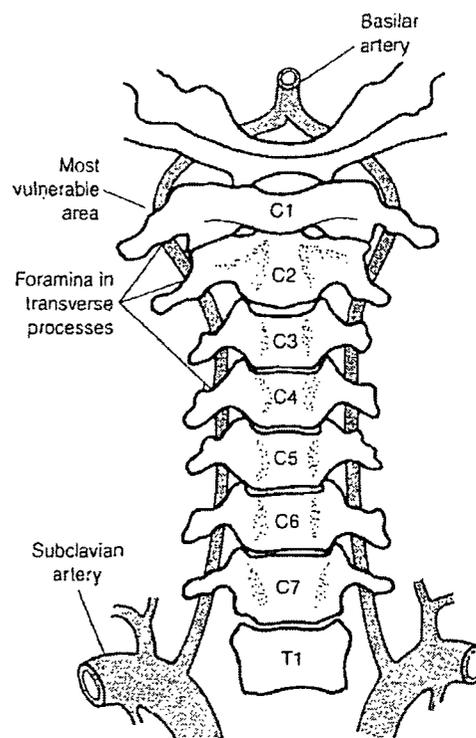


FIGURE 5.38 The origin and course of the vertebral arteries. They arise from the subclavian arteries at the level of the sternoclavicular joints and ascent via the foramina in the transverse process.

side of the neck of the victim of a fatal assault. A blow from a fist, foot or blunt weapon may land in the region between the angle of the jaw to the side of the back of the neck, the area below the ear being the most common place to find an injury.

There may be no external sign at all, but on dissection of the neck, a subcutaneous or deep bruise may be found. Unfortunately, this is an area that is not routinely dissected at autopsy, the usual incision for the removal of the neck organs being too far anterior to reveal many of these injuries, which usually lie in the strong neck muscles. This is probably why the syndrome was not recognized until a few years ago – and also an explanation why so many subarachnoid haemorrhages were said to be due to a berry aneurysm that could not be found because the bursting had destroyed it. No doubt a number of subarachnoid bleeds are due to rupture of non-aneurysmal vessels or to an aneurysm too small to find, but equally, unrecognized head trauma, with or without vertebral artery damage, must have accounted for some of the remainder.

In some autopsies, there is no external neck injury and the cause of death is unknown until the cranium is opened,

and a fresh subarachnoid haemorrhage discovered. A decision then needs to be made whether to remove the brain to search for a ruptured berry aneurysm – or begin specialized techniques to seek a rupture of a vertebral artery (described below). If there is a history of assault or other trauma, as opposed to a presumed natural death, then particular methods should be employed. The two avenues are not mutually exclusive, as long as the pathologist is aware of his objectives. The vessels can be clamped off at the base of the brain and the brain removed for a search for an aneurysm or other bleeding-point without spoiling the other techniques.

Mechanism of vertebral artery trauma

When a head is rapidly rotated by a blow that lands at the junction of the head and the neck, there may be a sudden lateral rocking (tilting sideways) at the atlanto-occipital joint, accompanied by rotation of the head. There may also be an element of hyperextension or hyperflexion, the whole episode forming a complex pattern of sudden abnormal movement at the atlanto-occipital junction. It may be that the unexpected impact may allow more unrestrained rotation and angulation of the head, due to absence of anticipatory muscle tensing in the large paravertebral and sternomastoid muscles; this may be exacerbated by alcoholic intoxication causing slow protective responses, as most of such episodes occur during altercations related to drinking sessions.

The mechanics are not fully understood and probably differ from case to case but, whatever the mechanism, the vertebral artery can become damaged either:

- in the canal within the first cervical vertebra (whether or not the transverse process is fractured)
- just below the axis, in the space between the transverse processes of the axis and atlas
- as it emerges from the exit of the canal in the atlas to penetrate the spinal dura just below the foramen magnum
- probably much more frequently within the subarachnoid space above the foramen magnum or even higher in its course towards the confluence with the basilar artery – and even the basilar itself. The type of damage is usually a tear or dissection of the wall of the vertebral artery.

Vertebral artery damage was originally said to allow blood to track under arterial pressure within the adventitia and to appear in the subarachnoid space after the vessel has penetrated the dura and arachnoid, though, as discussed above, the likelihood seems slight of these minute lesions

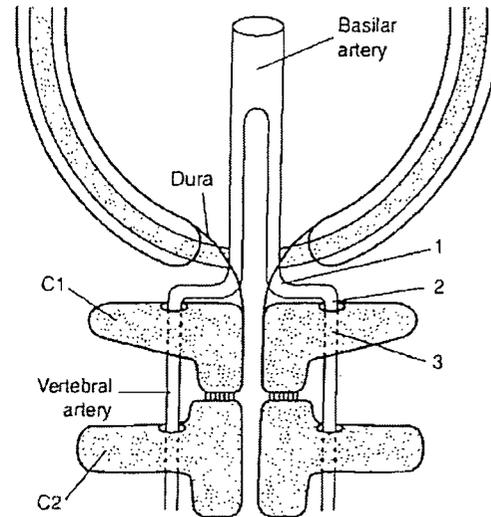


FIGURE 5.39 *Most vulnerable points for trauma to the vertebral arteries: (1) at penetration of dura; (2) at exit from atlas transverse process; (3) in osseous canal in atlas.*

allowing the large volume of blood seen in most subarachnoid haemorrhages to flow through the small periarterial window in the dura.

Autopsy demonstration of basillovertebral artery damage

When circumstantial evidence suggests a subarachnoid haemorrhage following trauma or where a bruise is seen on the side of the neck, vertebral artery damage should be suspected, then confirmed or eliminated. The first intimation that it may have occurred may be when the skull-cap is removed and a subarachnoid haemorrhage is discovered.

If the view is taken that most subarachnoid haemorrhage following upper neck trauma is due to intracranial vascular damage, then logically it is unnecessary to use time-consuming and laborious procedures which slow up the completion of the case. In many such deaths, there will be no lesion in the upper cervical spine or extracranial vertebral arteries and even if there is, it is likely to be a concomitant lesion, occurring synchronously, but with no cause-effect relationship with the intracranial vessel rupture.

However, there may be academic satisfaction in demonstrating the concomitant neck lesion, even if it played no role in producing the subarachnoid haemorrhage. In such a case, when the deceased has been in a fight or had some violence applied to the side of his neck, the same routine should be employed.

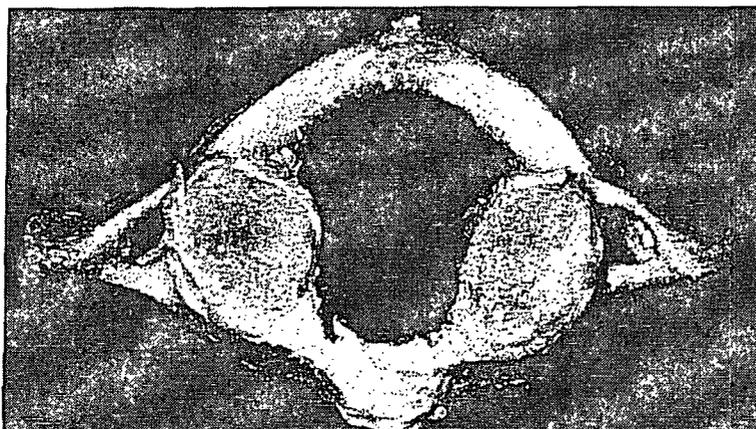


FIGURE 5.40 *The first cervical ('atlas') vertebra viewed from above to show the foramina in the transverse processes through which the vertebral arteries pass.*

Different pathologists have different procedures, but the following would be a reasonable method of investigation:

- Radiographs of the upper cervical region, both anteroposterior and lateral, should be taken as these may (rarely) reveal a fracture of the transverse process of the atlas vertebra. Such a fracture is, however, present in only a minority of vertebral artery injuries – and even when present, straight X-rays may not reveal it.
- Post-mortem angiograms should be taken if facilities exist. There are several methods of performing these. The lower neck is carefully dissected to reach the origins of the vertebral arteries. The best method is to open the subclavian vessels and identify the ostia of the vertebrals, which are the first (most medial) branches of the subclavian arteries. These can be cannulated and contrast media injected, one side at a time, while cervical and skull radiographs are taken. This method tends to fill many intracranial vessels and produce a confused, blurred picture.

More satisfactorily, the brain should be carefully lifted from the skull and, as soon as the basilar artery is accessible, it should be clamped off with surgical forceps and/or ligated and the brain completely removed as usual after the basilar artery has been transected just above the clamp. Alternatively, each vertebral artery can be clamped or ligated in its terminal course. The angiogram is now performed, either by perfusing up one vessel and forcing contrast medium back down the other via the clamped basilar, or by consecutive injections on either side. Only sufficient medium to fill the system should be introduced to avoid spillage from small branches which obscure the films. The object is to detect any significant leakage from a vertebral vessel in its upper course, usually within the foramen of the atlas or just outside the atlanto-occipital membrane or within the spinal theca.

- As by definition, there will have been a substantial subarachnoid haemorrhage, this sometimes makes it difficult to identify and ligate the basilar or vertebral vessels. The brain should be minutely examined for berry aneurysms or other vascular malformations as, if they are present (and ruptured), then further investigation of vertebral artery damage is pointless. Assuming no such bleeding point is found, the upper cervical region is dissected to determine whether the vertebrals have been injured. When no X-ray or angiographic facilities exist, this is the only method available.

The upper cervical spine should be exposed by a posterior approach, which is continued up to meet the transverse scalp incision. The spine should be freed from the surrounding muscles, taking care to detect and record any muscular bruising in the vicinity. It should then be sawn through at about C4 level or lower.

The occipital bone should then be cut through on each side from the transverse skull saw-cut and prolonged down on each side of the foramen magnum, and then across the clivus to release the central part of the floor of the posterior fossa, carrying the foramen magnum and attached upper spine. Alternatively, using a power saw with a wedge-shaped blade, a square may be cut out of the floor of the posterior fossa to detach the spinal block. If possible, X-rays should be taken of the detached portion, as it may be possible to see a fracture of the transverse process of the atlas better than in the intact body.

The block of bone removed should then be decalcified by prolonged immersion in a large volume of 10 per cent formic acid.

After a week, the spine will be soft enough to slice with a scalpel, so that the lateral parts of the transverse processes can be shaved away, taking care not to get near the arterial foramina. The block is then returned to fresh decalcifying fluid for a further week.

Once the bone has been softened enough to be cut easily by a sharp knife, the transverse processes can be further shaved down on each side to the level of the foramina, so exposing the artery in its whole course. Both sides should be so dissected, the last stages being taken deeper and deeper with extreme care to remove the lateral walls of the canals without damaging the underlying vessel. Naturally, the crucial area is in the upper one or two vertebrae, but though laborious, this method provides the most elegant and convincing demonstration.

Specimens should be taken of any obvious or suspicious breach in the wall for histological examination. The usual type of damage is either a frank tear of the intima and media, or a dissection that allows blood to track through the adventitia.

The papers by Vanezis, Simonsen, and Cameron and Mant, among others, should be consulted for further details of the recommended procedures, but the above is a practical approach to demonstrating vertebral artery lesions. Once again, the relationship must be assessed of any tiny dissection, to the volume of blood found in the cranial meninges – and a decision made as whether this is more likely to be a concomitant injury to intracranial vessels rather than a solitary external vertebral artery lesion.

HEAD INJURIES IN BOXERS

It has become increasingly apparent in recent years that persons who indulge in boxing are at risk of both acute and chronic damage to their brains. In some countries, including parts of Scandinavia, both professional and amateur boxing is banned for this reason. The acute injuries are less common but occur during or soon after the fight itself. A number of boxers have died in the ring or after removal to hospital and, in these fatalities, by far the most common lesion is a subdural haemorrhage. Extradural bleeding almost never occurs, because boxing injuries rarely cause skull fractures. Occasionally a subarachnoid haemorrhage may occur in rare cases where a berry aneurysm is present.

Much attention has been paid to the chronic changes in boxers' brains, which are very common and give rise to what is generally known as the 'punch-drunk' syndrome. It appears that the length of time for which a boxer has been involved in fighting is more important than the number of serious traumatic events he has suffered. It is a cumulative process and many episodes of minor head injury add up to produce the typical lesions described in the now extensive publications on the subject.

There are both anatomical and microscopic lesions in such brains, both in professionals and amateurs exposed over a number of years. The clinical symptoms do not concern

the pathologist, but they are explained by the morphological abnormalities. Grossly affected brains may show some cortical atrophy and slight hydrocephalus; the septum pellucidum is characteristically perforated with enlargement of the cavum and tearing of the septal leaves. The fornices and adjacent corpus callosum may be thinned or torn, and throughout the brain substance there may be scars and patches of gliosis. Neurones are lost from the cerebellum and the substantia nigra, the latter often losing pigment.

Another change that has intrigued neuropathologists is the development of an Alzheimer's-like condition, with neurofibrillary tangles throughout the cerebral cortex and brainstem, though no senile plaques are present.

CEREBRAL INJURIES

Though bleeding or infected scalp injuries, depressed fractures, meningitis and substantial meningeal haemorrhage can themselves cause death, in most fatal head injuries it is damage to the substance of the brain itself that is lethal.

In medico-legal practice it is sometimes difficult to convince lawyers that, in most instances, a victim does not die of a simple fractured skull, but that the fracture is evidence only of a substantial head injury, being a 'marker' for concomitant brain damage, which was the real lethal lesion. Similar problems of communication exist with a fracture of the hyoid bone in strangulation, lay persons often being under the misapprehension that a broken hyoid is a mortal injury, instead of merely being an inconstant marker of pressure on the neck.

The neuropathology of brain damage is a large and complex subject, the more subtle varieties requiring both specialist techniques for demonstration and expert knowledge for interpretation. The textbooks of Graham, Adams and Leestma are recommended for detailed description and discussion of cerebral trauma.

The mechanisms of production of some traumatic lesions are matters of conflicting theories, but the forensic pathologist still has to be aware of the general principles of causation in order to offer some interpretation of the injuries.

As always, caution has to be employed, as both the pathological and clinical manifestations of a head injury may appear to be at variance with the degree of force applied to the head. There is a wide range of lesions from a given insult to the head and it is dangerous to be too dogmatic in theorizing about the magnitude of an injury that gave rise to the demonstrable lesions.

It is similarly unwise to hypothesize too firmly about what clinical features must have occurred when the head injury was sustained – for example, quite severe head

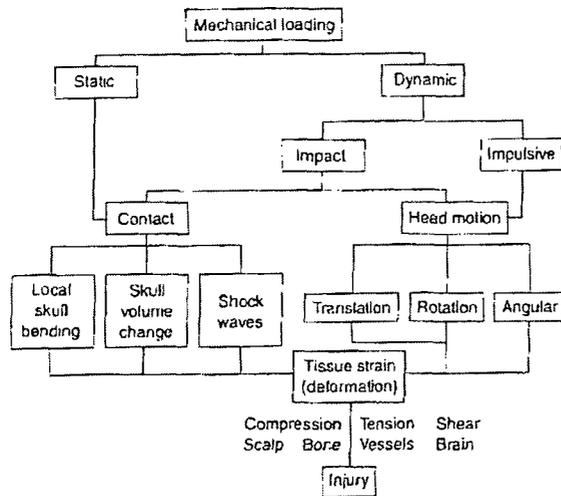


FIGURE 5.41 Mechanical events that contribute to primary brain injury. (Reproduced from Greenfield's *Neuropathology*, 7th edn, 2002 by kind permission of Arnold.)

injuries have been known to be unaccompanied by concussion, while other apparently slight damage has often been followed by prolonged unconsciousness even ending in death. The well-known aphorism of Munro (1938) must be kept in mind that: 'Any type of head injury can give rise to any type of intracranial damage.'

The mechanism of brain damage

The brain may be injured in the following ways:

- ☒ by direct intrusion, either by a foreign object such as a penetrating weapon, bullet or other missile – or fragments of skull in a compound fracture where the skull is disrupted. In these open wounds the mechanism of the damage to the brain is obvious, though of course it may be compounded by the second type of injury described below
- ☒ by deformation of the brain in closed head injuries. Here the mechanism of injury is complicated and variable, with several competing theories of causation that have been put forward since the eighteenth century (Ledran 1751, Morgagni 1761). A public debate was held in 1766 in Paris, where rival theories of coup and contrecoup brain damage were hotly defended at the Academy of Surgeons.

The brain is almost incompressible and purely axial impact may give rise to little or no damage. It is extremely rare, however, for an impact not to impart some rotatory

movement and it seems agreed that this component is the main culprit in causing brain damage. What is now clear is that no actual blow or fall need be suffered by the head to cause severe and even fatal brain damage. It is the change in velocity – either acceleration or deceleration – with a rotational rather than solely axial element, that leads to damage. The surface of the head need never contact any hard object or surface, though recent research indicates that the quantum of energy delivered by impact is of a far greater order of magnitude than non-impact violence, which lessens the former conviction that shaking, as in child abuse, is a common and potent mechanism in the production of intracranial damage.

In most head injuries – notably traffic accidents and falls – there is marked deceleration of the moving head on contact with a fixed surface but, in many criminal and combat injuries, the head is accelerated by a blow. In either case the initial sudden change in velocity is applied to the scalp and skull, the latter then transmitting the change to the brain via the anatomical suspensory system within the cranium. This system is slightly flexible and consists of the falx and tentorium, which divide the cranial cavity into three major compartments; these contain the two cerebral hemispheres, the cerebellum and the brainstem. When violent relative movements take place between the brain and the dura, forming the partitions of the cranium, the cerebral tissue can become damaged against both the sharp edges and the flat surface of these membranes. In addition, vessels traversing the subdural and subarachnoid spaces can be torn by such relative movements, especially in old people where cerebral atrophy may have widened these spaces.

Among the competing theories of impact brain damage are:

- ☒ the rotational shear force theory
- ☒ the pressure gradient theory
- ☒ the vibration theory
- ☒ the transmitted wave force theory
- ☒ the brain displacement theory
- ☒ the skull deformation theory.

These hypotheses overlap and most are correct in some aspect. Because experiments on primates and mechanical models have been pursued vigorously in recent years, it is now widely accepted that there are marked pressure changes within the cranium on impact (Yanagida *et al.* 1989). When a head falls against the ground, pressure momentarily increases at the impact point but falls to a negative value diametrically opposite. As these suction or cavitation effects are more damaging to neural and meningeal tissue than pressure, this is good evidence for claiming that contrecoup damage is largely a result of this vacuum effect.

The actual physical disruption of cerebral tissue is caused, according to both Gurdjian and Holbourn, by one or more of the following processes:

- 1 compression of the constituent units, by their being forced together
- 2 tension of the units, which pulls them apart
- 3 sliding or 'shear' strains, which move adjacent strata of tissue laterally. The usual homely example is given of a pack of playing cards being displaced, so that each card slides upon its neighbour.

Transient deformation of the skull almost certainly contributes to brain damage (Rowbotham 1964). The area of the skull beneath an impact becomes momentarily depressed even if it does not fracture and therefore may impinge on the underlying brain causing compression, as in 1, above. This is responsible for the typical cone-shaped contusions on the cortex, with the base at the surface, as the impact – possibly via short-lived oscillations of decreasing amplitude – injures the cortex and passes a diminishing force down into the deeper layers.

Simultaneously, other areas of the skull must bulge outward to accommodate the deformation – the so-called 'struck-hoop' action – when it is suggested that a 'rarefaction' remote from the impact may cause tension damage, as in 2, above.

More important is 3, being laminar deformity or 'shear stress' caused by the angular rotation of the head. As the head is pivoted on the first cervical vertebra, almost any impact on jaw, face or cranium will produce an angular momentum, the acceleration being conveyed first to the skull.

Alternatively, if the head is moving and is suddenly arrested, then the skull will decelerate first and the momentum of the brain will cause it to continue in motion, again almost certainly with some rotatory component.

In either the deceleration or acceleration mode, the skull and brain cannot change their velocities simultaneously and the brain will speed up or slow down only by virtue of the restraint provided by the dural septa and the configuration of the interior of the skull. In other words, the brain is either retarded or set into motion secondarily by the skull, especially by the dural septa and the bony prominences.

This restraint will occur first – and with maximum effect – on the most superficial layers of the cortex. These in turn will drag on the next deeper layer and so on until the difference in velocity is equalized – but this will have been at the expense of laminar tearing of the cerebral tissue and its associated blood vessels. In addition to this shearing damage, the brain may be forced against the sharp edge of the tentorial opening and the lower edge of the falx, causing damage to the base of the cerebrum, the corpus callosum and the brainstem. Impact against the side wall of the skull

and against the falx may cause diffuse contusion of the cortex. The cerebellum tends to suffer less damage, as it is much smaller and lighter than the cerebrum and there is less room for relative movement in the more tightly enclosed posterior fossa. The configuration of the interior of the cranium is thought to be partly responsible for the common localization of cerebral damage at the tips and undersurface of the frontal and temporal lobes. The rough floor of the anterior fossa, the sharp edge of the wing of the sphenoid and the massive bar of the petrous temporal bone are in contrast to the smooth inner surface of the vault of the skull.

Cerebral contusion

When either linear or, more often, laminar stresses are applied to the cortex, this soft tissue may disrupt. Part of the injury is directly upon the neurocellular structure, but damage to vessels is an important component. If the cortex still retains its shape, but is bruised and swollen, this constitutes 'contusion'. A greater degree of disruption, sufficient to produce macroscopic tearing, is termed 'laceration', but the difference is only one of degree. In gross head injuries, such as crushing, missile wounds and other major penetration, the degree of laceration may lead to partial or even complete extrusion of the brain from the cranial cavity.

In the usual type of cortical contusion seen in a closed head injury, the cortex is blue or red from haemorrhage, though if survival has lasted for some time, there may be added discoloration from associated cortical infarction. The haemorrhage may be diffuse or may be punctate and is often a mottled purplish red when confined to the cortex. Extension into the underlying white matter tends to be pure red in fresh lesions. The lesion is often wedge-shaped, with the base on the surface, tapering away into the deeper layers.

Cerebral laceration

Laceration of the cortex is an extension in severity of contusion in which mechanical separation of the tissue can be seen. When widespread, but relatively superficial, the cortex appears to have a 'red velvet' appearance, which becomes progressively more tattered as the severity increases.

When it is even more severe, the cerebral surface becomes fissured, fragments of cortex may detach and deep lacerations run into the depths of the hemisphere, sometimes reaching even the deep ganglia or ventricles. There may be deep haemorrhage and – especially in the frontal and temporal lobes – the lacerations may be continuous with areas of traumatic haemorrhage. In cerebral lacerations and most contusions, the pia mater and often the arachnoid are torn, so that blood from damaged cortical vessels leaks into the

subarachnoid and even subdural spaces. The corpus callosum is commonly torn, especially at its posterior end. This must be distinguished from damage caused by clumsy removal of the brain at autopsy (as must tears of the cerebral peduncles), but when genuine, may represent either a guilotine effect of the free lower edge of the falx or differential lateral movement of one hemisphere relative to the other, again because of unilateral restraint by the falx. Lacerations and contusions are most often found in those areas of the brain where the cortex is most likely to come into contact with irregularities in the internal profile of the skull. The undersurface of the temporal lobes and the orbital surface of the frontal lobes suffer most often.

Traumatic intracerebral haemorrhage

Substantial areas of haemorrhage, either infiltrating the brain tissue or forming actual haematomas, are common in severe head injuries. Some are primary, occurring at the time of impact or soon afterwards; others are secondary and caused by changes in intracranial pressure or bleeding into infarcts caused by vascular damage. These are all seen more often since artificial ventilation has been available, as victims of severe head injuries now survive longer so that there is time for secondary lesions to become apparent.

In the cerebral hemispheres, deep haemorrhage can be caused by coup or contrecoup mechanisms (see below) and may be situated anywhere within the hemispheres. They may rupture into the ventricular system or through the overlying cortex. In some severe contrecoup lesions, there may be large haematomas in one or both frontal lobes with overlying cortical contusion and laceration. These may rupture through the cortex into the meningeal spaces, forming what is sometimes called a 'burst lobe'. Such haemorrhages sometimes pose a problem for the pathologist when they occur in older subjects, especially those with hypertension and perhaps cerebral atherosclerosis. When a scalp injury is present – and perhaps even a fractured skull – it may be difficult to decide if a head injury (such as a fall) was responsible for the cerebral haemorrhage, or whether a sudden 'stroke' caused by a natural cerebral haemorrhage resulted in the fall. The problem is discussed further in the chapter on natural death, but briefly, the presence of left ventricular (cardiac) hypertrophy, a history of hypertension and the site of the (usually solitary) haemorrhage tends to point to a natural bleed. This is especially so if the large size of the lesion seems inconsistent with the degree of head injury sustained. Hypertensive lesions tend to be in the thalamus, external capsule, pons and cerebellum, and are more often occipital than frontal or temporal. Having said that, on occasions it can be impossible to differentiate the two conditions.

Primary brainstem haemorrhage

Secondary brainstem bleeding is dealt with under 'cerebral oedema' so here we are concerned with haemorrhage that occurs at the time of injury. As mentioned above, hypertensive haemorrhages not associated with trauma can occur in the midbrain, especially the pons. These tend to be large, explosive lesions that greatly swell the pons and disrupt the central part of the stem, usually with a ragged rim of white matter around the periphery. Traumatic haemorrhage in the brainstem is often a well-circumscribed lesion, sometimes rounded, which lies laterally in the tegmentum, the shape of the midbrain being undistorted (unlike a secondary bleed into an elongated stem).

The typical site is between the aqueduct and the outer end of the substantia nigra. Primary stem haemorrhages are usually associated with occipital impacts and the victim is often unconscious from the time of the injury, as opposed to the lucid interval and gradual decline of those who suffer secondary stem lesions because of a developing cerebral oedema or space-occupying meningeal haematoma.

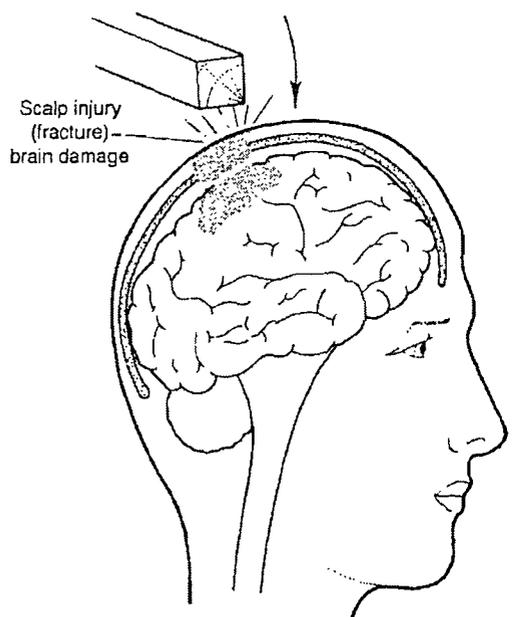
Coup and contrecoup damage

Whatever the underlying mechanics of cerebral damage, one aspect is of considerable practical importance to the pathologist. When a mobile head is struck with an object, the site of maximum cortical contusion is most likely to be beneath or at least on the same side as the blow. This is the so-called 'coup' lesion. When a moving head is suddenly decelerated, as in a fall, though there might still be a 'coup' lesion at the site of impact, there is often cortical damage on the opposite side of the brain – the 'contrecoup' lesion.

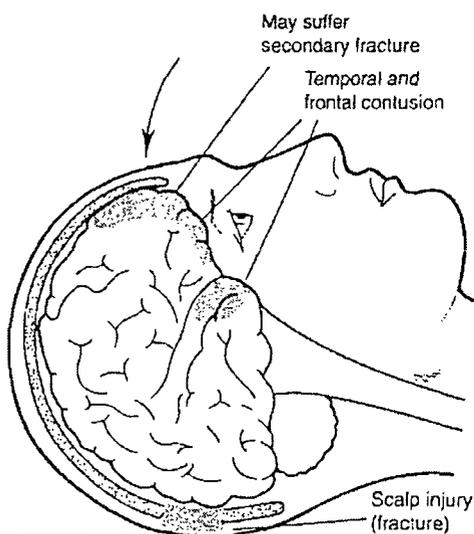
The mechanism of the 'coup' and 'contrecoup' injuries has long been debated – at least since the time of the famous Paris meeting of 1766. The controversy has been continued, especially by Courville (1942) and by Holbourn (1943), but no satisfactory resolution has been agreed though the work on intracranial pressures by Yanagida *et al.* seems to provide proof that a 'vacuum' occurs at the contrecoup site.

The following practical points should be considered:

- There may be no coup damage at all, only contrecoup.
- There need be no fracture of skull, even in the presence of severe coup and contrecoup lesions.
- The most common site for contrecoup injury is in the frontal and temporal lobes. It is often at the tips and on the undersurface of these lobes, and may be symmetrical, if a fall on the occiput has occurred.
- In temporal or parietal impacts, the contrecoup lesions are likely to be diametrically opposite on the



(a)



(b)

FIGURE 5.42 (a) 'Coup' brain damage to fixed head. (b) 'Contrecoup' brain damage to moving (decelerated) head.

contralateral surface of the brain, but exact geometrical correspondence is not necessarily present.

- It is virtually unknown for a fall on the frontal region to produce occipital contrecoup. This is thought to be due to the anatomical configuration of the floor of the cranium, but the reasons are by no means understood.
- In a temporal impact, the contrecoup damage may be not be on the contralateral hemisphere, but on the



(a)



(b)

FIGURE 5.43 (a) Typical contrecoup injury to the tips of the frontal and temporal lobes of a brain. The victim was pushed over in a brawl and fell backwards, striking his occipus and causing a small scalp laceration (b). There was no contusion to the posterior part of the brain.

opposite side of the ipsilateral hemisphere from impact against the falx cerebri.

- The degree of contrecoup damage may be severe, sufficient to cause blood-filled cavitation in the deep cortex and underlying white matter, especially in the frontal lobes and tips of the temporal lobes.

- ☒ With severe frontal contrecoup from a fall on the occiput, the transmitted force may be sufficient to fracture the thin bone of the floor of the anterior fossa. Such cracks in the roofs of the orbits may allow meningeal haemorrhage to seep into the orbits and appear as 'black eyes'. In assaults where a fall has occurred, care must be taken not to attribute such periorbital bleeding to direct punches.
- ☒ Though contrecoup contusion is classically caused by deceleration of a falling head, it can also occur when a fixed head is struck. If the victim is already lying on the ground or against some other unyielding surface, a heavy blow on the upper side may cause typical contrecoup lesions either in the contralateral temporal or parietal cortex, or against the falx on the inner side of the ipsilateral lobe. In these circumstances, there is often coup damage as well. No external scalp injury need be visible.
- ☒ The interpretation of contrecoup lesions is most reliable in the form of cortical contusions or lacerations. Meningeal haemorrhage, either subdural or subarachnoid, may also arise in association with a contrecoup lesion, but its diagnostic value is virtually nil compared with cortical damage when interpreting a falling or fixed head injury. Where no associated cortical contusion is present, it is quite unsafe to rely upon a unilateral meningeal haemorrhage as an indicator of the type of head injury.

Concussion

Concussion is a clinical, not a pathological entity, but the pathologist must consider it, as it is related to intracranial lesions and he is often questioned about it in court proceedings. Concussion, according to Wilson (1946), is 'a disorder of cerebral function which follows immediately upon the impact of a force to the head'. A more full definition is offered by Trotter (1914): 'a transient paralytic state due to head injury which is of instantaneous onset, does not show any evidence of structural cerebral injury and is always followed by amnesia from the actual moment of the accident'.

Some neurologists would also include post-concussion symptoms within the definition of concussion, even in the absence of initial coma, following a head injury. There may also be evidence of depressed medullary function, which can affect cardiorespiratory action. Denny-Brown and Russell (1941) showed that the rate of change of velocity of the head was important in producing concussion, which rarely developed if the speed threshold was less than ~ 8.5 m/s (28 ft/s).

It is an extremely common, but not inevitable, sequel to any significant mechanical insult to the brain. Though in general terms its duration is loosely related to the severity of the injury, there are many exceptions. Gross skull and brain damage have occurred with little or no apparent concussion, though concussion may be so transient that the subject may not even fall to the ground. Relatively minor head injuries have given rise to prolonged unconsciousness so, once again, it is most unwise to be dogmatic about retrospective estimates of concussion.

There is considerable controversy about the cause of concussion, from the unacceptable 'traumatic neurosis' on the one hand (which cannot be true) to claims for the inevitable demonstration of physical lesions on the other.

Courville (1953) has discussed the condition in depth and there seems to be no reason to doubt that some mechanical process does temporarily disrupt the function, if not necessarily the structure, of the neuronics apparatus. Changes in the nucleus and cytoplasm of neurones, the composition of the cerebrospinal fluid and in the electroencephalograph have all been inconstantly reported (see 'Diffuse neuronal and axonal injury' below).

True concussion may last for seconds or minutes. If prolonged unconsciousness extends into hours, days or longer, then there is likely to be some structural brain damage. Occasionally what appears to be simple concussion proves to be fatal, causing respiratory paralysis, though at autopsy no significant lesions are found.

Where a victim of 'simple' concussion dies of some incidental non-neurological condition, autopsy usually reveals no macroscopic damage, though sometimes there is slight cerebral oedema and scattered non-specific petechial haemorrhages may be found. There seems to be a connection between concussion and rotatory movements of the head, which are usually responsible for obvious structural damage because, when a head is fixed before impact, loss of consciousness may not occur. The classic example is trapping of a head against a wall or being jammed between buffers.

That shear stresses are instrumental in causing neurone damage seems confirmed by the frequency in which concussion occurs in boxing contests, where a blow on the jaw is the ultimate in producing a rotational movement of the cranium.

Concussion may be followed by a 'post-concussion state' characterized by headaches, unsteadiness and anxiety. This seems a genuine phenomenon, though it has been pointed out that it may be overlain by a 'compensation syndrome' whilst civil litigation is in progress over responsibility for the accident, which often clears up rapidly once the claim is settled.

Retrograde amnesia is almost inevitably associated with concussion, though, like concussion itself, it may be so transient as to escape notice. A protective mechanism, it seems to be caused by loss of sensory input before the latter is transferred to permanent memory storage in the brain.

Though commonly only of minutes' duration, it can extend to several days before the head injury. Though there is often a later recovery of much of this lost period, the memory of events immediately before the incident rarely returns, which may fortuitously be a protective device. Concussion has been attributed to several causes, including the undoubted vasomotor disturbances that take place after a head injury. Another theory is the impaction of the brain into the foramen magnum or tentorial opening, but the most acceptable hypothesis is 'diffuse neuronal injury'.

DIFFUSE NEURONAL AND AXONAL INJURY

According to Graham *et al.* (2002), diffuse brain damage exists in four principal forms: diffuse vascular injury, diffuse axonal injury, hypoxic brain damage and diffuse brain swelling. Diffuse vascular injury consists of multiple small haemorrhages throughout the brain and is virtually restricted to patients, who die within 24 h, whereas the latter three are encountered in patients who survive long enough to reach the hospital. Recent research and new immunohistochemical methods have shown that traumatic axonal injury is much more common than previously realized, and that axons can also be diffusely damaged by other processes than head injury. Therefore, Geddes *et al.* (2000) suggested new definitions of the terminology to get rid of the incoherent use of terms in the literature:

- Axonal injury (AI) is a non-specific term referring to damage to axons of any aetiology.
- Traumatic axonal injury (TAI), is a damage to axons caused by trauma, which may vary from small foci of axons to more widespread brain damage, diffuse TAI is the most severe form of traumatic axonal damage (originally termed 'DAI').
- Diffuse axonal damage (DAI), first described as a clinicopathological syndrome of widespread axonal damage throughout the brain, including the brainstem, should not be used as a term without reference to the aetiology, because axonal injury may be caused by other pathological processes.

Experimental work has suggested that diffuse axonal injury is primarily a non-impact rotational acceleration-deceleration phenomenon, deformation by stretching probably being the most significant factor. A low level of injury causes transient changes in the permeability of the axolemma, gradually leading to ionic changes, accumulation of fluid and axonal swelling and eventually, with an increasing grade of injury, to intracellular Ca^{2+} accumulation, proteolysis and collapse of the cytoskeleton.

The disruption of axons leads to bulbous and clubbed 'retraction balls or globes' on the axons in the cerebral hemispheres, cerebellum and brainstem. Disturbance of axonal transport causes accumulation of substances in damaged fibres, that can be demonstrated immunohistochemically. At present, β -amyloid precursor protein (β APP) is considered to be the most reliable indicator of axonal damage, revealing axonal injury within 2–3 hours of the insult, whereas it takes about 12–18 hours for axonal bulbs to become visible on routine or silver stains. However, one should keep in mind that β APP is not specific to head injury but is an indicator of derangement of fast axonal transport, which has also been demonstrated in other pathological conditions.

It is preferable to examine the brain after proper fixation, usually after 10–14 days in 4 per cent buffered formaldehyde and, where possible, consultation with a neuropathologist with experience in forensic practice. Due to the diffuse nature of the axonal injury, extensive and systematic sampling is essential, as the diagnostic yield is directly related to the number of blocks taken. In addition to any obvious focal pathology, the minimum set of samples should include corpus callosum and parasagittal posterior frontal white matter, splenium of the corpus callosum, deep grey matter to include posterior limb of the internal capsule, cerebellar hemisphere, midbrain including the decussation of superior cerebellar peduncle, and pons including superior or middle cerebellar peduncles. In addition to these, it may also be advisable to include corpus callosum and parasagittal anterior frontal white matter and temporal lobe including hippocampus (Geddes *et al.* 2000).

Retraction globes are most numerous in the corpus callosum, the superior peduncles, the parasagittal white matter, the medial lemnisci and the corticospinal tracts, but can be seen anywhere in the white matter. The retraction globes or axonal bulbs have been shown to lose positive staining for β APP after about a week but to persist in adjacent varicose axons up to 30 days. After some weeks in those who survive long enough, clusters of microglia, presumably part of a repair process, congregate at the site of ruptured axons. These are best seen in 20 μm thicker sections stained with cresyl violet. One problem with the microscopic evidence of both microglial clusters as well as diffuse axonal injury (as demonstrated by β APP expression), is that neither of them is specific as a marker for trauma: retraction globes may also be seen around the periphery of natural lesions such as cerebral infarcts and haemorrhages and microglial clusters have been reported in viral/HIV encephalitis, previous global hypoxia and fat embolism. Geddes *et al.* (2000) conclude in their excellent review that 'the demonstration of traumatic axonal damage is likely to be of limited use in most forensic situations, except perhaps to confirm that there has been a head injury'.

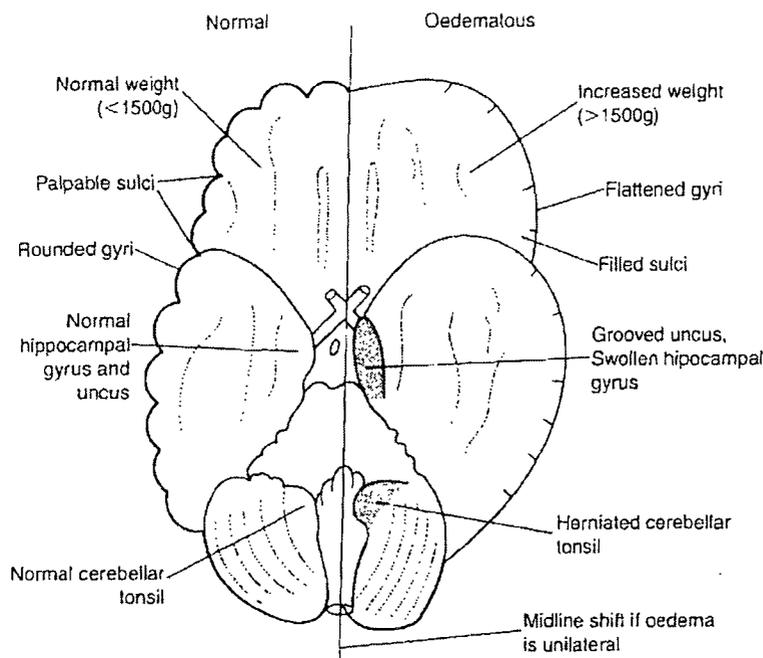


FIGURE 5.44 Signs of cerebral oedema.

Cerebral oedema

Swelling of the brain tissue may be a local phenomenon around almost any lesion, be it contusion, laceration, tumour or infarct, but here we are more concerned with generalized oedema. Swelling of the brain is extremely common after a substantial head injury, especially in children. Though it is an almost inevitable accompaniment of almost all intracerebral damage as either a local or general phenomenon, it can occur as the sole abnormality – and not infrequently prove fatal, particularly in young victims.

Oedema may well be related to diffuse neuronal injury and to concussion itself. It is the most common cause of raised intracranial pressure, being seen more often than localized space-occupying lesions such as haematomas and tumours, though of course these often coexist with cerebral oedema.

As with concussion, its cause is obscure and hence is the source of considerable controversy. The amount of fluid in the brain increases and the total weight may increase by at least 100 grams, mainly in the white matter. The site of the excess fluid is obscure, as the cut brain surface does not appear wet as do connective tissues elsewhere in an oedematous body. It was formerly thought that there was no true extracellular space in neural tissue and that the fluid must therefore be intracellular, but electron microscopic studies have revealed an extracellular compartment, which is much wider in the white matter (up to 80 nm) than in the grey matter (up to 20 nm), which explains the preference of oedema for the white matter.

The autopsy features of cerebral oedema are readily recognized. On removing the calvarium, the dura is stretched and tense, the brain bulging through the first incision in the membrane. The gyri are pale and flattened, and the sulci filled, giving the normally corrugated cerebral surface a smoothness that can easily be felt at autopsy. The cut surface is pale and, especially in children, the ventricles may be reduced to slits by the swelling of the adjacent white matter.

Severe cerebral oedema causes the larger volume cerebral hemispheres to press down upon the tentorium and herniate through the midbrain opening. The hippocampal gyrus may impact in the opening, lesser degrees causing grooving of the unci. Both these effects may lead to haemorrhage and necrosis at the sites of pressure, especially where the sharp edge of the tentorium cuts into the cerebral tissue. The tonsils of the cerebellum may be impacted or 'coned' into the foramen magnum, and sometimes are forced down into the upper part of the spinal canal. The pathologist must be careful not to mistake the normal anatomical grooving that often exists around the cerebellar tonsils for 'coning'. There should be other signs of brain swelling and true tonsillar herniation will show discoloration or even necrosis of the ischaemic, trapped tissue.

Cerebral oedema may be the only intracranial abnormality found at autopsy after a substantial head injury has occurred. This seems to be more commonly found in children and, in the absence of any other demonstrable lesions, the cause of death has to be attributed to this swelling of the brain, compressing the vital centres in the brainstem.

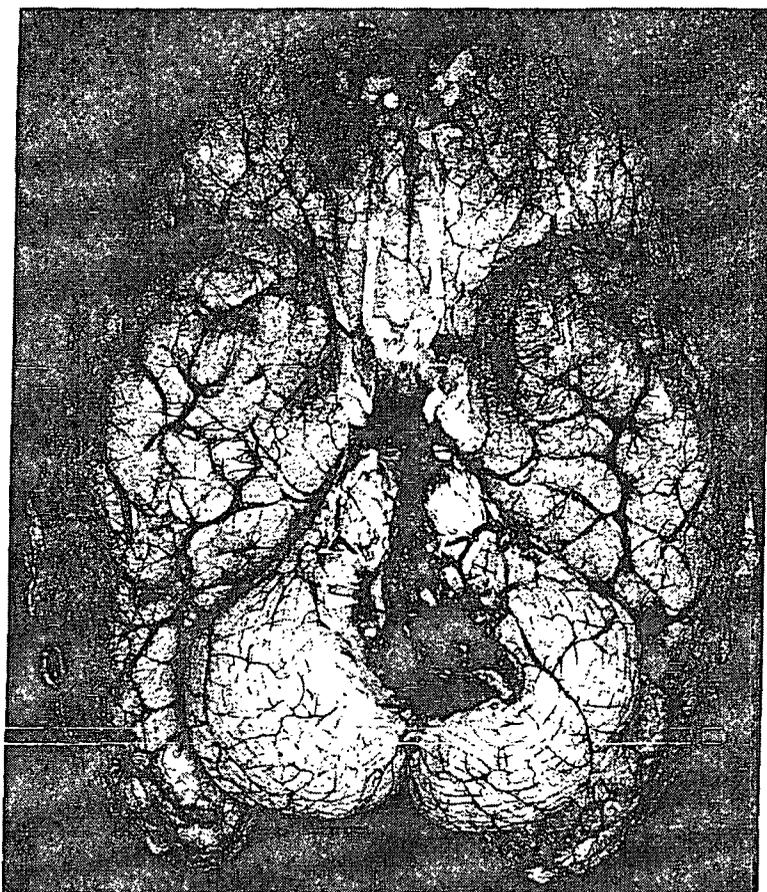


FIGURE 5.45 Tonsillar herniation as a consequence of lumbar puncture in a patient with increased intracranial pressure. The pressure gradient has forced the cerebellar tonsils into foramen magnum and caused compression of the brainstem and patient's death. (Reproduced by kind permission of Professor H Kalimo.)

Obviously, many other cases of cerebral oedema resolve either spontaneously or with treatment, so that fortunately no opportunity arises to prove its existence by post-mortem examination. Such oedema is, however, not infrequently found during surgical exploration for a meningeal haemorrhage, the latter sometimes being presented though often only brain swelling is demonstrated.

Cerebral oedema may be self-potentiating, in that once it begins as a result of direct brain trauma, the consequent rise in intracranial pressure then impairs the venous return from the intracranial sinuses. The pressure is insufficient to restrict the arterial inflow, so further congestion and swelling occur. This may lead to worsening cerebral hypoxia and oedema to the stage of actual cerebral infarction and brain death – again, a distressing common syndrome seen especially in child victims of head injuries, usually from road accidents.

In addition to mechanical damage, cerebral oedema can be caused or worsened by hypoxia. Many head injuries may be associated with damage to other parts of the body, such as the thorax – and because the airway may be compromised in many unconscious victims, there may be an element of

hypoxia in a considerable proportion of cases of cerebral oedema. Capillary permeability may be increased in a number of states, leading to weakening of the so-called 'blood-brain barrier' formed by the capillary endothelium and basement membrane, together with the astrocyte foot-plates. Cerebral oedema, either traumatic or hypoxic, can develop with surprising rapidity, especially in children. Macroscopic evidence of brain swelling can be seen at autopsy in cases where the interval between trauma or onset of hypoxia and death was less than one hour.

Several methods are available to reverse oedema, including hyperventilation, which acts by providing full oxygenation and reducing the peripheral carbon dioxide tension, thus causing constriction of arterioles and a reduction in brain volume and transudation.

HISTOLOGICAL DIAGNOSIS OF EARLY CEREBRAL HYPOXIA

Unfortunately for the pathologist, most of the hypoxic conditions seen in forensic practice cause death too quickly

for any recognizable histological changes to develop. Acute deaths from strangulation, suffocation and choking occur within minutes or less, even excepting the sudden vasovagal type of cardiac arrest.

There are, however, occasions when a longer period of survival occurs after an acute hypoxic episode and in some of these there may be the opportunity to detect histological changes in the central nervous system. Probably a minimum of 2–4 hours of survival is necessary for unequivocal changes to be observed, though some neuropathologists claim to detect signs after as little as 1 hour. More cautious investigators prefer 4 hours as the minimum. A short post-mortem interval is a considerable advantage in searching for these changes, as they are subtle and can be overlaid by post-mortem autolysis, even if the latter is slight.

It may be helpful to seek the assistance of a neuropathologist in detecting or confirming the relatively minor changes seen in a brain affected by hypoxia, but with experience, a good knowledge of the normal appearances and a laboratory that is able to turn out constantly good-quality sections and uniform staining, any forensic pathologist can build up a considerable expertise in this localized aspect of neuropathology. The parts of the brain most suitable for seeking evidence of hypoxic damage are the hippocampus (especially Sommer's area), the cerebellar folia, the globus pallidus in the basal ganglia, and the boundary zones of the cerebral cortex where the grey matter is most vulnerable because it lies at the terminal reaches of the arterial supply (for example, at the occipitotemporal junction where the watershed between middle and posterior cerebral arteries lies). The third and fourth cortical layers are best for displaying the histological changes of hypoxia.

The following is a summary of the stages and changes that may be seen using different staining methods. The brain should be prefixed by suspension in formol saline for several weeks, though if this is impossible, small blocks should be wet-cut from the target areas of the brain mentioned above and immediately fixed in a large volume of formalin.

Stage 1

The earliest change is microvacuolation in the neurone cytoplasm, which may be seen in 2–4 hours. The cell outline remains smooth and its size is normal but, internally, small vacuoles appear in the cell body, and the proximal parts of the axon and dendrites. There may be a slight eosinophilia in haematoxylin and eosin stains and a violet colour in Luxol Fast Blue.

The microvacuolar change is said to begin earlier in small neurones and last for a shorter time, being present for up to 4–6 hours after insult in large cells and 2–4 hours in cerebellar Purkinje cells, though these times are arbitrary.

Stage 2

In the next stage, more prolonged hypoxia causes distortion in the shape of the neurones, the cell body becoming shrunken and staining darker with aniline dyes. The Nissl granules become fine and are dispersed more widely throughout the cell. Some vacuoles remain, the cytoplasm shows marked eosinophilia in haematoxylin and eosin, and is bright blue to mauve in Luxol Fast Blue stain. The nucleus becomes triangular and may be placed eccentrically in the cell body. The nucleolus becomes obscured and the nucleus stains a darker blue than normal with Luxol Fast Blue. This change is seen after up to 6 hours survival or later in large cells.

Stage 3

The ischaemic cell stage, described last, becomes worse during the following day or two if survival continues. The cell shrinks further, becoming narrow and tapered in many cases. Eosinophilia and blueness when stained with Luxol Fast Blue persists, but small spherical or irregular bodies begin to adhere to the exterior of the cell membrane. These incrustations persist longer than the remnants of the cytoplasm, which vanish, leaving the bare nucleus. The dark, shrunken nucleus becomes more apparent and survives for a number of days before karyolysis occurs.

Stage 4

Homogenizing cell damage is best seen in the Purkinje cells, in which the cytoplasm is uniformly eosinophilic with no Nissl granules. The nucleus stays dark and triangular until it vanishes and leaves a pale 'ghost' of the cell body with a vague perimeter.

There are parallel changes in the glial elements as well as in the neurones. From 4 to 12 hours after a hypoxic insult there may be an increase in the number of astrocytes around damaged neurones. There is a proliferation of astrocytes (which show mitoses) some 4–6 days later if the patient survives. This is most noticeable in the cerebellum and persists for 10–14 days. Fibrous astrocytes proliferate, but not the oligodendroglia. Microglia transform into rod cells, which may lie at right angles to the edge of the cortex, radially in the hippocampus and in the molecular layer of the cerebellar cortex. A later stage of cerebral hypoxic damage is the accumulation of lipid droplets and scavenging of myelin. Groups of microglia around dead neurones (neuronophages) may be obvious even at lower powers of the microscope.

SECONDARY BRAINSTEM LESIONS

When severe brain swelling (or a space-occupying lesion such as a large subdural or extradural haematoma) causes

raised intracranial pressure above the tentorium, amongst the possible sequelae is compression of the midbrain against the free edge of the tentorium. This may be unilateral, causing grooving of a cerebral peduncle (called 'Kernohan's notch') associated with subpial petechial haemorrhages and often hemiplegia. When symmetrical, the oedema forces the undersurface of the cerebrum against the tentorium so that the hippocampal gyrus is squeezed into the opening. This elongates the midbrain in an anteroposterior direction as well as grooving the uncus and, in extreme cases, infarcting part of the parahippocampal gyrus. In addition, the distortion stretches, and compresses the paramedian and nigral vessels that supply the midbrain, leading to haemorrhages and infarcts in the upper pons and midbrain. Damage to cranial nerves and to the circulation of the cerebrospinal fluid may be added complications. The calcarine cortex on the medial aspect of the occipital lobe may be infarcted by the posterior cerebral artery being trapped around the edge of the tentorium by the cerebral herniation.

Secondary lesions are almost exclusively midline or paramedian haemorrhages, or haemorrhagic necrosis placed centrally in the upper pons and midbrain, though some may be obliquely placed in the substantia nigra. They may be difficult to distinguish from primary brainstem haemorrhages that arise at or soon after the original head injury – the secondary lesions tend to obscure the primary, rather than the converse. In children, the typical stem haemorrhages may not be present as in adults, but instead the medulla oblongata may be buckled or kinked due to fixation of the upper spinal cord by the denticulate ligaments (see work by Crompton).

SPINAL INJURIES

The spine and head should be thought of as part of the same system in relation to trauma. In recent years, a closer association – both skeletal and neurological – has been acknowledged between the two structures. For instance, interruption of ascending fibres in the cervical cord has been shown to be associated with neuronal chromatolysis in the brainstem and Spicer and Strich have shown that haemorrhage into spinal root ganglia may be associated with head injury. Electroencephalographic changes have been shown to occur in half the victims of cervical spine whiplash injury.

Though all segments of the spine are vulnerable to trauma, the cervical part holds the most interest for the forensic pathologist, mainly because of its close association with head injuries and vehicular accidents. The upper two cervical vertebrae provide most of the rotational movement of the head, whilst the lower neck allows flexion and extension. Violent force applied to the head tends to damage those parts of the neck corresponding with this functional

distinction. Spinal damage may be caused by compressional, hyperflexion and hyperextension stress.

Compression damage

This occurs when the victim falls from a height either onto his feet or his head, though in the latter case, head injuries may overshadow damage to the spine, as well as absorbing most of the impact.

When a person falls a long distance onto the feet, the kinetic energy of the deceleration may be absorbed by fractures of the feet, legs and pelvis, but can be transmitted up the spinal column. This can be fractured at one or more points, or the force may cause the upper cervical spine to impinge on the base of the skull and cause a 'ring fracture' around the foramen magnum as the spine is rammed into the posterior fossa.

A fall onto the head may also cause the 'burst atlas' injury, where the impact of the occipital condyles in an axial direction wedge the superior atlantal articulating facets apart and split the ring of the vertebra. The posterior arch can also be fractured in hyperextension by compression between the occiput and the posterior spine of the axis.

Compression fractures of vertebral bodies may occur, most commonly in the lower dorsal and upper lumbar zone, particularly T12 and L1. There is less chance of spinal cord injury compared with the angulation injuries described below, unless posterior extrusion of a disc or backward displacement of fragments of a disrupted vertebral body occurs.

Hyperflexion and hyperextension injury

Of the two, hyperextension is much more dangerous in causing spinal damage, possibly because flexion is protected by contraction of the strong posterior neck muscles, whereas the weak anterior longitudinal ligament is incapable of preserving the integrity of the cervical spine during hyperextension. In frontal or rearward motor vehicle crashes, which comprise 80 per cent of accidents, there is usually a hyperflexion and hyperextension component to the spines of the occupants, though head restraints and seatbelts restrict the range of movement.

Where a car undergoes violent frontal deceleration, the subject's head will swing down into hyperflexion and, unless restrained, will then strike the fascia or windscreen, and rebound into hyperextension.

When the vehicle is struck from the rear, the head will fly into hyperextension first unless a head-rest is available; such collisions often then smash the vehicle into the one in front, when a deceleration hyperflexion takes place.

Whatever the cause, a whole range of lesions can follow, both in the cervical, and to a lesser extent into the thoracic and lumbar segments. Bleeding into the surrounding muscles,

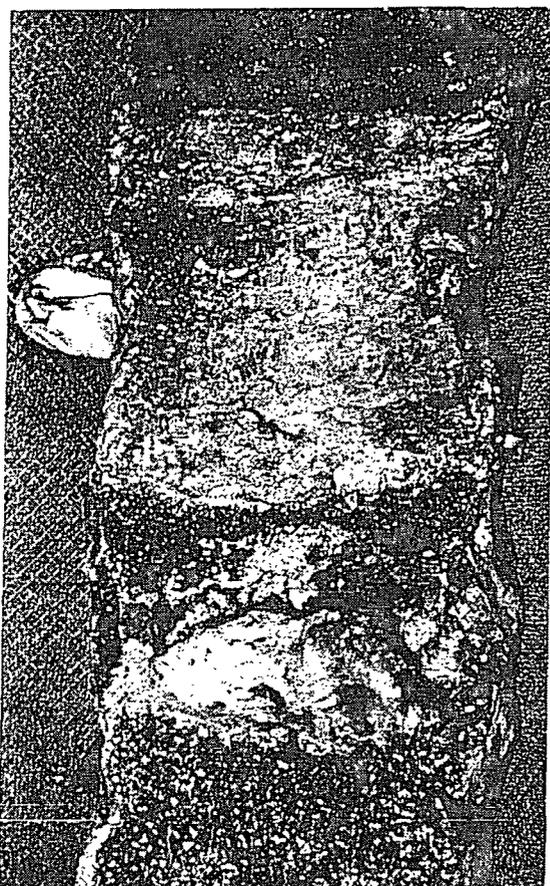


FIGURE 5.46 *Crushing of a thoracic vertebral body with disintegration of the adjacent disc following a violent hyperflexion in a car driver sustaining a deceleration injury. These lesions are best displayed at autopsy by cutting a longitudinal slice down the anterior aspect of the spinal column with an electric saw.*

rupture of the anterior longitudinal ligament, and tearing of intervertebral discs and of the annulus fibrosus may occur.

Nerve roots may be torn or compressed, and the spinal canal may be narrowed, distorted or even almost obliterated by fracture dislocations of the vertebrae. Compression, ischaemia, haemorrhage and even pulping of the spinal cord is the most serious complication. In older persons with cervical spondylosis, further narrowing of the canal by trauma may cause neurological effects that would not occur in younger subjects.

The most common part of the spine to be injured is the region of the upper two cervical vertebrae. In a series of fatal motor car accidents reported by Mant (1978), 35 per cent of passengers and 30 per cent of drivers suffered cervical spine damage. Of these the most common was a dislocation of the atlanto-occipital joint and Mant claims that one-third of all victims of fatal motor vehicle accidents

suffer this lesion, even though it is often undetected at autopsy. The mechanism of fracture of the atlas is said to be an axial impact via the head, when the occiput is held rigidly in line with the spine by contracted neck muscles. This most frequently occurs when the vertex of the lowered head violently strikes the windshield of a decelerating vehicle. Fracture or dislocation of the atlas or fracture of the odontoid of the axis can all occur from this type of impact. Hyperextension injuries tend to force the vertebral body forwards and, if there is significant displacement, the arch is intruded into the spinal canal with the consequent risk of cord damage. In addition, hyperextension – especially in the presence of cervical cord enlargement – can cause the ligamentum flavum to corrugate and intrude into the anterior part of the spinal canal to impinge upon the cord. When the injury is due to hyperflexion, dislocation of a vertebra causes it (or its fragments) to tilt backwards, again compromising the lumen of the spinal canal.

Dislocation of cervical vertebra can occur under many conditions. An anterior dislocation can be caused by a fall onto the back of the head and a unilateral dislocation is common when a head strikes a windscreen. Posterior dislocation may be caused by blows to the jaw or face that jolt the head backwards with a hyperextension element. Falling onto the face, especially from a height (such as down a staircase), is a likely cause. Fracture of the odontoid peg of the axis can occur from a variety of violent movements of the head on the neck, and is sometimes associated with fracture of the skull or mandible. Where gross injury to the spinal column is inflicted – such as a relatively high-speed motor vehicle or railway accident – the cord may be transected by a guillotine action of the two displaced fragments. This is most often seen on the upper or mid-thoracic region, from direct impact or gross ‘whiplash’ effects. The thoracic aorta at this level is often torn at the point where the descending arch meets the spine, even in the absence of a spinal fracture.

Damage to the cervical spine in hanging is rarely seen in the usual suicidal hangings in which sudden death is caused by carotid compression or – much less often – asphyxia. The drop in such cases is small or even absent, the subject merely slumping his weight against the neck restraint. Spinal damage can occasionally occur, however and the author has seen a fractured neck when a heavily built soldier stepped from a lavatory seat with the rope attached to the overhead cistern.

Judicial hanging with a long drop causes a severe fracture dislocation (rather than fracture) of the cervical spine, often with complete severance of the two fragments and transection of the spinal cord. Where such hanging is carried out with the knot of the suspension point beneath the chin, a violent hyperextension is produced that often fractures the axis and dislocates it from the third cervical vertebra. With a heavy person and a long drop, there may be complete decapitation.

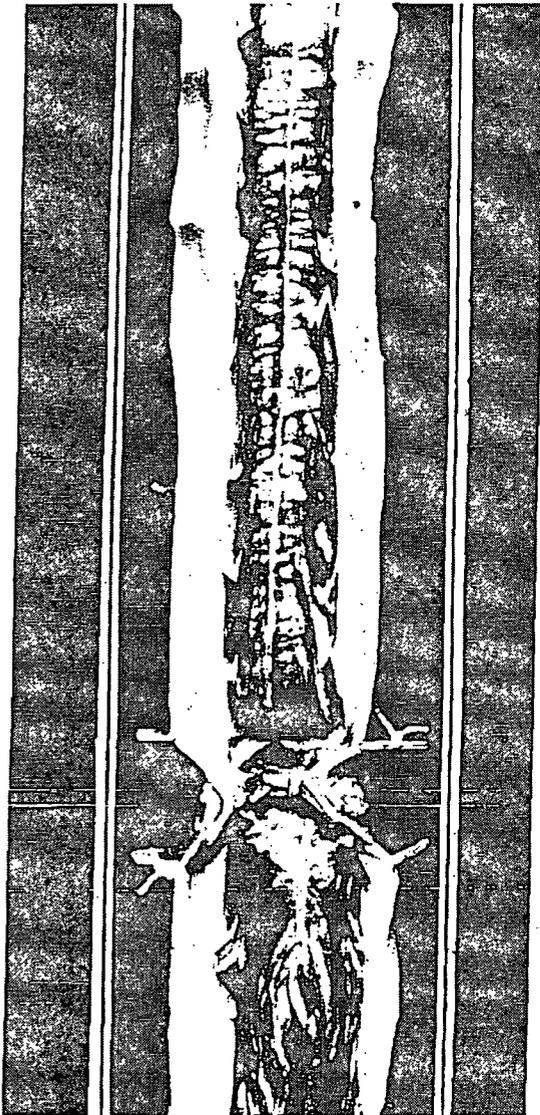


FIGURE 5.47 Crushing and transection of the lower spinal cord following a hyperflexion fracture of the lower thoracic vertebrae. The spinal cord ends at about the lower border of the first lumbar vertebra, the cauda equina occupying the remainder of the lumbar spinal canal. This injury occurred several years before death, and the dura is constricted and adherent to the remnants of the cord. At autopsy the cord and its membranes should be pinned out and fixed for a proper examination to be made.

Spinal cord injury

Most damage to the spinal cord arises from intrusion of some part of the spinal column into the canal, be it bony fragments or displacement, ligamentum flavum, disc annulus or extruded nucleus pulposus.

There may be bleeding into the space outside the spinal dura causing a space-occupying lesion in the canal that can compress the cord, or bleeding may occur within the dura, either from ruptured vessels or from haemorrhage in the cord itself. Damage to the cord may also occur in the absence of any apparent intrusion into the canal, in a manner similar to that seen in the brain in closed head injuries without a fracture. Haematomyelia and oedema may develop without any obvious mechanical defect at that level in the spinal column. Such injuries must be attributed to some momentary collision of the cord against the wall of the canal or a transient deformity of the profile of the canal that did not exceed the threshold for either fracture, dislocation or ligament rupture of the vertebral column.

The damage in the cord may extend for several segments above and below the point of impact. A haemorrhage within the cord tends to occupy the central grey matter more than the white columns, because of the softer and more vascular nature of the former tissue. Infarction of the cord can occur either in association with contusion, or because the local blood supply – often the anterior spinal artery – has been damaged.

An originally firm cord may undergo liquefaction ('myelomalacia') over several days or even longer, with progressive worsening of neurological symptoms.

REFERENCES AND FURTHER READING

- Adams JH. 1982. Diffuse axonal injury in non-missile head injury. *Injury* 13:444–5.
- Adams H, Mitchell DE, Graham DI, *et al.* 1977. Diffuse brain damage of immediate impact type. Its relationship to 'primary brain-stem damage' in head injury. *Brain* 100:489–502.
- Adams JH, Graham DI, Murray LS, *et al.* 1982. Diffuse axonal injury due to nonmissile head injury in humans: an analysis of 45 cases. *Ann Neurol* 12:557–63.
- Adams JH, Graham DI, Gennarelli TA. 1983. Head injury in man and experimental animals: Neuropathology. *Acta Neurochir Suppl (Wien)* 32:15–30.
- Adams JH, Doyle D, Graham DI, *et al.* 1984. Diffuse axonal injury in head injuries caused by a fall. *Lancet* 2:1420–2.
- Adams JH, Doyle D, Graham DI, *et al.* 1985. Microscopic diffuse axonal injury in cases of head injury. *Med Sci Law* 25:265–9.
- Adams JH, Doyle D, Graham DI, *et al.* 1985. The contusion index: a reappraisal in human and experimental non-missile head injury. *Neuropathol Appl Neurobiol* 11:299–308.

References and further reading

- Adams JH, Doyle D, Graham DI, *et al.* 1986. Gliding contusions in nonmissile head injury in humans [published erratum appears in *Arch Pathol Lab Med* 1986 Nov;110:1075]. *Arch Pathol Lab Med* 110:485-8.
- Adams JH, Doyle D, Graham DI, *et al.* 1986. Deep intracerebral (basal ganglia) haematomas in fatal non-missile head injury in man. *J Neurol Neurosurg Psychiatry* 49:1039-43.
- Adams JH, Doyle D, Ford I, *et al.* 1989. Diffuse axonal injury in head injury: definition, diagnosis and grading. *Histopathology* 15:49-59.
- Adams JH, Doyle D, Ford I, *et al.* 1989. Brain damage in fatal non-missile head injury in relation to age and type of injury. *Scott Med J* 34:399-401.
- Adams JH, Graham DI, Gennarelli TA, *et al.* 1991. Diffuse axonal injury in non-missile head injury. *J Neurol Neurosurg Psychiatry* 54:481-3.
- Adams JH, Corsellis JA, Duchon LW. 1992. *Greenfield's neuropathology*, 5th edn. Edward Arnold, London.
- Adams JH, Jennett B, McLellan DR, *et al.* 1999. The neuropathology of the vegetative state after head injury. *J Clin Pathol* 52:804-6.
- Adams JH, Graham DI, Jennett B. 2000. The neuropathology of the vegetative state after an acute brain insult. *Brain* 123(Pt 7):1327-38.
- Adams JH, Graham DI, Jennett B. 2001. The structural basis of moderate disability after traumatic brain damage. *J Neurol Neurosurg Psychiatry* 71:521-4.
- Adelson L. 1974. *The pathology of homicide*. Thomas, Springfield.
- Blumbergs PC, Jones NR, North JB. 1989. Diffuse axonal injury in head trauma. *J Neurol Neurosurg Psychiatry* 52:838-41.
- Bostrom K, Helander CG, Lindgren SO. 1992. Blunt basal head trauma: rupture of posterior inferior cerebellar artery. *Forensic Sci Int* 53:61-8.
- Bronilow A, Burns J. 1985. Technique for removal of the vertebral arteries. *J Clin Pathol* 38:1400-2.
- Caffey J. 1946. Multiple fractures in the long bones of infants suffering from chronic subdural haematoma. *Am J Radiol* 56:163-73.
- Cameron JM, Mant AK. 1972. Fatal subarachnoid haemorrhage associated with cervical trauma. *Med Sci Law* 12:66-70.
- Casson IR, Siegel O, Sham R, *et al.* 1984. Brain damage in modern boxers. *JAMA* 251:2663-7.
- Coast GC, Gee DJ. 1984. Traumatic subarachnoid haemorrhage: an alternative source. *J Clin Pathol* 37:1245-8.
- Contostavlos DL. 1971. Massive subarachnoid hemorrhage due to laceration of the vertebral artery associated with fracture of the transverse process of the atlas. *J Forensic Sci* 16:40-56.
- Contostavlos DL. 1995. Isolated basilar traumatic subarachnoid hemorrhage: an observer's 25 year re-evaluation of the pathogenetic possibilities. [letter; comment] *Forensic Sci Int* 73:61-74.
- Corsellis JA, Bruton CJ, Freeman Browne D. 1973. The aftermath of boxing. *Psychol Med* 3:270-303.
- Courville CB. 1942. Coup-contre coup mechanism of craniocerebral injuries: some observations. *Arch Surg* 45:19-43.
- Courville CB. 1953. *Comotio cerebri*. San Lucas Press, Los Angeles.
- Courville CB. 1964. *Forensic neuropathology*. Callaghan and Co, Mundelein.
- Critchley M. 1957. Medical aspects of boxing particularly from a neurological standpoint. *Br Med J* 1:357-62.
- Crompton MR. 1971. Brain stem lesions due to closed head injury. *Lancet* 1:669-71.
- Crompton R. 1985. *Closed head injuries*. Edward Arnold, London.
- Dalgaard J. 1957. Brain injuries as a cause of oesophagogastrroduodenal ulceration. *J Forensic Med* 4:110-27.
- Dawson SL, Hirsch CS, Lucas FV, *et al.* 1980. The contrecoup phenomenon. Reappraisal of a classic problem. *Hum Pathol* 11:155-66.
- Deck JH, Jagadha V. 1986. Fatal subarachnoid hemorrhage due to traumatic rupture of the vertebral artery. *Arch Pathol Lab Med* 110:489-93.
- Denny-Brown D. 1945. Cerebral concussion. *Physiol Rev* 25:296-325.
- Denny-Brown D, Russell WR. 1941. Experimental cerebral concussion. *Brain* 64:93-164.
- Dowling G, Curry B. 1988. Traumatic basal subarachnoid hemorrhage. Report of six cases and review of the literature. *Am J Forensic Med Pathol* 9:23-31.
- Duhaime AC, Gennarelli TA, Thibault LE, *et al.* 1987. The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 66:409-15.
- Duhaime AC, Alario AJ, Lewander WJ, *et al.* 1992. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 90:179-85.
- Edberg S, Angrist A. 1967. Intra-cranial pressure changes following impact of human cadaver heads. *J Forensic Sci* 12:60-8.

- Editorial. 1973. Boxing brains. *Lancet* 2:1064-5.
- Farag AM, Franks A, Gee DJ. 1988. Simple laboratory experiments to replicate some of the stresses on vertebro-basilar arterial walls. An investigation of possible mechanisms of traumatic subarachnoid haemorrhage. *Forensic Sci Int* 38:275-84.
- Fell DA, Fitzgerald S, Moiel RH, et al. 1975. Acute subdural hematomas. Review of 144 cases. *J Neurosurg* 42:37-42.
- Flamm ES, Demopoulos HB, Seligman ML, et al. 1977. Ethanol potentiation of central nervous system trauma. *J Neurosurg* 46:328-35.
- Ford R. 1956. Basal subarachnoid haemorrhage and trauma. *J Forensic Sci* 1:117-26.
- Freytag E. 1963. Autopsy findings in head injuries from blunt forces: statistical evaluation of 1367 cases. *Arch Pathol* 75:74-80.
- Garfield J. 2002. Acute subdural haematoma in a boxer. *Br J Neurosurg* 16:96-9; discussion 99-101.
- Geddes JF, Whitwell HL, Graham DI. 2000. Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. *Neuropathol Appl Neurobiol* 26:105-16.
- Geddes JF, Hackshaw AK, Vowles GH, et al. 2001. Neuropathology of inflicted head injury in children. I. Patterns of brain damage. *Brain* 124:1290-8.
- Geddes JF, Vowles GH, Hackshaw AK, et al. 2001. Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants. *Brain* 124:1299-306.
- Gennarelli TA. 1983. Head injury in man and experimental animals: clinical aspects. *Acta Neurochir Suppl (Wien)* 32:1-13.
- Gennarelli TA. 1993. Mechanisms of brain injury. *J Emerg Med* 11(Suppl 1):5-11.
- Gennarelli TA, Graham DI. 1998. Neuropathology of the head injuries. *Semin Clin Neuropsychiatry* 3:160-175.
- Gennarelli TA, Thibault LE. 1982. Biomechanics of acute subdural hematoma. *J Trauma* 22:680-6.
- Gennarelli TA, Thibault LE, Adams JH, et al. 1982. Diffuse axonal injury and traumatic coma in the primate. *Ann Neurol* 12:564-74.
- Gennarelli TA, Thibault LE, Tipperman R, et al. 1989. Axonal injury in the optic nerve: a model simulating diffuse axonal injury in the brain. *J Neurosurg* 71:244-53.
- Gentleman SM, McKenzie JE, Royston MC, et al. 1999. A comparison of manual and semi-automated methods in the assessment of axonal injury. *Neuropathol Appl Neurobiol* 25:41-7.
- Goonetilleke UK. 1980. Injuries caused by falls from heights. *Med Sci Law* 20:262-75.
- Graham DI. 2001. Paediatric head injury. *Brain* 124:1261-2.
- Graham DI, Adams JH. 1971. Ischaemic brain damage in fatal head injuries. *Lancet* 1:265-6.
- Graham DI, Adams JH. 1972. *The pathology of blunt head injuries*. In: Critchley M (ed.), *Scientific foundation of neurology*. Heinemann Medical Publications, London.
- Graham DI, Lantos PL (eds). 2002. *Greenfield's neuropathology*, 7th edn. Arnold, London/ New York/New Delhi.
- Graham DI, McLellan D, Adams JH, et al. 1983. The neuropathology of the vegetative state and severe disability after non-missile head injury. *Acta Neurochir Suppl (Wien)* 32:65-7.
- Graham DI, Lawrence AE, Adams JH, et al. 1988. Brain damage in fatal non-missile head injury without high intracranial pressure. *J Clin Pathol* 41:34-7.
- Graham DI, Ford I, Adams JH, et al. 1989. Fatal head injury in children. *J Clin Pathol* 42:18-22.
- Graham DI, Clark JC, Adams JH, et al. 1992. Diffuse axonal injury caused by assault. *J Clin Pathol* 45:840-1.
- Graham DI, McIntosh TK, Maxwell WL, et al. 2000. Recent advances in neurotrauma. *J Neuropathol Exp Neurol* 59:641-51.
- Graham DI, Raghupathi R, Saatman KE, et al. 2000. Tissue tears in the white matter after lateral fluid percussion brain injury in the rat: Relevance to human brain injury. *Acta Neuropathol (Berl)* 99:117-24.
- Gurdjian ES, Webster JE. 1958. *Head injuries*. Churchill, London, UK.
- Gurdjian ES, Webster JE, Lissner HR. 1949. Studies on skull fracture with particular reference to engineering factors. *Am J Surg* 78:736-42.
- Gurdjian ES, Webster JE, Lissner HR. 1950. The mechanism of skull fracture. *Radiology* 54:313-39.
- Harris LS. 1991. Postmortem magnetic resonance images of the injured brain: effective evidence in the courtroom. *Forensic Sci Int* 50:179-85.
- Harwood-Nash CE, Hendrick EB, Hudson AR. 1971. The significance of skull fractures in children. A study of 1,187 patients. *Radiology* 101:151-6.
- Hijdra A, Vermeulen M, van Gijn J, et al. 1984. Respiratory arrest in subarachnoid hemorrhage. *Neurology* 34:1501-3.
- Holbourn AH. 1943. Mechanics of head injuries. *Lancet* 245:438-41.

References and further reading

- Huelke DF, O'Day J, Mendelsohn RA. 1981. Cervical injuries suffered in automobile crashes. *J Neurosurg* 54:316-22.
- Imajo T. 1996. Diffuse axonal injury: its mechanism in an assault case. *Am J Forensic Med Pathol* 17:324-6.
- Imajo T, Kazee AM. 1992. Diffuse axonal injury by simple fall. *Am J Forensic Med Pathol* 13:169-72.
- Imajo T, Roessman U. 1984. Diffuse axonal injury. *Am J Forensic Med Pathol* 5:217-22.
- Imajo T, Challener RC, Roessmann U. 1987. Diffuse axonal injury by assault. *Am J Forensic Med Pathol* 8:217-19.
- Jafari SS, Maxwell WL, Neilson M, et al. 1997. Axonal cytoskeletal changes after non-disruptive axonal injury. *J Neurocytol* 26:207-21.
- Jafari SS, Neilson M, Graham DI, et al. 1998. Axonal cytoskeletal changes after nondisruptive axonal injury. II. Intermediate sized axons. *J Neurotrauma* 15:955-66.
- Jamieson KG, Yelland JD. 1968. Extradural hematoma. Report of 167 cases. *J Neurosurg* 29:13-23.
- Johnson CP, Lawler W, Burns J. 1993. Use of histomorphometry in the assessment of fatal vertebral artery dissection. *J Clin Pathol* 46:1000-3.
- Johnson CP, How T, Scraggs M, et al. 1994. The poor biomechanical response of the vertebral artery applied longitudinal force. *J Pathol Suppl*:234.
- Johnson DL, Boal D, Baule R. 1995. Role of apnea in nonaccidental head injury. *Pediatr Neurosurg* 23:305-10.
- Johnson J. 1969. Organic psychosyndromes due to boxing. *Br J Psychiatry* 115:45-53.
- Johnson P, Burns J. 1995. Extracranial vertebral artery injury - evolution of a pathological illusion? [letter; comment] *Forensic Sci Int* 73:75-8.
- Kaur B, Ruddy GN, Timperley WR. 1999. The possible role of hypoxia in the formation of axonal bulbs. *J Clin Pathol* 52:203-9.
- Kerr D. 1954. *Forensic medicine*, 5th edn. A. & C. Black, Edinburgh, p. 114.
- Knight B. 1979. Trauma and ruptured cerebral aneurysm. *Br Med J* 1:1430-1.
- Kubo S, Kitamura O, Orihara Y, et al. 1998. Immunohistochemical diagnosis and significance of forensic neuropathological changes. *J Med Invest* 44:109-19.
- Leadbeater S. 1994. Extracranial vertebral artery injury - evolution of a pathological illusion? [see comments] *Forensic Sci Int* 67:33-40.
- Leadbeater S. 1995. Letter to editor. *Forensic Sci Int* 73:78-9.
- Ledran H. 1751. *Observatis des chirurgies*. Osmont, Paris.
- Leestma JE. 1988. *Forensic neuropathology*. Raven Press, New York.
- Li L, Smialek JE. 1994. The investigation of fatal falls and jumps from heights in Maryland (1987-1992). *Am J Forensic Med Pathol* 15:295-9.
- Lindenberg R. 1955. Lesions of the corpus callosum following blunt mechanical trauma to the head. *Am J Pathol* 31:297-301.
- Lindenberg R, Freytag E. 1957. Morphology of cerebral contusions. *Arch Pathol* 63:23-6.
- Lindenberg R, Freytag E. 1960. The mechanism of cerebral contusions: a pathologic-anatomic study. *Arch Pathol* 69:440-4.
- Lindenberg R, Freytag E. 1970. Brainstem lesions characteristic of traumatic hyperextension of the head. *Arch Pathol* 90:509-15.
- Lindsay KW, McLatchie G, Jennett B. 1980. Serious head injury in sport. *Br Med J* 281:789-91.
- Lundberg GD. 1985. Brain injury in boxing. *Am J Forensic Med Pathol* 6:192-8.
- Madro R, Chagowski W. 1987. An attempt at objectivity of post mortem diagnostic of brain oedema. *Forensic Sci Int* 35:125-9.
- Maloney AFJ, Whatmore WJ. 1969. Clinical and pathological observations in fatal head injuries: a five year survey of 173 cases. *Br J Surg* 56:23-30.
- Mansell PW, Hunt AC. 1968. Major head injuries in fights and brawls. *Med Sci Law* 8:181-7.
- Mant AK. 1978. Injuries and death in motor vehicle accidents. In: Mason JK (ed.), *Pathology of violent injury*. Edward Arnold, London.
- Mant AK. 1993. Injuries and death in motor vehicle accidents. In: Mason JK (ed.), *Pathology of violent injury*, 2nd edn. Edward Arnold, London.
- Marek Z. 1981. Isolated subarachnoid hemorrhage as a medicolegal problem. *Am J Forensic Med Pathol* 2:19-22.
- Marlet JM, Barreto Fonseca JdP. 1982. Experimental determination of time of intracranial hemorrhage by spectrophotometric analysis of cerebrospinal fluid. *J Forensic Sci* 27:880-8.
- Martland MS. 1928. Punch drunk. *JAMA* 91:1003-5.
- Mawdsley C, Ferguson SR. 1963. Neurological disease in boxers. *Lancet* 2:795-7.

- McCormick WF. 1980. The relationship of closed-head trauma to rupture of saccular intracranial aneurysms. *Am J Forensic Med Pathol* 1:223-6.
- McCrory P. 2002. Boxing and the brain. Revisiting chronic traumatic encephalopathy. *Br J Sports Med* 36:2.
- McKissock H. 1960. Extradural haemorrhage; observations on 125 cases. *Lancet* 2:167-74.
- Missliwetz J. 1995. Fatal impalement injuries after falls at construction sites. *Am J Forensic Med Pathol* 16:81-3.
- Mitchell DE, Adams JH. 1973. Primary focal impact damage to the brainstem in blunt head injuries. Does it exist? *Lancet* 2:215-18.
- Miyazaki T, Kojima T, Chikasue F, et al. 1990. Traumatic rupture of intracranial vertebral artery due to hyperextension of the head: reports on three cases. *Forensic Sci Int* 47:91-8.
- Moar JJ. 1987. Traumatic rupture of the cervical carotid arteries: an autopsy and histopathological study of 200 cases. *Forensic Sci Int* 34:227-44.
- Morgagni J. 1761. *De sedibus et causis morborum per anatomen indagatis*. Typographia Academia, Lovanii.
- Moritz AR. 1942. *Pathology of trauma*. Lea & Febiger, Philadelphia.
- Munro D. 1938. *Cranio-cerebral injuries*. Oxford University Press, Oxford.
- Munro D, Merritt HH. 1936. Surgical pathology of subdural haematoma. Based on a study of 105 cases. *Arch Neurol Psychiatry* 35:64-78.
- Oehmichen M. 1994. Brain death: neuropathological findings and forensic implications. *Forensic Sci Int* 69:205-19.
- Oehmichen M, Raff G. 1980. Timing of cortical contusion. Correlation between histomorphologic alterations and post-traumatic interval. *Z Rechtsmed* 84:79-94.
- Oehmichen M, Eisenmenger W, Raff G, et al. 1986. Brain macrophages in human cortical contusions as indicator of survival period. *Forensic Sci Int* 30:281-301.
- Oehmichen M, Meissner C, Schmidt V, et al. 1999. Pontine axonal injury after brain trauma and nontraumatic hypoxic-ischemic brain damage. *Int J Legal Med* 112:261-7.
- Oehmichen M, Theuerkauf I, Meissner C. 1999. Is traumatic axonal injury (AE) associated with an early microglial activation? Application of a double-labeling technique for simultaneous detection of microglia and ai. *Acta Neuropathol (Berl)* 97:491-4.
- Oehmichen M, Ochs U, Meissner C. 2000. Histochemical characterization of cytotoxic brain edema. Potassium concentrations after cerebral ischemia and during the postmortem interval. *Exp Toxicol Pathol* 52:348-52.
- Oehmichen M, Meissner C, König HG. 2001. Brain injury after survived gunshot to the head: reactive alterations at sites remote from the missile track. *Forensic Sci Int* 115:189-97.
- Oppenheimer DR. 1968. Microscopic lesions in the brain following head injury. *J Neurol Neurosurg Psychiatry* 31:299-306.
- Povlishock JT. 1992. Traumatically induced axonal injury: pathogenesis and pathobiological implications. *Brain Pathol* 2:1-12.
- Povlishock JT. 2000. Pathophysiology of neural injury: therapeutic opportunities and challenges. *Clin Neurosurg* 46:113-26.
- Povlishock JT, Christman CW. 1995. The pathobiology of traumatically induced axonal injury in animals and humans: a review of current thoughts. *J Neurotrauma* 12:555-64.
- Raisanen J, Ghogassian DF, Moskvitch M, et al. 1999. Diffuse axonal injury in a rugby player. *Am J Forensic Med Pathol* 20:70-2.
- Roberts AH. 1970. *Brain damage in boxers*. Pitman Medical, London.
- Root I. 1992. Head injuries from short distance falls [see comments]. *Am J Forensic Med Pathol* 13:85-7.
- Rowbotham OF. 1964. *Acute injuries of the head*, 4th edn. Churchill Livingstone, Edinburgh.
- Royal College of Physicians of London. 1969. *Report on the medical aspects of boxing*. Royal College of Physicians of London, London.
- Schellhas KP, Latchaw RE, Wendling LR, et al. 1980. Vertebrobasilar injuries following cervical manipulation. *JAMA* 244:1450-3.
- Schellinger PD, Schwab S, Krieger D, et al. 2001. Masking of vertebral artery dissection by severe trauma to the cervical spine. *Spine* 26:314-19.
- Shapiro HA, Gordon I, Benson SD. 1988. *Forensic medicine - a guide to principles*, 3rd edn. Churchill Livingstone, Edinburgh.
- Sheffield EA, Weller RO. 1980. Age changes at cerebral artery bifurcations and the pathogenesis of berry aneurysms. *J Neurol Sci* 46:341-52.
- Sherriff FE, Bridges LR, Sivaloganathan S. 1994. Early detection of axonal injury after human head trauma using immunocytochemistry for beta-amyloid precursor protein. *Acta Neuropathol (Berl)* 87:55-62.

References and further reading

- Simonsen J. 1967. Fatal subarachnoid haemorrhage in relation to minor head injuries. *J Forensic Med* 14:146-55.
- Simonsen J. 1976. Massive subarachnoid haemorrhage and fracture of the transverse process of the atlas. *Med Sci Law* 16:13-6.
- Simpson RH, Berson DS, Shapiro HA. 1985. The diagnosis of diffuse axonal injury in routine autopsy practice. *Forensic Sci Int* 27:229-35.
- Skold G. 1985. Injuries to pathologically changed cervical vertebrae. *Am J Forensic Med Pathol* 6:163-6.
- Smith DH, Chen XH, Xu BN, et al. 1997. Characterization of diffuse axonal pathology and selective hippocampal damage following inertial brain trauma in the pig. *J Neuropathol Exp Neurol* 56:822-34.
- Smith DH, Nonaka M, Miller R, et al. 2000. Immediate coma following inertial brain injury dependent on axonal damage in the brainstem. *J Neurosurg* 93:315-22.
- Somerville A. 1961. Subarachnoid haemorrhage due to trauma without visible external injury. *Med Sci Law* 2:67-9.
- Spicer EJ, Strich SJ. 1967. Haemorrhages in posterior-root ganglia in patients dying from head injury. *Lancet* 2:1389-91.
- Spillane JD. 1962. Five boxers. *Br Med J* 2:1205-6.
- Storey PB. 1969. The precipitation of subarachnoid haemorrhage. *J Psychosom Res* 13:175-82.
- Strassman G. 1949. Formation of haemosiderin and haematoidin after traumatic and spontaneous cerebral haemorrhages. *Arch Pathol* 47:205-10.
- Strich SJ. 1961. Shearing of nerve fibres as a cause of brain damage due to head injury: a pathological study. *Lancet* 2:443-7.
- Strich SJ. 1970. Lesions in the cerebral hemispheres after blunt head injury. *J Clin Pathol Suppl R Coll Pathol* 4:166-71.
- Strich SJ. 1976. Cerebral trauma. In: Blackwood W, Corsellis JAN (eds), *Greenfield's neuropathology*, 3rd edn. Edward Arnold, London, pp. 327-60.
- Symonds CP. 1940. Concussion and contusion of the brain. In: Brock (ed.), *Injuries of the skull, brain and spinal cord*. Baillière, London.
- Symonds CP. 1962. Concussion. *Lancet* 1:1-5.
- Tatsuno Y, Lindenberg R. 1974. Basal subarachnoid hematomas as sole intracranial traumatic lesions. *Arch Pathol* 97:211-5.
- Terespolsky PS. 1972. Post-traumatic epilepsy. *Forensic Sci* 1:147-65.
- Thiagaraj D, Ming T, Cyn A. 1983. Patterns of injury in deaths due to falling from heights. *Proc First Asian Pacific Congr Legal Med*, p. 399.
- Tomei G, Spagnoli D, Ducati A, et al. 1990. Morphology and neurophysiology of focal axonal injury experimentally induced in the guinea pig optic nerve. *Acta Neuropathol (Berl)* 80:506-13.
- Tomlinson BE. 1970. Brain-stem lesions after head injury. *J Clin Pathol Suppl R Coll Pathol* 4:154-65.
- Trotter W. 1914. Concussion. *Br J Surg* 2:271-91.
- Vanezis P. 1979. Techniques used in the evaluation of vertebral artery trauma at post-mortem. *Forensic Sci Int* 13:159-65.
- Vanezis P. 1984. *Vertebral artery trauma*. Medical Faculty, University of Bristol, Bristol.
- Vanezis P. 1986. Vertebral artery injuries in road traffic accidents: a post-mortem study. *J Forensic Sci Soc* 26:281-91.
- Vanezis P. 1989. *Pathology of neck injury*. Butterworths, London.
- Vanezis P, Chan KK, Scholtz CL. 1987. White matter damage following acute head injury. *Forensic Sci Int* 35:1-10.
- Voigt GE. 1981. Small hemorrhages in the brain stem. A sign of injury? *Am J Forensic Med Pathol* 2:115-20.
- Wang H, Duan G, Zhang J, et al. 1998. Clinical studies on diffuse axonal injury in patients with severe closed head injury. *Chin Med J (Engl)* 111:59-62.
- Weber W. 1984. Experimentelle Untersuchungen zu Schädelbruchverletzungen des Säuglings. [Experimental studies of skull fractures in infants] *Z Rechtsmed* 92:87-94.
- Wilkinson AE, Bridges LR, Sivaloganathan S. 1999. Correlation of survival time with size of axonal swellings in diffuse axonal injury. *Acta Neuropathol (Berl)* 98:197-202.
- Wilson JV. 1946. *The pathology of traumatic injury*. Livingstone, Edinburgh.
- Yamashima T, Friede RL. 1984. Why do bridging veins rupture into the virtual subdural space? *J Neurol Neurosurg Psychiatry* 47:121-7.
- Yanagida Y, Fujiwara S, Mizoi Y. 1989. Differences in the intracranial pressure caused by a 'blow' and/or a 'fall' - an experimental study using physical models of the head and neck. *Forensic Sci Int* 41:135-45.
- Zhang X, Niu W. 1993. A study of enzymohisto-chemistry of cerebral cortical injury. *Forensic Sci Int* 59:19-24.
- Zuo ZJ, Zhu JZ. 1991. Study on the microstructures of skull fracture. *Forensic Sci Int* 50:1-14.

Mechanical Properties and Anthropometry of the Human Infant Head

Michael T. Prange, Jason F. Luck, Alan Dibb, Chris A. Van Ee*, Roger W. Nightingale, and
Barry S. Myers

Injury and Orthopaedic Biomechanics Laboratory, Duke University

*Design Research Engineering, Novi, MI

ABSTRACT - The adult head has been studied extensively and computationally modeled for impact, however there have been few studies that attempt to quantify the mechanical properties of the pediatric skull. Likewise, little documentation of pediatric anthropometry exists. We hypothesize that the properties of the human pediatric skull differ from the human adult skull and exhibit viscoelastic structural properties. Quasi-static and dynamic compression tests were performed using the whole head of three human neonate specimens (ages 1 to 11 days old). Whole head compression tests were performed in a MTS servo-hydraulic actuator. Testing was conducted using nondestructive quasi-static, and constant velocity protocols in the anterior-posterior and right-left directions. In addition, the pediatric head specimens were dropped from 15cm and 30cm and impact force-time histories were measured for five different locations: vertex, occiput, forehead, right and left parietal region. The compression stiffness values increased with an increase in velocity but were not significantly different between the anterior-posterior and right-left directions. Peak head acceleration during the head impact tests did not significantly vary between the five different impact locations. A three parameter model that included damping represented the pediatric head impact data more accurately than a simple mass-spring system. The compressive and impact stiffness of the pediatric heads were significantly more compliant than published adult values. Also, infant head dimensions, center of gravity and moment of inertia (I_{yy}) were determined. The CRABI 6-month dummy impact response was similar to the infant cadaver for impacts to the vertex, occiput, and forehead but dramatically stiffer in lateral impacts. These pediatric head anthropomorphic, compression, and impact data will provide a basis to validate whole head models and compare with ATD performance in similar exposures.

KEYWORDS -- head injury, skull fracture, pediatric, children, ATD, CRABI, fall

INTRODUCTION

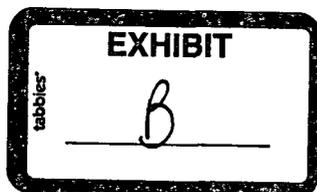
Traumatic brain injury is the most common cause of death in childhood (CDC 1990). Brain injuries resulting in hospitalization or death occur in at least 150,000 children per year, at a rate of over 200 per 100,000 children. Head injury in infancy results in higher morbidity and mortality than that seen in older children (Lucrsson 1993). Head injuries cause approximately 75% of the pediatric hospitalizations due to trauma and approximately 30% of childhood injury deaths are caused by head injury (Kraus et al. 1990; James 1999). Ten percent of children suffer from a significant head injury at some point during their school years (James 1999).

Head injuries constitute a greater proportion of all injuries in the infant population (≤ 1 year old) than older children and infants have a greater likelihood of having an intracranial injury after following blunt head trauma (Burd et al. 1969; Gotschall and Luchter 1999; Greenes and Schutzman 1999). The percentage of infant skull fractures are approximately

double that of any other pediatric age group (Gotschall and Luchter 1999). The majority of accidental pediatric head injuries involve motor vehicle collisions (Kraus et al. 1990; Gotschall and Luchter 1999; James 1999).

Since the mid 1980s, much of the basic biomechanics of the head have been studied and the findings published in peer-reviewed journals. Quantitative geometric, constitutive, and structural data are available for the adult head and computational methods are progressing rapidly. Despite this increase in understanding, the mechanical properties of the pediatric head are all but unknown. As an example, recent helmet standards for children have been forced to use adult criteria owing to a lack of pediatric tolerance data (Myers 1997).

One factor that makes pediatric head injury unique is that children are not miniature adults. Children have bodies with different geometric and mass proportions as compared to adults. In a study by Burd et al., the shape of the human body was examined from birth



Prange, M.T. et al.

until adult age (Burdick et al. 1969). At birth the head makes up about 1/4 of the infant's total height and decreased to 1/7 at adulthood. Also at birth, the child has a long trunk with the upper limbs longer than the lower limbs and as the child grows these proportions change drastically (Burdick et al. 1969).

The pediatric skull differs from that of the adult owing to differences in geometry and materials. The newborn has a high and slightly protruding forehead and the infant cranium is longer and rounder than the adult head. The pediatric cranial bones are thin, pliable plates composed of partially calcified bony tissue with an immature diploe. The cranial bones of a child are separated by relatively broad syndesmosis sutures. Fontanelles are located at the junction of these sutures with the most prominent "soft spot" located in the frontal region of the top of the skull. These fibrous tissue sutures allow the skull to expand to accommodate the rapidly growing brain and birthing process. The sutures generally fuse by age two and the anterior fontanelle closes at approximately 2.5 years of life (Ridgway and Weiner 2004).

Several studies relate pediatric animal surrogates to the human child to validate the use of the animal models. Usually the correlation of the pediatric surrogate to the human child is based on a developmental relationship of a shared biological structure or set of structures but again definitive human data are absent (Dobbing 1981; Prasad and Daniel 1984; Thibault and Margulies 1998; Margulies and Thibault 2000). Margulies and Thibault investigated the rate and age dependency of porcine skulls by testing samples of cranial bone and suture to failure in bending (Margulies and Thibault 2000). Unfortunately, the authors were only able to examine one full-term human infant specimen for validation.

Comprehensive studies of mechanical properties of human adult skull and cranial bone have been performed (Hodgson et al. 1967; Hodgson and Patrick 1968; Thomas et al. 1968; Hubbard 1971; Hubbard et al. 1971; McElhaney et al. 1976; Ono et al. 1980). These studies tested the human adult cranial bone in tension, compression, simple shear, and torsion. Unfortunately, there have been few works that attempt to quantify the mechanical properties of the human pediatric skull (McPherson and Kriewall 1980; Kriewall et al. 1981; Margulies and Thibault 2000). These few studies found infant cranial bone stiffness to be approximately 10% of adult cranial bone. Despite these positive contributions, impact, viscoelastic, and quasi-static

data on selected age groups are still needed to validate surrogate models and devices.

Along with the lack of data regarding the mechanical properties of the pediatric head, little documentation of detailed pediatric anthropometry exists. Data exist on the anatomical dimensions of children at various ages but no studies have investigated the location of the head center of gravity or quantified the head moments of inertia (Schneider et al. 1986).

Because of this paucity of pediatric data, scaling methods have been employed to determine the head injury tolerance of the child based on adult data (Ommaya et al. 1967; Mertz et al. 1989; Melvin 1995; Irwin and Mertz 1997). These analytical scaling techniques are based on differences in head size, head mass, cranial bone stiffness, and brain stiffness. To date, little human pediatric data exist to benchmark these scaling techniques.

Currently, the biomechanical response of the human infant head is unknown. This paper reports the compressive and impact response using infant cadaveric specimens and derives lumped parameter estimates of infant head structural properties. We hypothesize that the properties of the human pediatric skull differ from the human adult skull and exhibit viscoelastic structural properties. The compression and impact response of a 6-month old anthropomorphic test dummy were measured and compared to the infant cadaver results.

METHODS

Specimen procurement and preparation

Human pediatric cadaver specimens were used to determine the static and dynamic properties of the whole infant head. These biomechanical tests were conducted using three unembalmed fresh-frozen human infant specimens of ages 1, 3, and 11 days after birth. The head was separated from the cervical spine and the mandible was removed while keeping the scalp intact. Maximum head length and maximum head breadth for each specimen were measured using a spreading caliper. After the experimental protocol was complete, the scalp was dissected and the skull was examined for fractures or other tissue damage. The use of pediatric cadaveric tissue was approved by the Duke University IRB.

Anthropometry

Each of the three specimens was imaged using high resolution computed tomography (CT). With the use of cadaveric tissue, these scans could be taken with the highest voltage and current settings (140 kV, 400 mA). This resulted in detailed scans at 0.43 mm/pixel resolution with a slice thickness of 0.625mm at 0.625mm intervals. The CT data was imported into the Amira™ software package (TGS, Inc., San Diego CA) to perform a three-dimensional reconstruction of the head anatomy. The coordinate system for this reconstruction was orientated such that the Frankfort plane was horizontal, the midsagittal plane was vertical, and the origin was located at the level of the superior margin of the right auditory meatus in the midsagittal plane. Head height (auditory meatus to vertex), facial height, and bizygomatic diameter were measure from the CT reconstruction. Locations of the most anterior and most posterior point on the right occipital condyle were determined and the center of the condyle was defined as the average of these coordinates.

After the testing protocol described below was complete, the mandible was reattached using surgical sutures. A pin was inserted into the superior margin of the right auditory meatus and right infraorbital foramen to identify the Frankfort plane. The center of gravity (Cg) of the head was determined using the method described by Walker et al (Walker et al. 1973). Two screw hooks were placed in the skull in the midsagittal plane. If a hook could not be secured in the compliant pediatric skull then a suture was placed through the scalp and used as the attachment point. The mass of the head with mandible and hooks was recorded. The head was suspended in the frame by one of the hooks and a digital image was taken of the right side of the head. A ruler and plumbline were included in the image to establish a scale and vertical direction. Another image was taken with the head suspended by the other hook. The location of the hooks, superior margin of the auditory meatus, infraorbital foramen, and plumbline were digitized from each image (Scion Image, Scion Corporation). The two images were registered by aligning the auditory meatus and Frankfort plane of each image. The Cg of the head was determined by the location of the intersection of the vertical lines passing through the suspension point of each image (Walker et al. 1973).

While suspended, the head was swung in the midsagittal plane. The motion of the head was recorded with a digital video camera at 50Hz and the period of the swing (T) was measured. The period of the motion was determined as the average period

over the first 10 swings. The error of the measured period using this method was approximately ± 2 msec. The mass moment of inertia about the y-axis (line in the transverse direction, I_{yy}) at the Cg was calculated using the mass moment of inertia of the pendulum at its suspension point and the parallel axis theorem:

$$I_{yy} = \frac{mgdT^2}{4\pi^2} - md^2 \quad (1)$$

where m is the mass of the head, g is the acceleration due to gravity, and d is the distance from the Cg to the suspension point (Walker et al. 1973). The mass of the hooks were less than 0.2% of the mass of the head therefore the hooks' moments of inertia were considered negligible.

Pediatric head compression

Experimental protocol- A series of compression tests were conducted to determine static and rate-dependent properties of the infant head in two directions. The maximum head deformation during the compression was limited to 5% of the total gauge length of the head in order to ensure that no damage or fractures occurred. The foramen magnum was loosely blocked with gauze to contain the intracranial contents prior to testing while imposing negligible resistance to extrusion during the experiments.

Using a parallel plate fixture and MTS hydraulic actuator, force-deflection data was recorded during whole head compression experiments. A uniaxial strain gauge load cell (S-type, Omega Engineering Inc.) was used to measure the compressive load and a LVDT was used to measure the position of the actuator. Data for all tests were recorded using a digital data acquisition system (National Instruments, Austin, TX). The head was positioned between the two smooth flat plates so that the compression direction was in the anterior-posterior (AP) or right-left (RL) direction and along the Frankfort plane. At the beginning of the experiment the actuator was adjusted to achieve approximately 0.5N of pre-compressive force. This pre-compressive force was necessary to maintain the specimen between the plates in the correct orientation. Before each test the head was repositioned and the gauge length was measured if necessary.

Prior to the test battery, the head was preconditioned at 50% of maximum displacement at 1 Hz for 60 seconds. The head was compressed at a constant velocity using four different rates. A test was conducted at 0.05 mm/s to determine the quasistatic

stiffness of the head. Three additional compression tests were conducted to the same displacement at constant velocities of approximately 1.0 mm/s, 10 mm/s, and 50 mm/s. These experiments were conducted in the AP and RL directions. Because of limitations of the hydraulic actuator, the 50 mm/s velocity tests resulted in the varying velocity during the end of the test. Therefore, this non-constant velocity data from the last 1 mm of displacement during these high rate experiments were omitted from the analysis. Also, the 50 mm/s tests were not recorded for the 11 day old specimen due to data acquisition errors during the experiment.

Analysis - Because the force-deflection data was nonlinear, each test was regressed between 50% through 100% maximum displacement in order to determine a stiffness value. The slope of this line was defined as the stiffness and the x-intercept of the projected line as the low-load displacement. The whole data set was also regressed using an exponential function:

$$F = A(e^{Bx} - 1) \quad (2)$$

where A and B are constants and F and x are the compressive force and displacement respectively. These analyses were repeated for each of the two compression directions and each of the four velocities. An average stiffness, low-load displacement, A, and B across specimens were calculated for each compression direction and velocity. Differences between direction and velocity were determined using 2-way analysis of variance (ANOVA) and Tukey tests for multiple comparisons. The significance level for the statistical analysis was set at 5%. Also, the coefficient of determination (r^2) values of the linear and exponential regression for each experiment were compared using a paired Student's t test.

Pediatric head impact

Experimental protocol - After the compression experiments were complete, voids in the cranial cavity were filled with water and the foramen magnum was sealed tightly with polymethylmethacrylate (PMMA) to contain the intracranial contents during the impact tests. The mass of the head was then measured and recorded. The head was held in a net and suspended by a string at a prescribed height. The head was released by burning the string which allowed the head to free fall in its original orientation without initial translation or rotation onto a flat smooth anvil. The force-time

history was recorded by a Kistler 3-axis piezoelectric load cell and data were recorded using a digital acquisition system at 10,000 Hz (3 day old specimen) or 100,000 Hz (1 and 11 day old specimens). Each head was impacted at approximately 15cm and 30 cm drop heights. The actual height of each drop test was measured using a ruler. At each of these drop heights the head was impacted once on each of five locations: vertex, occiput, forehead, right parietal bone, and left parietal bone. All tests were conducted nondestructively so that the head specimens could be used in subsequent experiments.

Analysis - Each impact was digitally filtered according to the SAE J211b Class 1000 filter specifications for head impact. Acceleration of the head was calculated by dividing the force data by the measured head mass. Peak acceleration, pulse duration, and the head injury criterion (HIC) were calculated from the acceleration-time data. Pulse duration was defined from the first point where the impact force was greater than 5 N and the point where the force trace returned to zero. An average peak acceleration, duration, and HIC were calculated for each impact location and each height. Differences between location and height were determined using 2-way analysis of variance (ANOVA) and Tukey tests for multiple comparisons. The significance level for the statistical analysis was set at 5%.

Analytical models of pediatric head impact

The head impact data was represented by two different mathematical models. First, the force-time data was modeled as a simple mass-spring system. The governing equation of this system is:

$$m\ddot{x} + kx = 0 \quad (3)$$

where m is the head mass, x is the head displacement during impact, \ddot{x} is the acceleration of the head during impact, and k is the stiffness of the spring.

The second method of representing the head impact data involved coupling a head mass to a three parameter viscoelastic model. This model consisted of one spring in parallel with another spring and dashpot that are in series (Figure 1). This model has been used previously to describe the stress relaxation and creep behavior of biological tissues (Fung 1993). During an impact after free fall, the behavior of this viscoelastic system is described by the following differential equation:

$$\ddot{x} + \frac{k_2}{c}\dot{x} + \left(\frac{k_2}{m} + \frac{k_1}{m}\right)x + \frac{k_1k_2}{cm}x = 0 \quad (4)$$

where k_1 and k_2 are the spring constants and c is the coefficient of viscosity of the dashpot.

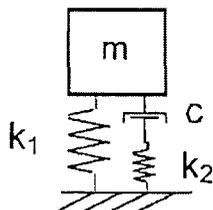


Figure 1: Schematic of three parameter solid model of head impact

For each impact, the spring constant k_1 was determined from the quasi-static (0.05 mm/s) head compression tests for that particular specimen. The value of k_1 for the occiput and forehead impacts was established as the linear stiffness from the quasi-static AP head compression tests. For the right and left parietal region impacts, k_1 was set to the linear stiffness from quasi-static RL direction compression experiments. Because head compression tests were not conducted in the superior-inferior direction, the average linear stiffness between the AP and RL directions were used as the k_1 value for the vertex impact models. The initial velocity of the head for both models was calculated using the measured drop height of the experiment.

These mass-spring and three parameter model differential equations were solved numerically to find the material constants for each drop (Matlab 6.1, The Mathworks, Inc.). The peak force of the measured data and the model solution of each drop were aligned by the half power method (Meirovitch 2001). The solutions of both models were optimized to find the material constants that minimize the residual sum of squares between the model solution and the measured data. The coefficient of determination (r^2) of the regressions used to determine the material constants were reported as well as the absolute percent error of the predicted peak head acceleration. The r^2 values of the three parameter model and mass-spring system solutions for each experiment were compared using a paired Student's t test.

For each specimen, a spring constant (k) and c and k_2 of the three parameter solid were found for each impact test. The relaxation time constant for the three parameter model was calculated as:

$$\tau_x = \frac{c}{k_2} \quad (5)$$

An average value for the spring constant and three parameter model time constant were averaged across height and impact location. Differences between each of these parameters were determined using 2-way analysis of variance (ANOVA) and Tukey tests for multiple comparisons. The significance level for the statistical analysis was set at 5%.

To validate the three parameter model, two sets of predictions were made and compared to the measured data. First, the force-time data of each 15 cm drop test were calculated using the material constants found from the 30cm drop data for the same specimen and impact location. Second, the material constants for the same specimen and impact location determined from the 15cm drop data were used to predict the force-time data of each 30cm drop. The predicted response and the measured data of each set of predictions were compared by determining r^2 for the pulse and absolute percent error of the calculated peak head acceleration. A Student's t test was used to compare differences between the r^2 values and peak acceleration error of the two prediction methods.

Anthropomorphic dummy head impact and compression

Experimental protocol- A series of drop tests were conducted to determine the impact response of the head of a Child Restraint and Airbag Interaction (CRABI) 6-month old anthropomorphic test device (ATD). Compression tests of the ATD head were performed to determine its static and dynamic structural properties. The CRABI head was compressed at 4 different constant velocities using the same protocol described above for the cadaver specimens. The dummy head was compressed to approximately the same forces measured in the cadaver experiments. The head was compressed in both the AP and RL directions.

Drop tests were also performed using the same protocol and instrumentation described above for the cadaver specimens. The head impacted a flat smooth anvil and the force-time history was recorded by a Kistler 3-axis piezoelectric load cell. The impact response was measured after drops from 15 and 30 cm onto 5 different impact sites on the head.

Analysis - As in the cadaver compression data analysis, each compression test was regressed between data taken from 50% through 100% maximum displacement in order to determine a stiffness value. The slope of this line was defined as the stiffness and the x-intercept as the low-load displacement. These analyses were repeated for each of the two compression directions and each of the

four velocities. An average stiffness and low-load displacement were calculated for each compression direction and velocity.

Each impact was digitally filtered according to the SAE J211b Class 1000 filter specifications for head impact. An average peak acceleration (peak force divided by head mass) and HIC were calculated for each impact location and each height. Differences between location and height were determined using 2-way ANOVA and Tukey tests for multiple comparisons.

RESULTS

Anthropometry

The average maximum head length, maximum head breadth, and head mass with mandible attached were 10.8 cm, 9.2 cm, and 620 grams respectively. The average location of the head center of gravity (Cg) was 0.4 cm anterior and 2.3 cm superior from the superior margin of the auditory meatus. The average location of the center of the occipital condyle was 1.4 cm posterior and 2.8 cm inferior from the Cg of the head. Average mass moment of inertia about the y-axis (line in the transverse direction) at the Cg was 4945 gm²cm². All anthropomorphic measurements for the three specimens are reported in Table A1.

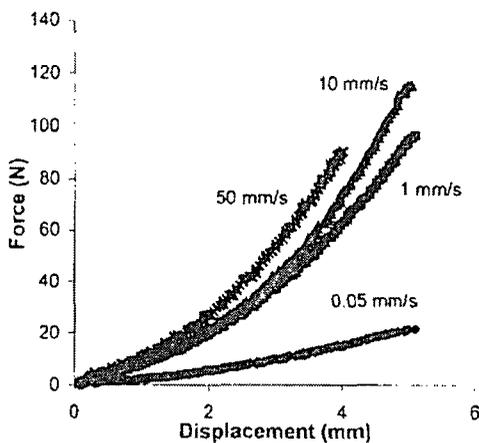


Figure 2: Typical infant cadaver force-deflection curves for head compressions at four different velocities in the AP direction (3 day old specimen).

Pediatric head compression

The force-deflection data for all tests displayed an initial toe region with increasing stiffness at the higher displacements (Figure 2). The linear regression of the data from 50% to 100% maximum deflection fit the data well with an average r^2 of 0.992 (Table A2). The exponential model also fit the individual responses well with an average r^2 of 0.998. A paired Student's t-test revealed that the r^2 value was significantly improved with an exponential fit ($p < 0.001$), however this difference is only 0.01 on average.

Statistical analysis revealed that the linear stiffness was dependent on deflection velocity ($p < 0.01$). The average stiffness values for the 0.05, 1.0, 10, and 50 mm/s constant velocity compression tests were 7.45, 23.3, 29.9, and 29.5 N/mm respectively (Table A2, Figure 3). The quasi-static (0.05 mm/s) stiffness was significantly less than the three higher rates however the 1.0, 10 and 50 mm/s stiffness values were not significantly different from each other. A change of velocity over three orders of magnitude (0.05 to 50 mm/s) increased the stiffness by a factor of four. Over a one order magnitude change of velocity from 1.0 to 10 mm/s, the stiffness increased only by a factor of 1.3 but this change was not statistically significant.

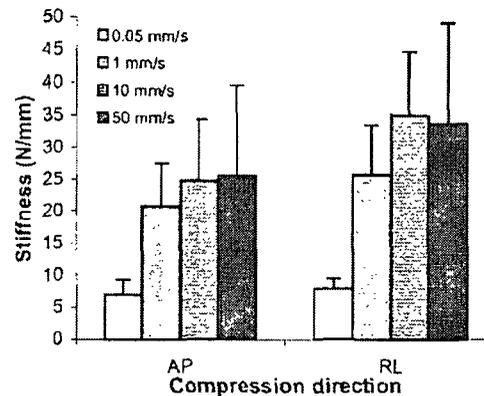


Figure 3: Stiffness values determined from head compression tests at different constant velocities in two different directions. The stiffness value determined from the quasi-static (0.05 mm/s) tests was significantly less than the three other higher velocities. Stiffness did not demonstrate a dependence on compression direction. Error bars indicate standard deviation.

The average low load displacement also showed dependence on the loading rate ($p < 0.01$). The average low load displacement across both directions for the 0.05, 1.0, 10, and 50 mm/s constant velocity compression tests were 1.1, 1.6, 1.6, and 1.2 mm respectively (Table A2). The quasi-static low load displacements were statistically different from the 1.0 and 10 mm/s experiments however this difference was only 0.5mm on average.

Stiffness and low load displacement did not demonstrate a dependence on compression direction. The average stiffness and low load displacement for anterior-posterior (AP) direction across all velocities were 18.9 N/mm and 1.40 mm respectively (Table A2). The average stiffness, and low load displacement for the right-left (RL) direction across all velocities were 24.8 N/mm and 1.39 mm respectively. The stiffness and low load displacement were not significantly different between the two compression directions ($p > 0.12$, Figure 3).

Pediatric head impact

Peak acceleration and HIC varied with drop height but did not change between impact location (Table A3, Figure 4). Average peak acceleration and HIC during the 30cm drop height impacts were 55.3 g and 84.1 respectively. Both these average values were significantly greater than the average peak acceleration and HIC values of 38.9 g and 32.9 respectively for the 15cm drop height impacts ($p < 0.001$, Figure 5). The acceleration pulse durations were not significantly different between the 15 cm and 30cm drop height impacts with an average pulse duration of 18.3 msec.

Peak acceleration and HIC measurements were not significantly different between any of the impact location sites ($p = 0.18$ and $p = 0.78$, Table A3). The peak acceleration and HIC for the impacts on the vertex were greater but not significantly different than impacts on the occiput, forehead, left and right parietal regions (Figures 6-7). Average pulse duration was longer for the vertex impacts, but again this difference was not statistically significant ($p = 0.89$). After all testing was complete, the specimens were dissected and no skull fractures or other permanent deformation was identified.

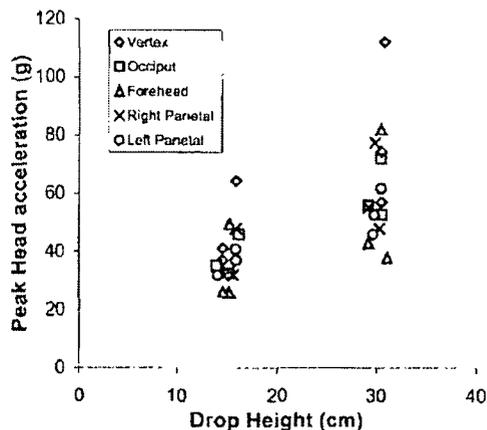


Figure 4: Peak acceleration and drop height for all infant cadaver impact tests.

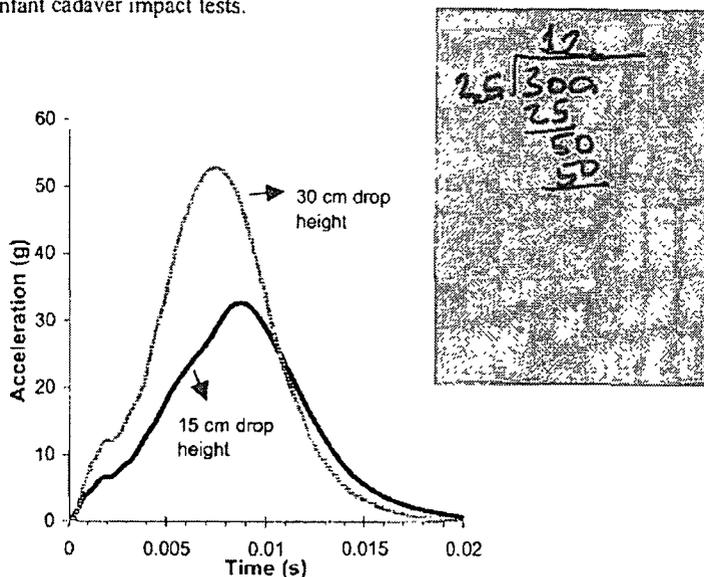


Figure 5: Typical infant cadaver head acceleration pulses from two different drop heights onto the same impact location (occiput, 11 day old specimen).

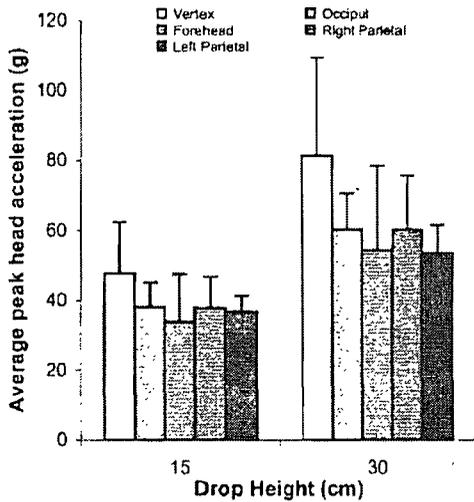


Figure 6: Average peak head acceleration at each height and impact location. Average peak accelerations were significantly different between drop height but did not vary between impact locations. Error bars indicate standard deviation.

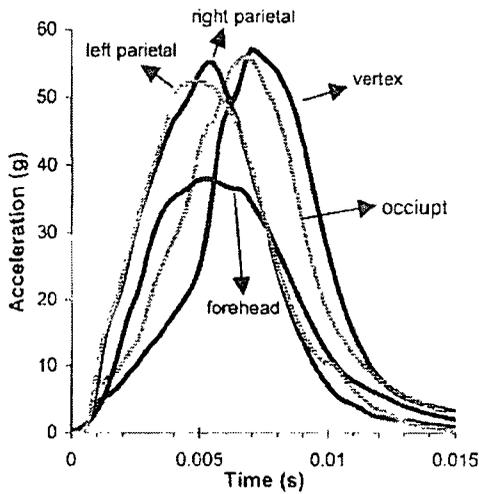


Figure 7: Typical infant cadaver head acceleration pulses from the same drop height (30 cm) onto the five different impact locations (1 day old specimen).

Analytical models of pediatric head impact

The mass-spring model was unable to fully represent the measured impact data (Figure 8). The average r^2 value across all tests was 0.81 ranging from 0.71 to 0.89. The absolute percent error of the predicted peak head acceleration was 16% on average with 21 out of 30 impact models over estimating the peak acceleration. The pulse duration of the model was shorter than the cadaver data for all impacts with an average percent error of 28%. Statistical analysis revealed that the spring constant (k) did not significantly change with impact location but did show a dependence with drop height (Table A4). The values of k for the occipital impacts were slightly lower than the other four locations but this difference was not statistically significant ($p=0.663$). The spring constant for the 30cm drop heights were an average of 1.4 times greater than the 15cm impacts ($p<0.05$). The average spring constant across all impact locations was 29.4 N/mm for the 15cm drops and 41.1 N/mm for the 30cm drops.

The three parameter model resulted in a better representation of the impact data than the mass-spring system (Figure 8). The average r^2 value for the three parameter model was 0.93 with a range of 0.83 to 0.98. Twenty eight out of the thirty impact models underestimated the peak head acceleration resulting in an average absolute percent error of 9.5%. The absolute percent error of the predicted pulse duration was 36% on average with all the impacts models predicting shorter durations than the cadaver data. The r^2 values for the three parameter models were significantly greater than the r^2 values for the mass-spring fit in every test with an average increase of 0.12.

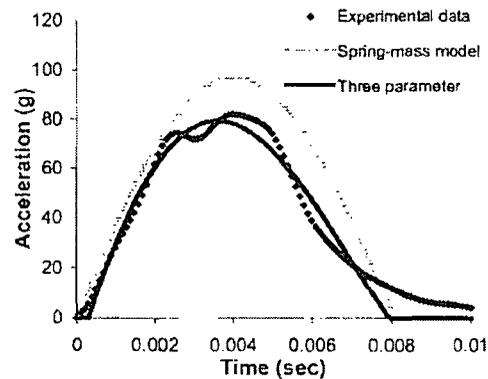


Figure 8: Typical infant cadaver head acceleration pulses with corresponding mass-spring and three parameter model estimations (3 day old specimen, forehead impact from 30 cm).

The relaxation time constant did not show a dependence on impact location and drop height (Table A5). The average values of τ_x in the vertex impacts were less than the other locations however this was not statistically different ($p=0.23$). The time constant for the 30 cm drops were shorter than the 15 cm drops but this difference was not significant ($p=0.45$).

The three parameter model was able to predict the measured data of one height given the parameters determined from the other drop height. Using the k_1 , k_2 and c values for the 15cm impacts of each specimen and location, the predicted 30cm impacts resulted in an average r^2 value of 0.87. The absolute percent error of the predicted peak acceleration was 19% on average. The predicted 15cm impacts using the 30cm drop height parameters resulted in higher r^2 values ($p<0.01$) and lower peak acceleration errors ($p<0.001$) when compared to the first set of predictions. This second method of predicting the lower height impact response from the higher height impact model parameters showed an average r^2 value of 0.92 with an average absolute peak acceleration error of 7.1%.

Anthropomorphic dummy head impact and compression

ATD peak acceleration and HIC varied with drop height and impact location (Table A6). Peak acceleration during the CRABI 30 cm drop height impacts onto the vertex, occiput, forehead, right parietal and left parietal region were 51.8, 61.6, 84.8, 144.9, and 214.5 g respectively. Impacts from 15 cm revealed peak accelerations of 33.8, 39.5, 37.3, 66.8, and 116.0 g onto the vertex, occiput, forehead, right parietal and left parietal region respectively. These values were significantly less than the peak accelerations for the 30 cm drop height impacts. Also, peak accelerations were significantly higher during the right and left parietal impacts than impacts to the occiput and vertex (Figure 9). Along with the increase in peak acceleration, the acceleration pulse for the right and left parietal impacts were shorter in duration than the other impact location (Figure 10). The dummy behaved similar to a mass-spring system with the pulse duration decreasing only slightly during the 30 cm drop tests.

The linear regression of the compression data fit the data well with an average r^2 of 0.999. Stiffness of the CRABI head increased with higher compression velocity. The stiffness values for the 0.05, 1.0, 10, 10,

and 50 mm/s constant velocity AP compression tests were 18.5, 22.4, 26.4, and 28.7 N/mm respectively (Table A7). The RL compression direction revealed stiffness values of 62.4, 82.9, 95.1 and 95.7 N/mm for 0.05, 1.0, 10, and 50 mm/s constant velocities respectively. The stiffness across all velocities for RL direction was approximately 2.5 times greater on average than the AP direction stiffness (Table A7). The average low load displacement showed little change with loading rate or compression direction.

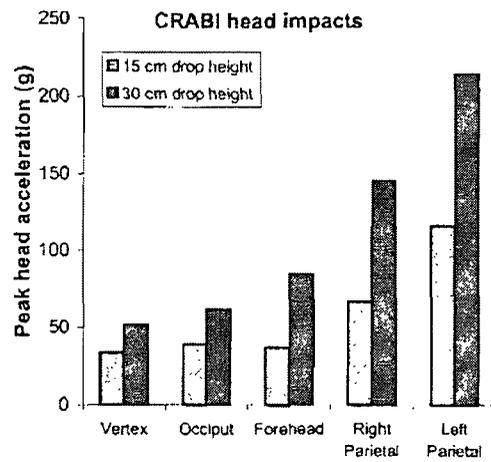


Figure 9: CRABI peak head acceleration for 5 impact locations from 15 cm and 30 cm drop heights. CRABI accelerations increased with drop height and lateral impact accelerations were greater than impacts on the vertex and occiput at both drop heights.

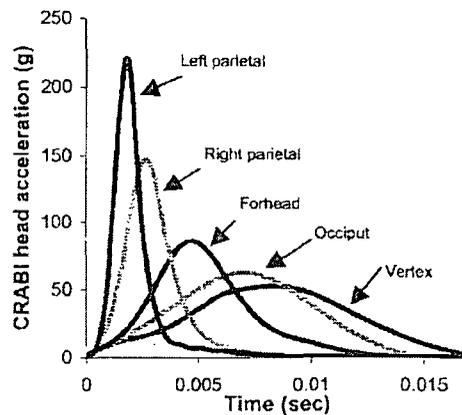


Figure 10: CRABI head accelerations from drop heights of 30cm onto the five different impact locations.

DISCUSSION

Limitations

One limitation of this study is the small number of specimens tested. Because pediatric cadaveric tissue is rarely available, these three specimens represent all the appropriate infant specimens that have been procured. However, with the paucity of pediatric biomechanical data, these experiments provide previously unavailable information about the human infant head dynamic response. Indeed, these pediatric results provide the first set of human head biomechanical data for the validation of scaling methods, animal experiments, computational models, and anthropomorphic dummy biofidelity.

Because pediatric cadaveric tissue is rarely available, this tissue was also used in a series of biomechanical experiments beyond the scope of this paper. For this reason compression and impact tests were conducted on the head specimens at sub-failure loads in order to ensure that no bone fracture or permanent skull deformation occurred. Therefore these studies examine the mechanics at sub-failure levels and do not assess the fracture biomechanics of the pediatric skull. Because no fractures occurred as a result of the drop tests, these results indicate that a peak acceleration of 55 g and a HIC value of 84 are below the infant skull fracture tolerance.

Another limitation of these studies is the boundary condition at the foramen magnum during the compression and impacts tests. Normally the brain and spinal cord are surrounded by cerebral spinal fluid (CSF) and the CSF flows between the intracranial cavity and the spinal canal through the foramen magnum. In our experimental protocol the cervical spine was separated from the head during a previous test. Also, the cadaveric tissue was frozen and thawed resulting in significant changes in the brain tissue properties. Previous studies show that the incompressible nature of the intracranial contents significantly affects the head impact response (Klinich et al. 2002). To simulate the incompressible brain material and CSF in our experiments, the head was filled with water and sealed to prevent extrusion of the intracranial contents. Similar methods have been used during adult head impact drop tests to replicate the incompressible nature of the head (Ono et al. 1980).

The head compression tests were conducted at significantly lower rates than impacts conditions. Because the intracranial contents are incompressible, tissue extrusion and CSF flow out of the foramen magnum could be possible during these slow, non-

impact deformation rates. To simulate this condition, the foramen magnum was open during the compression tests allowing the free movement of tissue out of the foramen magnum. Adult head compression tests have used a similar method to simulate the in vivo foramen magnum boundary condition (Thomas et al. 1968).

The cadaveric specimens tested in this study were newborn infants and younger than the designed age of the 6-month CRABI dummy. Scaling methods were used to design the child ATD head response based on cranial bone stiffness. Cranial bone has been shown to increase in stiffness from birth to 6-months of age (McPherson and Kriewall 1980; Melvin 1995; Irwin and Mertz 1997). Therefore, according to scaling methods, the predicted CRABI accelerations should be approximately 15% greater than the infant response.

Analytical models of pediatric head impact

This study presents a viscoelastic model to characterize the rate dependent structural properties of the infant head during impact. The three parameter model resulted in a better representation of the infant impact data than a mass-spring model (Figure 8). The three parameter model had a significantly higher r^2 value and lower error in predicting the peak acceleration. The average r^2 value for the mass-spring model was 0.81 demonstrating that the acceleration pulse is not well represented as sine wave that the mass-spring model predicts. However the average r^2 value for the three parameter model was 0.93 showing that this model better represented the shape of acceleration data. The additional parameter afforded the model the opportunity to fit both the magnitude and duration of the acceleration pulse independently for the three parameter model while the both of these parameters are coupled by the spring constant in the mass-spring model. Also, the three parameter model includes a viscous component and can better replicate the damped unloading response observed in the impact data.

Head impact

Comparison of the infant head impact results to previous adult data are complicated because of differences in experimental design and acceleration calculation methods. In some previous impact tests of adult head, accelerometers were attached to the skull and the head acceleration was measured directly. That method was not feasible with the infant specimens because the thin, pliable cranial bone made the fixation of accelerometers to the pediatric skull extremely difficult. Also, because of the broad, compliant sutures joining the cranial bones, the infant skull is not a rigid body and accelerometers would not produce an accurate measurement of head Cg acceleration. Therefore, infant head accelerations during drop tests were calculated using the measured force at impact and the mass of head.

Owing to the difference in acceleration calculation methods, comparison of our infant data were limited to those studies in which head accelerations could be estimated from impact force and average head mass reported in the literature. Average peak acceleration measured in the infant cadaver were 55 g with an average pulse duration of 18 msec during the 30 cm drop tests. Ono et al. reported an average peak head acceleration of 206 g and average duration of 2.2 msec during drops tests of adult heads from more than 40 cm onto a flat anvil that did not result in skull fracture (Ono et al. 1980). Nightingale et al. measured an average peak head acceleration of 167 g and pulse duration of 5 msec during cadaver drop tests from 53 cm (Nightingale et al. 1996). Average peak head acceleration and duration measured by Hodgson and Thomas using a rotating pallet were 172 g and 7msec respectively (Hodgson and Thomas 1971). The equivalent drop height of these tests were an average of 57 cm and all the tests resulted in skull fractures (Hodgson and Thomas 1971; Mertz 1985). All of these adult responses had greater peak acceleration and shorter pulse durations than the infant cadaver head impact response from drop heights greater than the heights used in the infant cadaver protocol. Assuming linearity, while peak acceleration will vary with impact velocity, pulse duration is independent of impact velocity. On this basis, the first-mode vibration frequency of the infant skull is significantly lower than the adult.

Other adult drop tests from similar heights to the pediatric experiments have been reported. Head acceleration from accelerometer data is reported by Hodgson and Thomas at equivalent heights of approximately 15 and 30 cm (Hodgson and Thomas 1971; Mertz 1985). Eight tests were conducted from

15±4 cm resulting in an average peak head acceleration of 107 g. Tests conducted at 30±3cm produced 205 g average peak acceleration with three out of the four tests resulting in skull fracture. Although the adult measurements were taken from accelerometer data, these values are considerably greater than the infant drop test results.

The majority of the adult head data were determined during impact to the forehead of the cadaver. Ono et al. investigated impacts to both the forehead and occiput (Ono et al. 1980). These data show no difference in the peak accelerations between the two impact locations. The infant cadaver also showed no dependence of the response with impact location.

Head compression

The infant cadaver showed an increase in structural head stiffness with an increase in rate of compression and revealed no directional dependence of stiffness over the range of velocities tested. In contrast, Hodgson et al. reported AP stiffness of the adult head during static flat plate compression to be approximately 50% greater than the lateral direction (Hodgson et al. 1967). Thomas et al. also found differences in the volume change due to compression in different directions (Thomas et al. 1968). In contrast, the infant cadaver data did not reveal statistically significant changes in stiffness between the AP and lateral compression at any velocity.

The stiffness values for adult head compression are dramatically different from the infant data. Thomas et al. reported a stiffness of approximately 2975 N/mm during static, point load compression in the AP direction of an adult cadaveric head. Adult stiffness values measured by Hodgson et al. during static compression using flat plates were 1590 N/mm in the AP direction and 1070 N/mm in the lateral direction. Dynamic point load stiffness was reported to be 7360 N/mm. The average compressive stiffness of the infant cadaver head, 22 N/mm, is approximately one to two orders of magnitude less than the adult values. Again, this shows the dramatically more compliant nature of the infant head.

Scaling of adult head impact data

To account for the compliant infant head in the absence of cadaver impact data, scaling techniques have been used to predict the pediatric response. These methods use differences in head size and cranial bone stiffness to scale adult impact data to the child. To examine the validity of these methods, the adult peak acceleration data reported in the literature were scaled using dimensional analysis described by Melvin (1995). Anthropomorphic data and cranial bone stiffness for adult and newborn ages result in ratios of 0.70 and 0.25 for the characteristic head length and skull stiffness respectively (Hubbard 1971; McPherson and Kriewall 1980; Schneider et al. 1986; Irwin and Mertz 1997). The ratio of the accelerations is directly proportional to the ratio of skull bending stiffness and inversely proportional to the characteristic head length ratio (Melvin 1995). Using this method, the adult head impact accelerations outlined above were scaled to the infant resulting in accelerations of 75 to 60 g (Hodgson and Thomas 1971; Ono et al. 1980; Mertz 1985; Nightingale et al. 1996). These adult data were from drop heights greater than the infant cadaver tests (Figure 11). However, the scaled values are only slightly higher than the measured infant cadaver accelerations from drops of 30 cm. Thus, these cadaver data provide support and validity to scaling methods currently in use to predict the infant impact response.

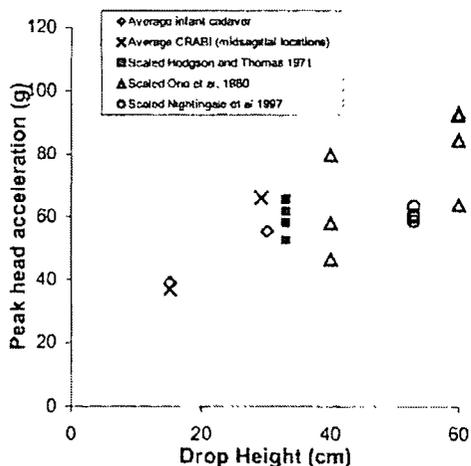


Figure 11: Peak acceleration and drop height for scaled adult cadaver, average CRABI (vertex, occiput, forehead), and average infant cadaver head impact tests. Filled symbols indicate skull fracture occurred as a result of impact and open symbols how non-fracture data.

Comparisons of cadaver and CRABI responses

With the absence of human pediatric cadaver data, scaling methods were used to design the child anthropomorphic test dummies (Irwin and Mertz 1997). To assess the biofidelity of the youngest child dummy, a 6-month old CRABI dummy head was tested using the same protocol as the infant cadaveric tissue. The CRABI head impact response was statistically similar to the cadaver data during the vertex, occiput, and forehead impacts from both 15 cm and 30 cm drops. However, significant differences were found between the lateral impacts when compared to the cadaver data. The peak acceleration and HIC values for the CRABI drops were not significantly different from the cadaver data during impacts onto the vertex, occiput, and forehead at both drop heights (t -test, $p > 0.20$, Figures 12-13). The right and left parietal CRABI head impacts resulted in significantly higher peak accelerations and HIC values ($p < 0.05$). HIC results were two to five times higher than the cadaver values for these lateral impacts. Both the cadaver and CRABI impacts did not show a change in pulse duration with increasing drop height. Acceleration pulse duration for the CRABI head drops were lower than the corresponding average cadaver response for all impact locations and heights however these differences were not statistically significant.

The compression tests showed similar results as the impact experiments. The CRABI stiffness values for the 1, 10, and 50 mm/s in the AP direction constant velocity tests were statistically similar to the infant cadaver results (Figure 14). In contrast, the dummy RL direction 0.05, 1, and 10 mm/s constant velocity tests were found to be approximately 3 to 8 times stiffer than the infant cadaver experiments (Figure 15). The quasi-static (0.05 mm/s) compression stiffness in both directions were significantly greater than the quasi-static stiffness of the cadaver specimens ($p < 0.05$). These data show that the CRABI 6-month dummy behaves similar to the infant cadaver for impacts to the vertex, occiput, and forehead. However, the dummy is dramatically stiffer than the human infant in response to a lateral impact.

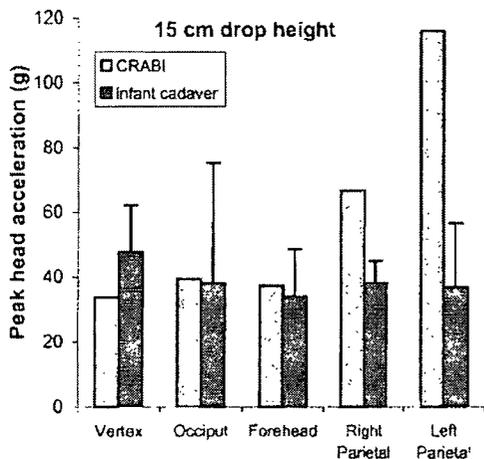


Figure 12: CRABI and infant cadaver peak head acceleration for 5 impact locations from 15 cm drop height. CRABI acceleration was similar to the cadaver data for the vertex, occiput, and forehead impacts but significantly greater for the lateral impact. Error bars indicate standard deviation.

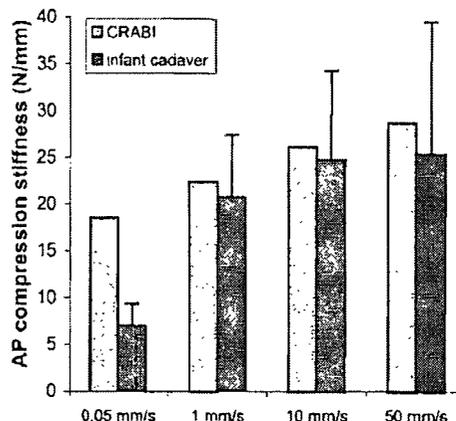


Figure 14: CRABI and infant cadaver head compression stiffness for the four constant velocity tests in the AP direction. CRABI results were statistically similar to the 1, 10, and 50 mm/s cadaver data.

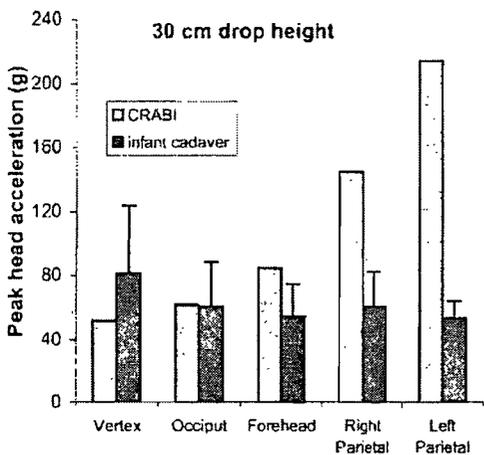


Figure 13: CRABI and infant cadaver peak head acceleration for 5 impact locations from 30 cm drop height. CRABI acceleration was similar to the cadaver data for the vertex, occiput, and forehead impacts but significantly greater for the lateral impact. Error bars indicate standard deviation.

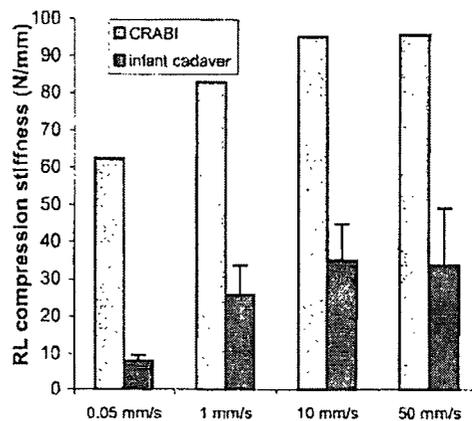


Figure 15: CRABI and infant cadaver head compression stiffness for the four constant velocity tests in the RL direction. CRABI stiffness values were significantly greater to the 0.05, 1, and 10 mm/s cadaver results.

CONCLUSIONS

This study reports the dynamic response, compression structural properties, and anthropometry of the human infant head. Drop tests revealed that the impact response of the infant head showed no dependence on impact location. Also, the structural stiffness of the infant head did not depend on compression direction. The compression tests revealed that the stiffness was dependent on velocity indicating the infant head behaves as a viscoelastic material.

These data show the infant head is dramatically more compliant than the adult. Previous adult head impact data had significantly greater peak acceleration and shorter pulse durations than the infant head impact response. Also the compressive stiffness of the adult cadaver head is considerably greater than the infant values. The infant cadaver impact tests showed similar results as scaled adult data and provide validity to current scaling methods to predict the infant impact response.

These data also show that the CRABI 6-month dummy response is similar to the infant cadaver for impacts to the vertex, occiput, and forehead. However, the dummy is dramatically stiffer than the human infant in response to lateral impacts. These pediatric head anthropomorphic, compression, and impact data will provide a basis to validate whole head models and compare with ATD performance in similar exposures.

ACKNOWLEDGMENTS

Funding for this project was provided by the National Highway Traffic Safety Administration, and the Southern Consortium for Injury Biomechanics at the University of Alabama, Injury Control Research Center. The 6-month CRABI dummy was provided by Wayne State University.

REFERENCES

- Burdi, A., Huelke, D., Snyder, R. and Lowrey, G. (1969) Infants and children in the adult world of automobile safety design: pediatric and anatomical considerations for design of child restraints. *Journal of Biomechanics* 2: 267-280.
- CDC (1990) Childhood injuries in the United States. *American Journal of Diseases of Children* 144(6): 627-46.
- Dobbing, J. (1981) The later development of the brain and its vulnerability. In *Scientific Foundations of Paediatrics*, ed. J. Davis and J. Dobbings, Heinemann Medical, London.
- Fung, Y. (1993) *Biomechanics: Mechanical Properties of Living Tissues*. Springer-Verlag, New York.
- Gotschall, C. and Luchter, S. (1999) Head injuries to motor vehicle occupants aged 0-5 years. Proc. Child occupant protection in motor vehicle crashes session. AAAM and IRCOBI, pp. p17-27. Professional Engineering Publishing.
- Greencs, D. S. and Schutzman, S. A. (1999) Clinical indicators of intracranial injury in head-injured infants. *Pediatrics* 104(4 Pt 1): 861-7.
- Hodgson, V., Gurdjian, E. and Thomas, L. (1967) Development of a model for the study of head injury. Proc. 11th Stapp Car Crash Conference, pp. 432-443. Society of Automotive Engineers, Warrendale, PA.
- Hodgson, V. and Patrick, L. (1968) Dynamic response of the human cadaver head compared to a simple mathematical model. Proc. 12th Stapp Car Crash Conference, pp. 280-301. Society of Automotive Engineers, Warrendale, PA.
- Hodgson, V. and Thomas, L. (1971) Comparison of head acceleration injury indices in cadaver skull fracture. Proc. 15th Stapp Car Crash Conference, pp. 190-206. Society of Automotive Engineers, Warrendale, PA.
- Hubbard, R. P. (1971) Flexure of layered cranial bone. *Journal of Biomechanics* 4(4): 251-63.
- Hubbard, R. P., Melvin, J. W. and Barodawala, I. T. (1971) Flexure of cranial sutures. *Journal of Biomechanics* 4(6): 491-6.
- Irwin, A. and Mertz, H. (1997) Biomechanical basis for the CRABI and Hybrid III child dummies. Proc. 41st Stapp Car Crash Conference, pp. 261-272. Society of Automotive Engineers, Warrendale, PA.
- James, H. E. (1999) Pediatric head injury: what is unique and different. *Acta neurochirurgica Supplementum*. 73: 85-8.
- Klinich, K., Hulbert, G. and Schneider, L. (2002) Estimating infant head injury criteria and impact response using crash reconstruction and finite element modeling. *Stapp Car Crash Journal* 46: 165-194.

- Kraus, J. F., Rock, A. and Hemyari, P. (1990) Brain injuries among infants, children, adolescents, and young adults. *American Journal of Diseases of Children* 144(6): 684-91.
- Kriewall, T., McPherson, F. and Tsai, A. (1981) Bending properties and ash content of fetal cranial bone. *Journal of Biomechanics* 14: 73-79.
- Luerssen, T. (1993) General characteristics of neurologic injury. In *Pediatric Trauma*, M. Eichelberger, Mosby Year Book, Inc., St. Louis.
- Margulies, S. S. and Thibault, K. L. (2000) Infant skull and suture properties: measurements and implications for mechanisms of pediatric brain injury. *Journal of Biomechanical Engineering* 122(4): 364-71.
- McElhaney, J., Roberts, V. and Hilyard, J. (1976) *Handbook of Human Tolerance*. Japan Automobile Research Institute, Tokyo.
- McPherson, G. and Kriewall, T. (1980) The elastic modulus of fetal cranial Bone: a first step toward understanding of the biomechanics of fetal head molding. *Journal of Biomechanics* 13: 9-16.
- Meirovitch, L. (2001) *Fundamentals of Vibrations*. McGraw-Hill, Boston.
- Melvin, J. (1995) Injury Assessment Reference Values for the CRABI 6-Month Infant Dummy in a Rear-Facing Infant Restraint with Airbag Deployment. SAE 950872. Society of Automotive Engineers, Warrendale, PA.
- Mertz, H. (1985). Biofidelity of the Hybrid III head, SAE 851245. Society of Automotive Engineers, Warrendale, PA.
- Mertz, H., Irwin, A., Melvin, J. W., Stalnaker, R. L. and Beebe, M. S. (1989) Size, weight and biomechanical impact response requirements for adult size small female and large male dummies. In *Automotive Frontal Impacts*, pp. 133-144. Society of Automotive Engineers, Warrendale, PA.
- Myers, B. S. (1997). An evaluation of a helmet standard for children, United States Consumer Product Safety Commission.: Republished in part in the U.S. Federal Register, 16 CFR Part 1203, 11711-11747, 1998.
- Nightingale, R. W., McElhaney, J. H., Richardson, W. J. and Myers, B. S. (1996) Dynamic responses of the head and cervical spine to axial impact loading. *Journal of Biomechanics* 29(3): 307-18.
- Ommaya, A., Yarnell, P., Hirsch, A. and Harris, E. (1967) Scaling of experimental data on cerebral concussion in sub-human primates to concussion threshold for man. Proc. 11th Stapp Car Crash Conference, pp. 73-80. Society of Automotive Engineers, Warrendale, PA.
- Ono, K., Kikuchi, A., Nakamura, M., Kobayashi, H. and Nakamura, N. (1980) Human head tolerance to sagittal impact reliable estimation deduced from experimental head injury using subhuman primates and human cadaver skulls. Proc. 24th Stapp Car Crash Conference, pp. 103-160. Society of Automotive Engineers, Warrendale, PA.
- Prasad, P. and Daniel, R. (1984) A biomechanical analysis of head, neck, and torso injuries to child surrogates due to sudden torso acceleration. Proc. 28th Stapp Car Crash Conference, pp. 25-40. Society of Automotive Engineers, Warrendale, PA.
- Ridgway, E. B. and Weiner, H. L. (2004) Skull deformities. *Pediatric Clinics of North America* 51(2): 359-87.
- Schneider, L., Lehman, R., Pflug, M. and Owings, C. (1986). Size and shape of the head and neck from birth to four years. UMTRI-86-2 University of Michigan Transportation Research Institute. Ann Arbor, MI.
- Thibault, K. and Margulies, S. (1998) Age-dependent material properties of the porcine cerebrum: effect on pediatric inertial head injury criteria. *Journal of Biomechanics* 31: 1119-1126.
- Thomas, L., Sezgin, Y., Hodgson, V., Cheng, L. and Gurdjian, E. (1968) Static deformation and volume changes in the human skull. Proc. 12th Stapp Car Crash Conference, pp. 260-270. Society of Automotive Engineers, Warrendale, PA.
- Walker, L., Harris, E. and Pontius, U. (1973) Mass, volume, center of mass, and mass moment of inertia of head and head and neck of human body. Proc. 17th Stapp Car Crash Conference, pp. 525-537. Society of Automotive Engineers, Warrendale, PA.

APPENDIX

Table A1: Anthropomorphic data for infant cadaver head specimens with mandible attached.

	Specimen			6 month CRABI ¶	
Specimen ID	P03	P05	P06		
Age (days after birth)	3	1	11	-	
Head mass (gm)	491.5	666.5	701.9	2110	
Body mass (gm)	1910	1881*	2583	7820	
Head length (cm) ‡	10.3	10.8	11.2	15.6	
Head breadth (cm) ‡	8.5	8.8	10.4	11.9	
Head height (cm) §	7.5	8.8	9.2	-	
facial length (cm) §	4.2	5.1	4.8	-	
Bizygomatic diameter (cm) §	5.5	6.2	5.9	-	
Anterior condyle	x (cm) §	-0.6	-0.7	-0.5	-
	z (cm) §	0.7	0.6	0.5	-
Posterior condyle	x (cm) §	-1.2	-1.4	-1.1	-
	z (cm) §	0.6	0.4	0.5	-
Center of condyle	x (cm)	-0.9	-1.0	-0.8	-
	z (cm)	0.7	0.5	0.5	-
Head Cg	x (cm)	-0.2	0.4	1.0	-
	z (cm)	-1.1	-2.9	-2.9	-
Head Iyy at Cg (gm*cm ²)	4813	5172	4850	-	

* body mass without limbs, ‡ measurements using calipers, § measurements from CT data.

¶ CRABI anthropometry from Irwin and Mertz 1997

Coordinate system for Cg and condyle measurements: origin at superior margin of auditory meatus, positive x direction in anterior direction along Frankfort, z positive in inferior direction.

Head height: length from auditory meatus to top of head, facial length: length between nasion and gnathion,

Table A2: Regression results from cadaver head compression tests in different directions and velocities.

Compression direction	Velocity	Specimen	Measured velocity (mm/s)	Linear fit			Exponential fit		
				Stiffness (N/mm)	Low load Displacement (mm)	r^2	A (N)	B (mm^{-1})	r^2
Anterior-posterior	0.05 mm/s	P03	0.05	5.6	1.2	0.99	9.0	0.25	1.00
		P05	0.05	9.7	1.1	1.00	16.2	0.25	1.00
		P06	0.05	5.5	1.6	0.99	4.6	0.32	1.00
	1.0 mm/s	P03	1.0	26.7	1.6	0.99	22.0	0.34	1.00
		P05	1.0	13.5	1.3	0.99	20.6	0.25	1.00
		P06	1.0	22.1	1.5	1.00	27.7	0.27	1.00
	10 mm/s	P03	10	34.8	1.8	0.99	17.2	0.41	1.00
		P05	10	15.8	1.2	0.99	28.9	0.23	1.00
		P06	10	23.8	1.9	0.99	14.4	0.37	1.00
	50 mm/s	P03	67	35.4	1.5	0.99	20.0	0.43	1.00
		P05	64	15.4	0.7	0.99	44.3	0.19	1.00
	Right-Left	0.05 mm/s	P03	0.05	9.8	1.0	1.00	17.9	0.25
P05			0.05	6.8	1.2	0.99	8.2	0.30	1.00
P06			0.05	7.2	0.7	0.98	24.3	0.17	0.99
1.0 mm/s		P03	1.0	34.7	1.6	1.00	16.2	0.45	1.00
		P05	1.0	21.1	1.7	0.99	6.3	0.53	1.00
		P06	1.0	21.3	1.8	0.98	7.6	0.49	1.00
10 mm/s		P03	10	44.7	1.6	0.99	20.0	0.46	1.00
		P05	10	25.4	1.7	0.99	9.0	0.50	1.00
		P06	10	34.9	1.7	0.99	15.4	0.45	1.00
50 mm/s		P03	58	44.6	1.2	1.00	30.6	0.41	1.00
		P05	55	22.8	1.2	0.99	14.3	0.44	0.99

Table A3: Results from cadaver head drop tests onto different impact locations and at 2 different heights.

Approximate Drop height	Location	Specimen	Measured drop height (cm)	Head mass* (gm)	Peak acceleration (g)	Duration (msec)	HIC
15cm	Vertex	P03	15.9	415.3	64.5	13.7	64
		P05	14.6	601.1	37.3	26.0	29
		P06	14.6	646.7	41.3	23.9	28
	Occiput	P03	16.2	415.3	46.0	12.9	48
		P05	14.0	601.1	35.4	22.5	24
		P06	15.2	646.7	32.7	23.5	25
	Forehead	P03	15.2	415.3	49.5	12.9	55
		P05	15.2	601.1	26.1	22.6	17
		P06	14.6	646.7	26.3	23.4	20
	Right Parietal	P03	15.9	415.3	48.0	12.8	49
		P05	15.6	601.1	32.2	25.0	24
		P06	15.2	646.7	33.4	20.1	22
	Left Parietal	P03	15.9	415.3	41.0	13.0	39
		P05	15.9	601.1	37.2	19.1	27
		P06	14.1	646.7	32.1	20.8	22
30cm	Vertex	P03	30.8	415.3	112.4	11.7	205
		P05	30.5	601.1	57.1	22.1	71
		P06	30.5	646.7	74.6	20.1	96
	Occiput	P03	30.5	415.3	72.1	12.8	109
		P05	29.2	601.1	55.9	18.8	71
		P06	30.5	646.7	52.9	20.6	75
	Forehead	P03	30.5	415.3	82.1	12.0	179
		P05	31.1	601.1	38.0	22.1	38
		P06	29.2	646.7	43.1	20.7	47
	Right Parietal	P03	29.8	415.3	77.6	12.9	138
		P05	29.2	601.1	55.2	18.5	70
		P06	30.3	646.7	48.0	17.0	59
	Left Parietal	P03	30.5	415.3	61.9	12.3	113
		P05	29.8	601.1	52.6	17.4	71
		P06	29.7	646.7	46.1	18.0	64

* head mass was measured and tests were conducted with the mandible removed from the specimen.

Table A4: Coefficients and results of modeling cadaver head impact tests as a mass-spring system (see equation 3)

Approximate Drop height	Location	Specimen	k (N/mm)	predicted peak acceleration (g)	duration (msec)	r^2
15cm	Vertex	P03	37.9	54.3	10.4	0.79
		P05	26.1	35.9	15.1	0.83
		P06	24.7	33.7	16.1	0.78
	Occiput	P03	29.3	48.2	11.8	0.85
		P05	22.1	32.4	16.4	0.82
		P06	21.9	32.4	17.1	0.86
	Forehead	P03	39.2	54.2	10.2	0.85
		P05	27.0	37.3	14.8	0.75
		P06	21.6	31.6	17.2	0.84
	Right Parietal	P03	37.4	54.0	10.4	0.85
		P05	37.1	44.3	12.7	0.79
		P06	21.7	32.2	17.1	0.75
	Left Parietal	P03	34.0	51.5	10.9	0.79
		P05	33.1	42.2	13.4	0.80
		P06	28.1	35.3	15.1	0.83
30cm	Vertex	P03	70.9	103.6	7.6	0.82
		P05	30.3	56.0	14.0	0.79
		P06	36.8	59.5	13.2	0.77
	Occiput	P03	36.8	74.3	10.5	0.81
		P05	35.6	59.4	12.9	0.82
		P06	30.3	54.0	14.5	0.85
	Forehead	P03	62.9	97.0	8.0	0.89
		P05	30.7	56.9	13.9	0.71
		P06	23.4	46.4	16.5	0.78
	Right Parietal	P03	46.2	82.3	9.4	0.85
		P05	49.2	69.8	11.0	0.81
		P06	34.2	57.2	13.7	0.79
	Left Parietal	P03	42.4	79.7	9.8	0.80
		P05	51.0	71.8	10.8	0.79
		P06	35.0	57.3	13.5	0.82

Table A5: Coefficients and results of modeling cadaver head impact tests as a three parameter model (see equation 4).

Approximate Drop height	Location	Specimen	c (N*sec/mm)	k ₂ (N/mm)	k ₁ (N/mm)	predicted peak acceleration (g)	duration (msec)	r ²
15cm	Vertex	P03P	0.276	74.1	7.7	55.8	7.4	0.91
		P05P	0.187	55.0	8.3	34.9	11.0	0.93
		P06P	0.233	55.6	6.4	34.7	11.1	0.88
	Occiput	P03P	0.218	28.5	5.6	40.2	11.3	0.95
		P05P	0.141	36.6	9.7	30.5	12.9	0.93
		P06P	0.201	29.7	5.5	29.2	14.3	0.95
	Forehead	P03P	0.239	57.5	5.6	47.6	8.5	0.96
		P05P	0.093	49.4	9.7	27.4	15.2	0.97
		P06P	0.136	23.9	5.5	24.5	16.4	0.97
	Right Parietal	P03P	0.174	30.5	9.8	42.6	10.5	0.96
		P05P	0.136	62.2	6.8	31.1	12.7	0.98
		P06P	0.155	31.4	7.2	28.6	14.3	0.83
	Left Parietal	P03P	0.116	33.1	9.8	38.7	10.9	0.97
		P05P	0.166	41.8	6.8	32.4	12.5	0.94
		P06P	0.177	30.8	7.2	28.6	14.0	0.90
30cm	Vertex	P03P	0.345	131.7	7.7	97.0	5.7	0.92
		P05P	0.192	78.6	8.3	53.8	10.0	0.93
		P06P	0.301	110.2	6.4	64.0	8.3	0.89
	Occiput	P03P	0.217	40.8	5.6	60.2	9.8	0.90
		P05P	0.186	54.5	9.7	50.6	10.9	0.94
		P06P	0.254	47.3	5.5	48.9	11.7	0.95
	Forehead	P03P	0.363	68.6	5.6	79.4	7.6	0.97
		P05P	0.105	67.1	9.7	41.7	14.5	0.94
		P06P	0.147	22.8	5.5	35.3	16.3	0.84
	Right Parietal	P03P	0.227	66.9	9.8	72.5	7.8	0.95
		P05P	0.209	63.2	6.8	51.2	10.4	0.97
		P06P	0.183	26.6	7.2	41.4	14.6	0.87
	Left Parietal	P03P	0.165	36.8	9.8	60.0	10.0	0.93
		P05P	0.194	60.7	6.8	49.9	10.8	0.98
		P06P	0.189	42.5	7.2	44.6	12.5	0.96

Table A6: Results from CRABI head drop tests onto different impact locations and at 2 different heights.

Approximate Drop height	Location	Measured drop height (cm)	Head mass (gm)	Peak acceleration (g)	Duration (msec)	HIC
15cm	Vertex	14.0	2095.3	33.8	19.4	38
	Occiput	15.2	2095.3	39.5	17.6	48
	Forehead	16.5	2095.3	37.3	19.4	41
	Right Parietal	14.9	2095.3	66.8	11.0	76
	Left Parietal	15.2	2095.3	116.0	10.7	142
30cm	Vertex	29.2	2095.3	51.8	18.0	99
	Occiput	29.2	2095.3	61.6	15.6	126
	Forehead	29.2	2095.3	84.8	14.0	157
	Right Parietal	30.5	2095.3	144.9	9.9	301
	Left Parietal	30.5	2095.3	214.5	9.6	488

Table A7: Regression results from CRABI head compression tests in different directions and velocities.

Compression direction	Measured velocity (mm/s)	Linear fit			Exponential fit		
		Stiffness (N/mm)	Low load Displacement (mm)	r ²	A (N)	B (mm ⁻¹)	r ²
Anterior-posterior	0.05	18.5	0.3	1.00	274.5	0.06	1.00
	1.0	22.4	0.4	1.00	253.0	0.07	1.00
	10	26.2	0.4	1.00	279.2	0.07	1.00
	42	28.7	0.4	1.00	211.5	0.10	1.00
Right-Left	0.05	62.4	0.4	0.99	48.9	0.56	1.00
	1.1	82.9	0.4	0.99	108.3	0.41	1.00
	10	95.1	0.3	0.99	117.2	0.43	1.00
	31	95.7	0.3	0.99	124.4	0.44	1.00

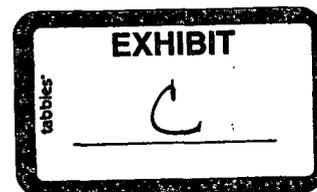
Head Impact Reconstruction - HIC Validation and Pedestrian Injury Risk

Thomas F. MacLaughlin and John F. Wiechel
S.E.A. Inc.

Dennis A. Guenther
Ohio State Univ.

MacLaughlin, T.F. et al

Reprinted from: Accident Reconstruction:
Technology and Animation III
(SP-946)



The appearance of the ISSN code at the bottom of this page indicates SAE's consent that copies of the paper may be made for personal or internal use of specific clients. This consent is given on the condition, however, that the copier pay a \$5.00 per article copy fee through the Copyright Clearance Center, Inc. Operations Center, 27 Congress St., Salem, MA 01970 for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to other kinds of copying such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, or for resale.

SAE routinely stocks printed papers for a period of three years following date of publication. Direct your orders to SAE Customer Sales and Satisfaction Department.

Quantity reprint rates can be obtained from the Customer Sales and Satisfaction Department.

To request permission to reprint a technical paper or permission to use copyrighted SAE publications in other works, contact the SAE Publications Group.



GLOBAL MOBILITY DATABASE

All SAE papers, standards, and selected books are abstracted and indexed in the SAE Global Mobility Database.

No part of this publication may be reproduced in any form, in an electronic retrieval system or otherwise, without the prior written permission of the publisher.

ISSN 0148-7191

Copyright 1993 Society of Automotive Engineers, Inc.

Positions and opinions advanced in this paper are those of the author(s) and not necessarily those of SAE. The author is solely responsible for the content of the paper. A process is available by which discussions will be printed with the paper if it is published in SAE transactions. For permission to publish this paper in full or in part, contact the SAE Publications Group.

Persons wishing to submit papers to be considered for presentation or publication through SAE should send the manuscript or a 300 word abstract of a proposed manuscript to: Secretary, Engineering Activity Board, SAE.

Printed in USA

Head Impact Reconstruction - HIC Validation and Pedestrian Injury Risk

Thomas F. MacLaughlin and John F. Wiechel
S.E.A.Inc.

Dennis A. Guenther
Ohio State Univ.

Copyright © 1993 Society of Automotive Engineers, Inc.

ABSTRACT

Experimental reconstructions of pedestrian accidents involving head injury sustained primarily from hood impact were conducted to determine the relationship between HIC and injury severity. The purpose was to establish the capability of predicting pedestrian head injury severity in simple laboratory tests.

The reconstruction test results were analyzed by a median ranking technique to provide a family of curves showing probability of injury of AIS 3, 4, and 5 severities as a function of HIC. This analysis method was used by Prasad and Mertz [1]¹ to develop a head injury risk curve from cadaver head impact test data. Results of the two analyses were compared to determine the degree of agreement between the HIC/injury-risk relationship derived from controlled experiments with cadavers and that derived from uncontrolled accidents involving live people.

The reconstruction test results also were used to derive a relationship between head injury risk (HIC) and vehicle impact speed. Specific accident cases are cited to illustrate the use of this relationship by the accident reconstructionist in estimating probable vehicle speed from injury outcome.

INTRODUCTION

The Head Injury Criterion (HIC) has been used successfully for over twenty years to assess head injury risk and develop countermeasures to protect against head injury in motor vehicle accidents. Prasad and Mertz [1] pointed out that the Wayne State Tolerance Curve, from which the HIC was derived, was developed from animal, cadaver, and human volunteer data. The long duration part of the curve is from non-injurious human volunteer tests. The short duration part (1 to 6 ms), of greater interest in the crash environment, is based on only six data points derived from human cadaver skull fracture experiments.

HIC/INJURY-RISK RELATIONSHIPS FROM CADAVER TESTS

To investigate the association of HIC with human

skull and brain injury, Prasad and Mertz selected four studies [2,3,4,5,6,7] utilizing human cadaver head impacts. The pooled skull fracture data base consisted of 54 impacts (25 skull fractures and 29 non-fractures), with HIC values ranging from 175 to 3400, and HIC durations, from 0.9 to 10.1 ms. The brain damage data base contained 43 drop and pendulum tests. HIC values were from 31 to 2351, with one at 3765, and HIC durations were 0.7 to 13.7 ms.

Prasad and Mertz used the median ranking technique reported by Mertz and Weber [8] to construct cumulative distribution curves of threshold HIC values for both skull fracture and brain damage from the cadaver data bases. The method requires the assumption that the threshold values are normally distributed. They note that the two injury risk curves are virtually identical. The brain damage curve, Figure 1, shows that the risk of life-threatening brain injury (AIS 4 or greater) is 56% for a HIC value of 1500, and 16% for a HIC of 1000.

Prasad and Mertz acknowledge that many serious doubts are associated with inferring human tolerances from cadaver test data, but that these data are the best available for this purpose. Recent experimental reconstructions of pedestrian accidents involving head injury provide a unique opportunity to attempt to confirm, with accident data, the results of Prasad and Mertz's analysis of cadaver test data.

PEDESTRIAN HEAD IMPACT RECONSTRUCTIONS

Over the last several years, the National Highway Traffic Safety Administration has conducted research on pedestrian head injury sustained from impact with motor vehicle surfaces (primarily vehicle hoods). A significant part of that research consisted of conducting experimental reconstructions of pedestrian accidents involving such impacts [9]. The purpose was to determine the relationship between HIC measured in the laboratory and injury severity seen in the real world, which would establish the capability of predicting pedestrian head injury in the laboratory.

The impact test device [10,11,12] consisted of a pneumatically-driven impacting ram and headform, which was confined to uniaxial motion. The headform was an adjustable-mass spherical aluminum fixture covered with dummy skin from a Hybrid III skull cap. It was subjected to

¹ Numbers in brackets indicate references.

the same head drop calibration requirement as the Hybrid III dummy head.

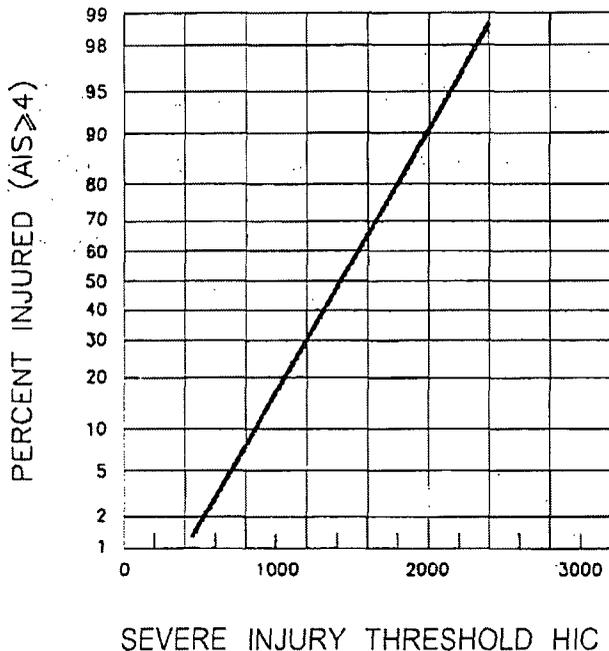


FIGURE 1
PREDICTED CUMULATIVE DISTRIBUTION CURVE
OF THRESHOLD HIC VALUES -- CADAVER BRAIN DAMAGE
(FROM REFERENCE 1)

The accident cases came from two NHTSA sources. One was an accident investigation study called PICS (Pedestrian Injury Causation Study) which took place in the late 1970s and focused on kinematics and injury from vehicle contacts [13]. A later study, PAIDS (Pedestrian Accident Investigation Data Supplement), was similar, but placed higher emphasis on collecting head injury data and vehicle dent depths and profiles [14].

Thirty-five cases (18 involving adults, 17 involving children) with well-documented head impacts were selected from these files. All impacts were against vehicle hoods except three, which were into windshields. Using vehicle impact speed estimates and geometric information from the files, computer simulations were run to provide estimates of head-to-hood (or head-to-windshield) impact velocities. Head impact tests were then conducted against the same locations on identical make/model vehicles as were reported in the accidents. In general, a head impact reconstruction involved several tests in which headform velocity and mass were adjusted, within reasonable bounds, until the extent of vehicle damage was duplicated.

In seven of the 35 cases, vehicle damage could not be reproduced within acceptable ranges of headform mass and velocity. These cases were discarded. Possible reasons for the lack of success are discussed in Reference 9; they ranged from suspected inaccuracies in the accident file to questions of whether the dent was caused by head impact only.

The remaining 28 cases (14 adults, 14 children) are shown in Table 1. This table lists the maximum AIS from head injury sustained by each accident victim and the HIC value determined from the reconstruction test which best

duplicated the vehicle damage. For the adults, maximum head AIS levels ranged from 1 to 6 and HIC values were from 187 to 3281. HIC durations were between 2.6 and 12.4 ms, except for the case where HIC was 187, for which the HIC duration was 21.6 ms (a windshield impact). Thus, even though, the accident victims sustained head impacts on generally less rigid surfaces than were used in most of the cadaver tests, HIC durations were very similar. Maximum head AIS levels for the child cases were either 1 or 2, and HIC values ranged from 202 to 1407.

The data from Table 1 are plotted in Figure 2. (In a manner similar to that for threat-to-life plots of Reference 8, the ordinate is shown as a broken line to illustrate that linear relationships do not exist between AIS values.) This figure shows a very abrupt transition in injury severity in the HIC range of approximately 1100 to 1400. Note that the child reconstructions do not help in defining this transition, since maximum head injury did not exceed AIS 2 for any of the children.

HIC/INJURY-RISK RELATIONSHIPS FROM PEDESTRIAN ACCIDENTS

The median ranking technique, used by Prasad and Mertz to produce Figure 1, was used to derive a family of HIC/injury-risk relationships from the pedestrian head impact reconstructions. Figure 3 shows these relationships for injury severities of AIS 3 or greater, AIS 4 or greater, and AIS 5 or greater. The data indicate a 50 to 60% probability of sustaining a head injury severity of at least AIS 3 when HIC is 1000, providing support for the commonly accepted value of 1000 as representing the onset of serious head injury. The data also suggest a 50 to 60% probability of sustaining an injury of at least AIS 5 severity for HIC 1500.

In Figure 4, the curve for AIS 4 or greater from Figure 3 is superimposed on the Prasad/Mertz cadaver brain damage curve of Figure 1. The degree to which the two relationships agree in the vicinity of HIC 1000 is very satisfying. The curves cross at a HIC value of 1200, both indicating a 30% probability of sustaining a head injury of at least AIS 4 severity. The relationships differ in that the pedestrian head injury curve is steeper. This suggests reduced confidence in using the curves for high and low probability estimates. The steepness of the pedestrian curves also emphasizes that their primary usefulness is in establishing threshold values.

The results of the combined analyses of the pedestrian accident reconstruction data and the cadaver test data tend to support 1) HIC 1000 as a valid injury criterion for live people, and 2) the use of cadavers for head injury research.

PEDESTRIAN HEAD INJURY RISK AND COLLISION SEVERITY

The reconstruction test cases were further analyzed to explore the relationship between pedestrian head injury risk and vehicle impact speed. Table 1 contains the vehicle impact speeds which were estimated by the accident investigators and reported in the case files. In most cases, ranges of speeds were given. In Figure 5, the maximum head AIS for each of the 28 cases which were reconstructed is

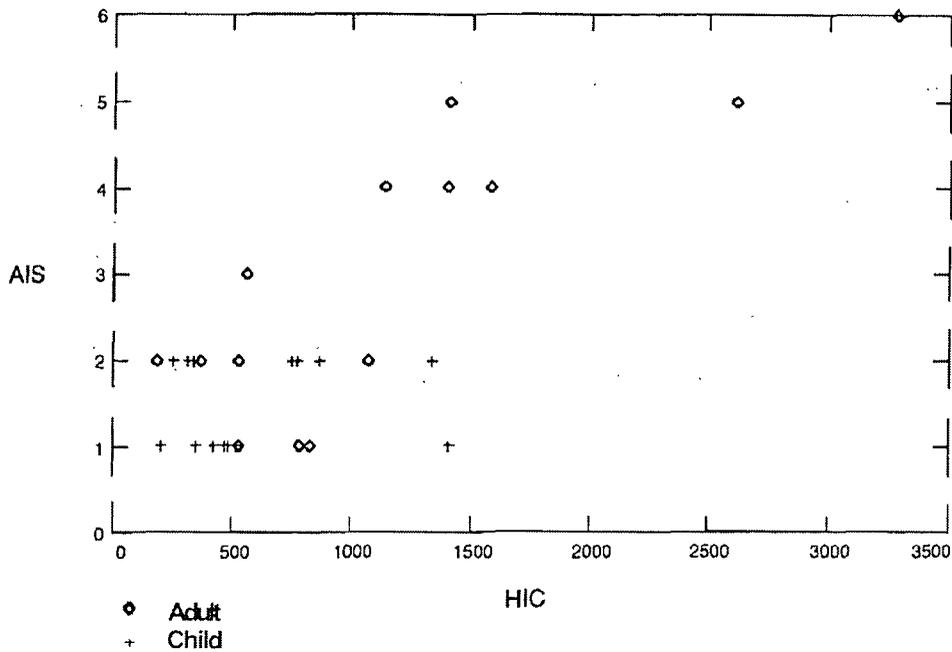


FIGURE 2

MAXIMUM HEAD AIS VS. HIC --
PEDESTRIAN ACCIDENT RECONSTRUCTIONS

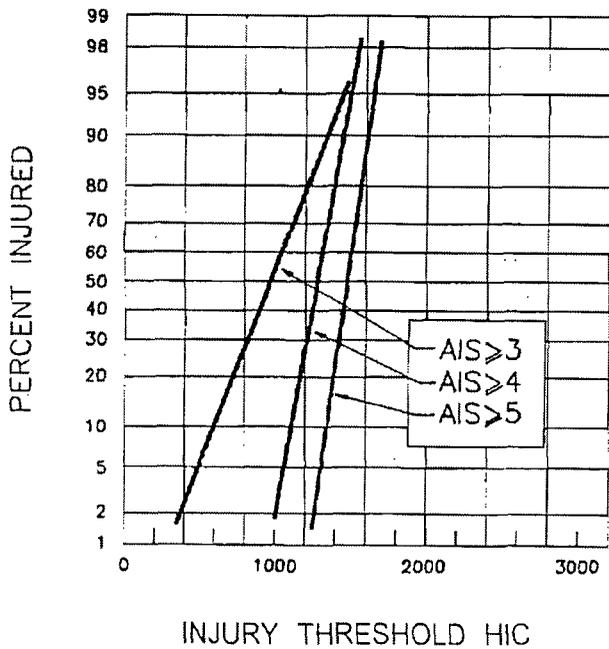


FIGURE 3
PREDICTED CUMULATIVE DISTRIBUTION
CURVES OF THRESHOLD HIC VALUES --
PEDESTRIAN ACCIDENT RECONSTRUCTIONS

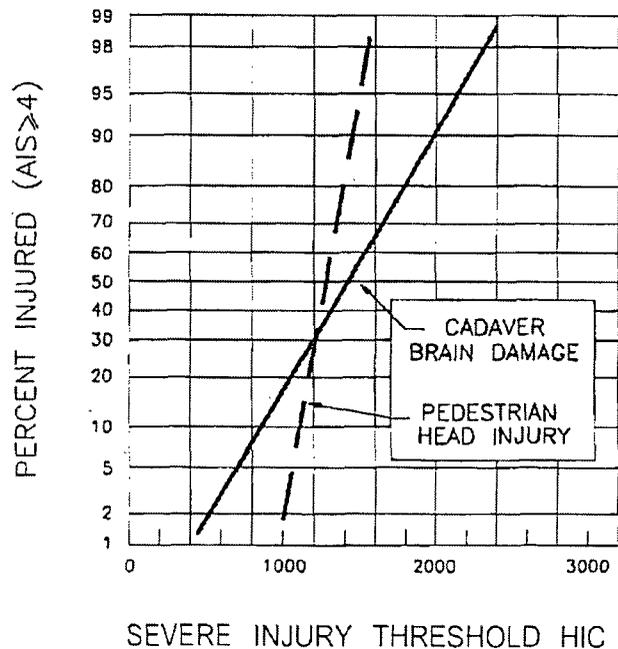


FIGURE 4
COMPARISON OF PREDICTED CUMULATIVE DISTRIBUTION
CURVES OF THRESHOLD HIC VALUES FROM
CADAVER BRAIN DAMAGE
AND PEDESTRIAN ACCIDENT RECONSTRUCTIONS

plotted against vehicle impact speed. Where speed ranges were reported, the median values were plotted. Although pedestrian head injury severity is influenced by many parameters, including localized hood stiffness and under-hood clearance, a distinct trend with impact speed is seen. A sharp transition also is evident at 10 to 12 m/s between minor/moderate and serious-to-fatal head injury.

The median ranking technique again was applied to estimate the probability of sustaining head injury of specific severity at given vehicle impact speeds. Two curves are shown in Figure 6, one for injury severity of AIS 3 or greater and one for AIS 5 or greater.² As in Figure 3, the curves are very steep, and they are useful primarily in establishing threshold speeds. They suggest, for example, the following: 1) The probability of receiving a serious head injury from contacting the vehicle hood or windshield when the vehicle impact speed is below 10 m/s is low. 2) At a speed of 12.5

m/s, however, the probability of a serious head injury is high. 3) And if the vehicle's speed is 15 m/s, there is a high probability of sustaining a critical or fatal head injury.

In most pedestrian accidents, the reconstructionist estimates vehicle speed from skid marks, analysis of the pedestrian's trajectory, and/or witness statements. The injury-speed relationships in Figure 6 often can be useful in providing an alternate means of estimating probable vehicle impact speed, using injury outcome. Three illustrative cases from S.E.A.'s accident files are presented below. It is emphasized that Figure 6 is not intended to offer indisputable evidence regarding vehicle-to-pedestrian impact speed for any given case. Indeed, one of the example cases illustrates that injury severity can be quite different from that predicted. Nonetheless, it often can be used to supplement other pieces of evidence to strengthen the likelihood of achieving a valid estimate.

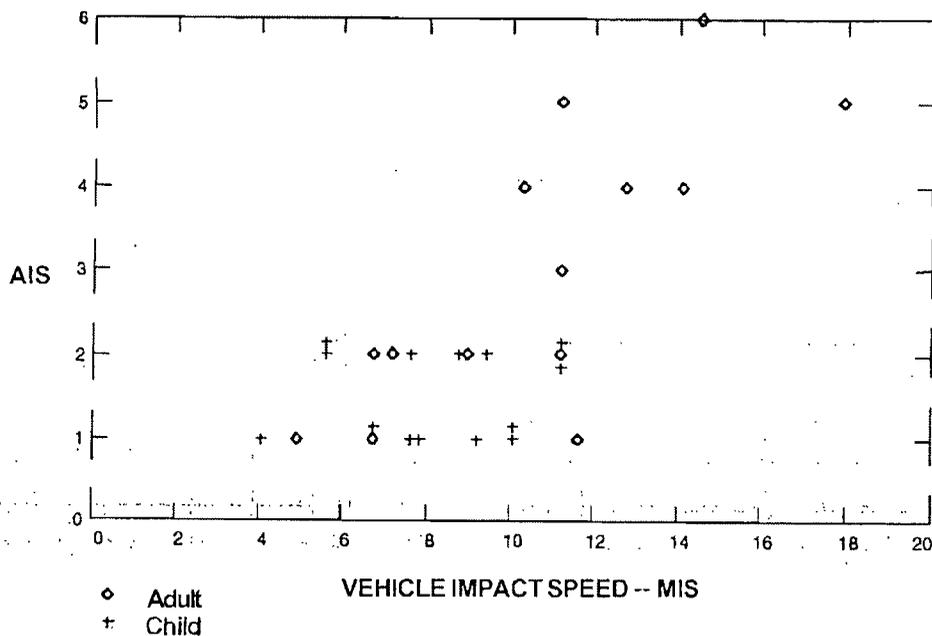


FIGURE 5
 MAXIMUM HEAD AIS VS. VEHICLE IMPACT SPEED --
 PEDESTRIAN ACCIDENT RECONSTRUCTIONS

² The single data point at AIS 3 is located such that the region of overlap for determining the AIS 3 curve is the same as that for AIS 4. This yields the same probability estimate for AIS 3 and AIS 4.

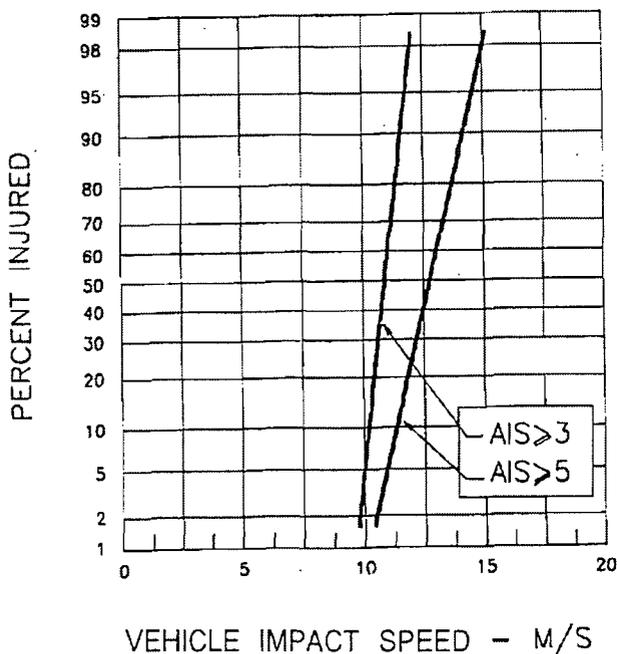


FIGURE 6
 PREDICTED CUMULATIVE DISTRIBUTION CURVES
 OF THRESHOLD VEHICLE IMPACT SPEEDS --
 PEDESTRIAN ACCIDENT RECONSTRUCTIONS

Case 1:

A six-year old boy ran into the street and was struck by a compact automobile. He sustained multiple leg fractures, a fractured skull and concussion, and abdominal injuries. Damage to the vehicle indicated that the pedestrian's head injury resulted from impact on the hood. The boy's final rest position was documented, but the point of impact was located only approximately. The car left skid marks on the pavement, but probably not until after impact occurred.

The speed limit at the accident site was 15.6 m/s. At issue was whether or not the car was exceeding the speed limit.

An analysis of the pedestrian's trajectory indicated the car's speed at impact was in the range of 12.5 to 18.8 m/s. The length of the skid marks suggested the vehicle was travelling about 14.8 m/s; however, if the driver released his brakes enough to stop skidding before coming to a stop, his travelling speed could have been higher.

Figure 6 indicates that the threshold vehicle impact speed corresponding to a 100% probability of sustaining a head injury of at least AIS 3 severity from hood or windshield contact is about 12.5 m/s, and, for an AIS 5 or 6 injury, it is slightly over 15 m/s. This tends to support a travelling speed estimate at or below the speed limit, and suggests the upper speed range predicted by the trajectory analysis is probably too high.

It was concluded that the driver probably was not exceeding the speed limit.

Case 2:

This accident occurred at night under poor visibility conditions. A sixteen-year old pedestrian was struck by a small sedan and thrown into the grass along the side of the road. The location of her final rest position was documented. She sustained severe leg fractures, but received no head injury even though her head impacted and substantially fractured the passenger-sidewindshield.

The driver reported that the girl walked into the road in the path of the car, the impact occurred, and then the car swerved off the pavement. The pedestrian said she was walking in the grass about 1.5 m from the edge of the road, and the driver went off the road and then struck her. Tire tracks in the mud at the side of the road documented where the car left the pavement, but there was no evidence locating the point of impact.

At issue in this case was whether the vehicle-to-pedestrian impact occurred in the road, as claimed by the driver, or off the road, as claimed by the pedestrian.

A trajectory analysis indicated that if the impact occurred in the road, the car had to have been going at least 13.4 m/s to have thrown the pedestrian to her final rest position. Figure 6 indicates that, at 13.4 m/s, the probability of sustaining a head injury of at least AIS 3 severity is 100%, and the probability of a critical-to-fatal head injury (AIS 5 or 6) is about 80%. The fact that she had no head injury suggests that the car's speed (again referring to Figure 6) was not over 10 m/s. Using this speed, the trajectory analysis indicated her location at impact to be at least 1.5 m from the pavement, supporting the girl's contention that she was not in the road at impact.

It was concluded that the driver drove off the pavement before striking the pedestrian.

Case 3:

An intoxicated nineteen-year old man was walking diagonally, in a southeasterly direction, across a street. He crossed the oncoming (westbound) traffic lane, and as he entered the eastbound lane, he walked into the left front fender of an eastbound car. His body wrapped onto the hood, and the back of his head impacted and substantially fractured the windshield on the driver's side. As the car stopped, he slid off the front of the hood and fell to the street. The accident occurred on a very dark night in a heavy rainstorm; visibility was very poor.

The pedestrian sustained a closed head injury, with substantial brain injury. Upon admission to the hospital, the examining physician noted a contusion on the victim's scalp posteriorly, consistent with the windshield impact. No other signs of head impact (such as might have occurred from pavement contact) were noted. He was hospitalized for three and a half months, and was

discharged to a rehabilitation hospital with persistent severe impairment and a guarded prognosis for functional recovery. Based on a detailed discharge summary, it was estimated his head injury severity was AIS 5.

The question at issue was the speed of the striking car.

The speed limit at the accident site was 8.9 m/s. The driver of the striking car said her speed was 6.7 m/s. Two witnesses (another pedestrian who was the victim's companion, and the driver of a westbound car) gave their estimate of the striking vehicle's speed as 6.7 to 8.9 m/s. A speed analysis, based on an estimate of the pedestrian's walking speed into the side of the car and the location of his contacts on the car, indicated that the car's speed probably did not exceed 7.6 m/s. All of these estimates fall in a region of Figure 6 which suggests low probability of the pedestrian sustaining a head injury severity exceeding AIS 2, which obviously conflicts with the medical evidence.

It was concluded that there was sufficient evidence that the striking car was not exceeding the 8.9 m/s speed limit. The pedestrian's injury severity in this case was regarded as an outlier to the information from which Figure 6 was derived.

In the first two cases, use of the injury-speed relationships is helpful in increasing the validity of the vehicle impact speed estimates. In case 3, however, the predicted impact speed from the injury outcome is clearly in conflict with other evidence which appears convincing. The reconstructionist in this case is best advised to reject the injury-speed prediction in favor of more solid information.

SUMMARY AND CONCLUSIONS

For many years, a HIC value of 1000 has been accepted as a reasonable threshold of serious head injury resulting from head impact in motor vehicle collisions. Most of the data supporting this value have come from experiments with human cadavers.

Data from experimental reconstructions of pedestrian head impacts against vehicle hoods and windshields which occurred in real accidents were analyzed. An abrupt transition from moderate to severe injury occurred over a HIC range of 1100 to 1400. Cumulative injury distribution curves were derived, using the same techniques as were used with cadaver impact data. A 50 to 60% probability of sustaining an injury of at least AIS 3 severity was predicted for a HIC value of 1000.

Results of the pedestrian reconstructions and the cadaver tests show reasonable agreement; both indicate a 30% probability of sustaining a head injury of at least AIS 4 at a HIC value of 1200. The results tend to support HIC 1000 as a valid criterion for live people, and tend to justify the use of cadavers for head injury research.

Relationships between pedestrian head injury risk and vehicle impact speed were derived. The probability of sustaining a serious head injury from impact against the hood or windshield is predicted to be low at vehicle speeds below

10 m/s. At 12.5 m/s, however, the probability appears to be high; and at 15 m/s, the predicted probability of sustaining a critical or fatal head injury is high.

The pedestrian head injury I vehicle impact speed relationships often can be useful to the accident reconstructionist as an alternate means of estimating probable vehicle speed, using injury outcome. However, they must be regarded as approximate, and should be used only as a supplement to other evidence to strengthen the likelihood of achieving a valid estimate.

REFERENCES

1. Prasad, P. and Mertz, H.J., "The Position of the United States Delegation to the ISO Working Group 6 on the Use of HIC in the Automotive Environment," SAE Technical Paper 851246, Government/Industry Meeting and Exposition, Washington, D.C., May 1985.
2. Got, C., Patel, A., Fayon, A., Tarriere, C., and Walfisch, G., "Results of Experimental Head Impacts on Cadavers: The Various Data Obtained and Their Relations to Some Measured Physical Parameters," Proc. 22nd Stapp Car Crash Conference, 1978.
3. Tarriere, C., Walfisch, G., Fayon, A., Got, C., Guillon, F., Patel, A., and Hureau, J., "Acceleration, Jerk and Neck Flexion Angle: Their Respective Influences on the Occurrence of Brain Injury," ISO/TC22/SC12/GT-6 (USA-13), Doc. No. 118, 1982.
4. Nahum, A.M., and Smith, R.W., "An Experimental Model for Closed Head Impact Injury," Proc. 20th Stapp Car Crash Conference, 1976.
5. Nahum, A.M., Smith, R.W., and Ward, C.C., "Intracranial Pressure Dynamics During Head Impact," Proc. 21st Stapp Car Crash Conference, 1977.
6. Hodgson, V.R., and Thomas, L.M., "Breaking Strength of the Human Skull versus Impact Surface Curvature," Wayne State University, Detroit, Final Report DOT HS 146 2 230, 1977.
7. Hodgson, V.R., Thomas, L.M., and Brian, J., "Concussion Levels Determined by HPR Windshield Impacts," Proc. 17th Stapp Car Crash Conference, 1973.
8. Mertz, H.J. and Weber, D.A., "Interpretations of the Impact Responses of a 3-Year-Old Child Dummy Relative to Child Injury Potential," 9th International Technical Conference of Experimental Safety Vehicles, U.S. D.O.T., NHTSA, Washington, D.C., 1982.
9. Hoyt, T. A., MacLaughlin, T. F., and Kessler, J. W., "Experimental Pedestrian Accident Reconstructions - Head Impacts," Final Report DOT HS 807 288, U.S. D.O.T., NHTSA, NTIS, Springfield, Virginia, June 1988.
10. Pritz, H. B., "Experimental Investigation of Pedestrian Head Impacts on Hoods and Fenders of Production Vehicles," SAE Paper No. 830055, February 1983.

11. Brooks, D.L., Collins, J.A., and Guenther, D.A., "Experimental Reconstructions of Real World Pedestrian Head Impacts," Final Report for DOT/NHTSA Basic Agreement No. DTNH22-83-A-072779, VRTC Task Order No. OSU-84-4059, College of Engineering, The Ohio State University, Columbus, Ohio, March 1985.
12. Kessler, J. W., "Development of Countermeasures to Reduce Pedestrian Head Injury," 11th International Technical Conference of Experimental Safety Vehicles, Washington, D.C., May 1987.
13. Pedestrian Injury Causation Study Data File, National Highway Traffic Safety Administration, Research and Development, National Center for Statistics and Analysis, Washington, D.C.
14. Pedestrian Accident Investigation Data Supplement Files, National Highway Traffic Safety Administration, Research and Development, National Center for Statistics and Analysis, Washington, D.C.

TABLE 1

PEDESTRIAN ACCIDENT CASES AND RECONSTRUCTION RESULTS

ACCIDENT FILE / CASE NO.	ADULT OR CHILD	VEHICLE IMPACT SPEED (MIS)	MAX HEAD AIS	RECON HIC
PAIDS / 82-08-205	ADULT	12.5-15.6	4	1571
PAIDS 181-09-202	ADULT	6.3-7.2	2	365
PAIDS / 83-02-204	ADULT	3.6-6.3	1	527
PAIDS / 81-08-202	CHILD	4.5-6.7	2	313
PAIDS 181-08-207	ADULT	8.9-13.4	2	531
PICS / 99-07-201	CHILD	6.7-8.5	2	336
PAIDS 183-02-201	ADULT	8.9-11.6	4	1394
PAIDS / 82-07-202	ADULT	8.9-13.4	3	559
PAIDS / 82-10-201	CHILD	6.7-10.7	2	751
PAIDS / 83-01-202	CHILD	8.9-11.2	1	202
PAIDS / 82-05-201	CHILD	8.0-10.7	2	774
PAIDS / 81-08-211	CHILD	8.5-9.8	1	487
PICS / 68-05-201	CHILD	4.5-6.7	2	251
PICS / 88-07-209	CHILD	4.0	1	423
PICS 179-06-201	CHILD	6.7	1	518
PICS / 99-07-201	CHILD	6.7-8.5	1	343
PICS / 67-11-206	ADULT	17.9	5	2613
PICS / 78-08-209	ADULT	13.4-15.6	6	3281
PICS / 78-03-211	ADULT	12.1-13.4	4	1137
PICS 179-08-219	ADULT	11.2	5	1403
PICS / 19-05-220	ADULT	6.7	1	785
PICS / 87-08-215	ADULT	7.2	2	1071
PAIDS 182-03-201	ADULT	6.7-11.2	2	187
PAIDS / 81-08-206	ADULT	10.7-12.5	1	832
PICS / 88-01-203	CHILD	8.9-11.2	1	1407
PICS / 10-01-216	CHILD	6.7-8.9	1	470
PICS 168-05-212	CHILD	11.2	2	1332
PAIDS / 81-08-204	CHILD	8.9-13.4	2	864

21

Pediatric Biomechanics

Narayan Yoganandan, Srirangam Kumaresan, Frank A. Pintar,
and Thomas A. Gennarelli

The literature is replete with studies dealing with the responses of the adult human load-bearing systems under physiologic and traumatic loading. These include prospective and retrospective clinical, epidemiologic, experimental (biologic and physical), and mathematical studies. Early work on the cranium and its contents has formed a basis for the widely used head injury criteria.^{45,46,131} Biomechanical investigations have been conducted to delineate facial tolerance secondary to impact.^{55,121,136,148,149,151,157} Similarly, human vertebral column studies including the spinal cord have focused on adult tolerances and injury mechanisms under various modes of loading.^{6,106,108,109,140,143,146,150,155,156} Investigations describing the tolerance of the adult human thorax/chest under frontal and side-impact loading modalities also exist.^{7,20-22,36,37,91,93,138,139,141,145,153,154,158} Likewise, the tolerances of the upper and lower extremities have been explored for the adult structure.^{10,11,64,92,105,144} The cited reference list is not inclusive. Despite these efforts, biomechanical data remain sparse for the growing pediatric group.

Human pediatric structures (e.g., vertebral column, brain, and skull) mature with age. The anatomic literature provides information on structural development. However, the biomechanical literature, as indicated, is still in its infancy particularly with regard to tolerance issues governing this developing population. Recent awareness of advanced safety and protection in a crash environment, promulgation of

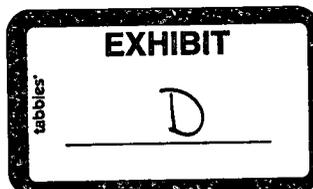
restraint legislation, and changes in vehicular design (e.g., restraint systems such as airbags) have underscored a need to obtain pediatric tissue tolerance under impact. This chapter reviews the biomechanical properties of children. Aspects of developmental anatomy and biomechanical knowledge are analyzed to assist in pediatric human injury prediction. Initially, emphasis is placed on neck structures, because airbag-induced neck injuries have accentuated the demand to obtain pediatric tolerance. These data may also assist in improving the biofidelity of child dummies. A similar discussion is given for the head. The presentation follows with a brief analysis of the thorax/chest and extremity structures. Detailed anatomics and constitutive relations are not given for all structures because of space constraints. However, an attempt is made to provide age-specific tolerance characteristics for these regions. In addition to the analyses of experimental and analytical studies, the newly proposed tolerance criteria are discussed for the neck, head, and thorax along with the basis for application to different child dummies. Finally, recommendations are made for future biomechanical studies.

Neck

Developmental Anatomy

The major structural components of the neck include the cervical spinal column and soft tissues. The cervical spine consists of seven

Yoganandan, N. et al. ch. 21



vertebrae (C1–C7). The intervertebral disk starts inferiorly from C2. Spinal ligaments interconnect from the base of the skull (occiput) to C7 and proceed distally. The spinal cord that originates from the foramen magnum is housed in the osseous-ligament anatomy of the spinal column. Muscles originate and insert at various locations along the neck and proceed along the cranial and caudal directions. A brief description of the developmental anatomy of the various components is given below.^{9,13,14,18,24,28,32,43,47,50,54,59,60,65,76,97–100,103,112,124,130,132–134}

Vertebrae

Atlas

The first vertebra (C1) is also called the atlas. It is formed from three primary ossification centers; one occurs in the anterior region and two occur bilaterally in the posterior neural arches. The former center develops several months following birth, while the other two centers are present at birth. The junction between the anterior and bilateral posterior centers is called the neurocentral synchondrosis. The two neural arch centers fuse or join dorsally by posterior synchondrosis. Fusion of the posterior synchondrosis occurs first. This is followed by fusion of the neurocentral synchondrosis. The spinal canal fully forms and attains the mature adult size following complete fusion of the primary ossification process.^{53,129,161}

Axis

The second vertebra (C2) is also called the axis. It is formed from five primary ossification

centers.^{9,97,100,113,130,133} One occurs in the centrum (vertebral body location), two occur bilaterally in the posterior neural arches, and two occur bilaterally in the odontoid process. The two centers in the odontoid are joined at birth. The odontoid process is connected to the body (of C2) by dentocentral synchondrosis. Paired neurocentral synchondroses form the connection between the two posterior arches and centrum. Development of the ossification process is as follows: Neural arches fuse posteriorly. This is followed by fusion of the centrum-neural arch and odontoid process-centrum. As in the case of the atlas, the spinal canal reaches its mature size following closure of the posterior and neurocentral synchondroses.^{53,129,161} In contrast, the dentocentral synchondrosis continues to mature, and, in certain cases, ossification never completes.

Typical Cervical Vertebrae

Each of the five vertebrae (C3–C7) is formed from three primary ossification centers, one in the anterior centrum and two in the posterior neural arches.^{9,23,25,40,97,100,130,133} The neurocentral synchondrosis is the joining element between the neural arches and centrum. Neural arches are connected to each other by posterior synchondrosis. The progression of the ossification process is as follows. Neural arches join posteriorly. This is followed by the joining of the anterior and posterior ossification centers. Similar to C1 and C2, the spinal canal size attains the adult dimension following completion of these primary ossifications.^{53,129,160} Figure 21.1 illustrates the characteristics of ossification in the atlas, axis, and a typical cervical vertebra.

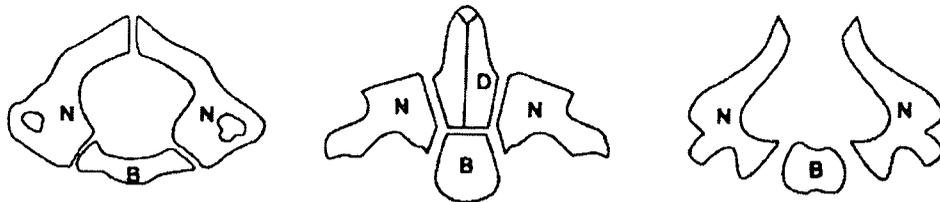


FIGURE 21.1. Primary ossification center in the atlas, axis, and a typical cervical vertebra (C3–C7). Three ossification centers in the atlas (left, superior view), five in the axis (middle, anterior view), and three in the typical cervical vertebra (right, superior view). N, neural arch; B, vertebral body; D, odontoid process.

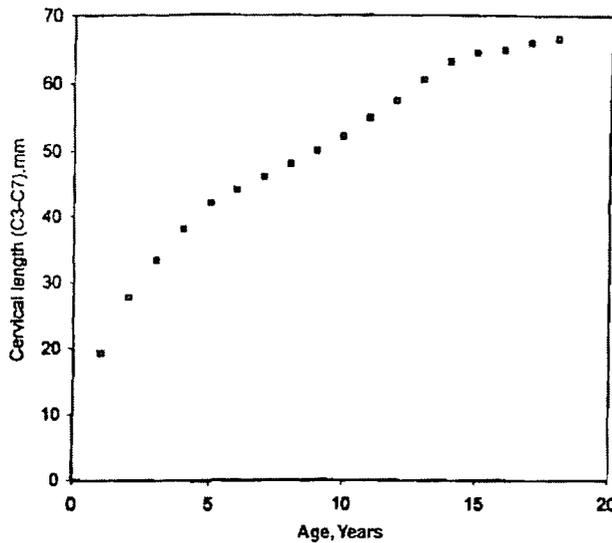


FIGURE 21.2. Variation of the summated height of the typical cervical vertebral bodies denoted as cervical length (from C3 to C7) as a function of age. (Data adapted from Kasai et al.⁶⁰)

The summated height of all the typical cervical vertebrae is included in Fig. 21.2.⁶⁰

Vertebral Growth

The presence of the three cartilaginous synchondrosis junctions allows for the latitudinal and longitudinal growth of the vertebra.^{13,18,44,47,99,112} With advancing age, vertebral bodies attain a more rectangular shape and increase in height secondary to the presence of superior and inferior growth plates. This growth continues until puberty. The growth plates contribute to the longitudinal development of the vertebral body. The latitudinal growth occurs by the expansion of the neural arch ossification center that develops into three independent growth zones: the pedicle, lamina, and transverse processes.

In general, the age group between newborn and 1 year can be skeletally represented by the presence of three primary ossification centers. The age group between 1 and 3 years can be represented by the fusion of the posterior synchondrosis. The age group between 3 and 6 years signifies complete fusion of all primary ossification centers. The process of secondary ossification, which manifests in the form of end plates, begins following attenuation of the primary ossification. The end plates contribute

further to the growth of the vertebral body. In addition, uncinat processes and uncovertebral joints begin to develop around puberty as a consequence of secondary ossification.^{17,26,49,51,72} These secondarily developed components are responsible for the saddle shape of the cervical vertebrae. Coupled motions (primary and secondary) of the cervical spine occur as a result of these changes.^{25,75,117,146}

Ligaments

All cervical vertebrae including the base of the skull are connected by soft tissue structures.¹¹⁷ Ligaments are unique to the upper cervical region, i.e., the occiput-atlas-axis complex^{25,94} (Fig. 21.3). Beginning anteriorly, the anterior longitudinal ligament is renamed as the anterior atlanto-occipital membrane from C1 to the occiput. The apical ligament attaches from the tip of the odontoid process of C2 to the occiput. The alar ligaments attach from the superior-lateral aspect of the odontoid process and run obliquely to the occiput. The cruciate ligament has a strong transverse portion that runs laterally around the odontoid process and attaches at both ends to the medial aspects of the arch of the atlas. The vertical cruciate ligament attaches from the occiput, just posterior to the apical ligament, intertwines with its transverse

A
ATLAN
M

C

K
CAP

F
c
t
r
i

F
i
t
c
l
t
:

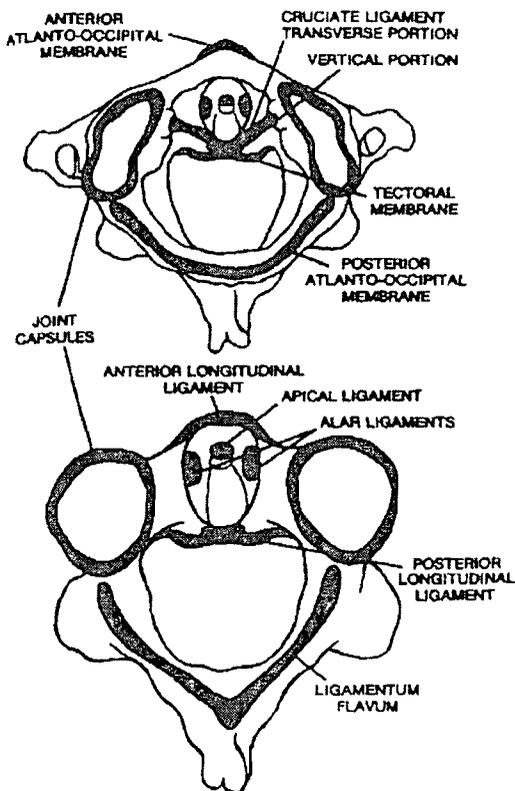


FIGURE 21.3. Schematic diagrams of the atlantoaxial complex. Top: Superior view of C1 and C2 showing the location of the major ligaments. Bottom: Superior view of C2 emphasizing the ligaments.

portion, and attaches again to the posterior-inferior aspect of the vertebral of the axis. The tectorial membrane attaches to the anterior one-third of the basilar occiput just posterior to the vertical cruciate ligament. This ligament tapers inferiorly to become continuous with the posterior longitudinal ligament. The posterior atlanto-occipital membrane connects the superior aspect of the posterior arch of the atlas to the occiput.¹⁴² Proceeding inferiorly from C2, the anterior and posterior longitudinal ligament, ligamentum flavum, bilateral capsular, and interspinous ligaments connect one vertebra to its immediate adjacent vertebra (Fig. 21.4).

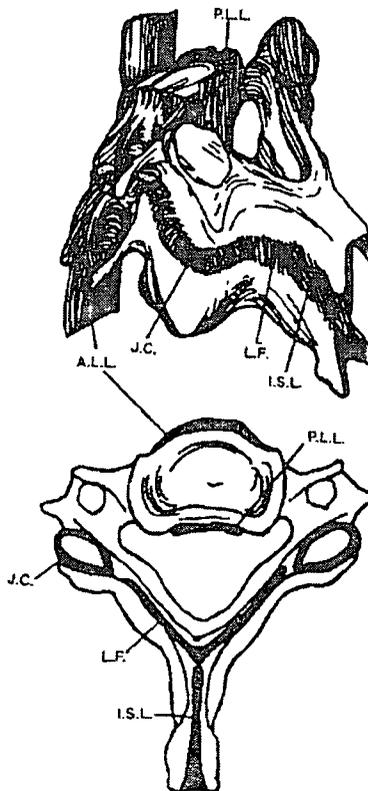


FIGURE 21.4. Schematic diagram of a typical cervical spinal unit emphasizing the ligaments. Top: Posterolateral view of a vertebra-disk-vertebra. Bottom: Superior view of a typical cervical vertebra showing the relative position of the spinal ligaments. A.L.L., anterior longitudinal ligament; P.L.L., posterior longitudinal ligament; L.F., ligamentum flavum; J.C., joint capsules; I.S.L., interspinous ligament.

Facet Joints

Bilateral facet joints form the posterior connection between the inferior articular process of the superior vertebra and the superior articular process of the inferior vertebra (Fig. 21.5). The two processes are connected by the synovial joint consisting of the synovial fluid, synovial membrane, articular cartilage, and capsular ligaments.^{9,25,60,68,71,97,100,133} Developmentally, orientations of the facet joints change and the variations depend on the vertebral level. The joints in the upper spine are less

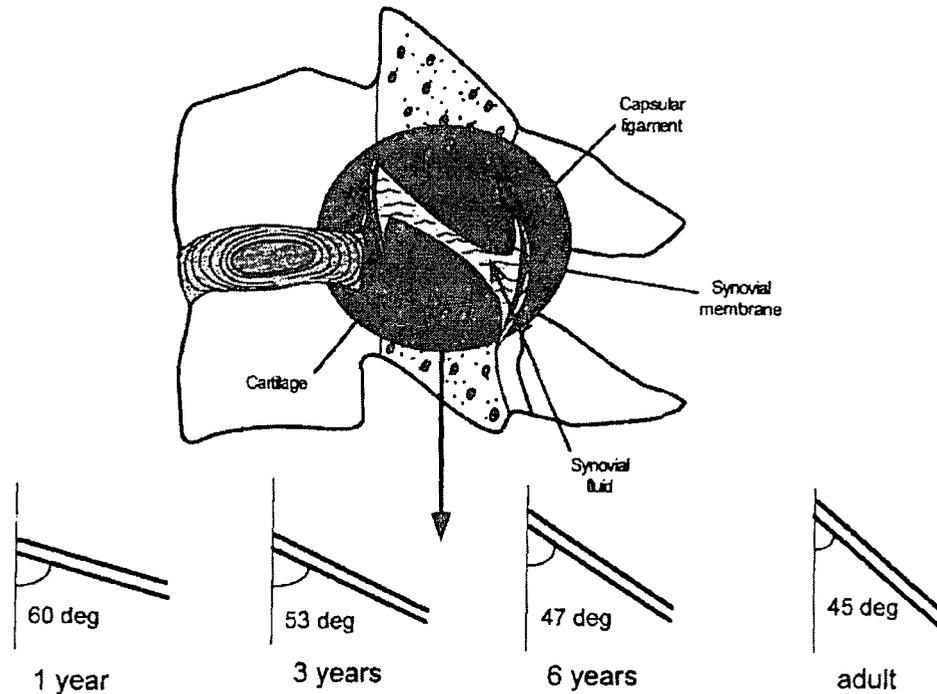


FIGURE 21.5. Exploded facet joint illustrating its components (top). Variation of the facet joint orientation as a function of age is shown (bottom).

oblique (more horizontal) compared to the lower regions. The degree of obliquity gradually increases with advancing age (Fig. 21.6). The more horizontal orientation of the facet joint at the upper level anatomy, together with the softer intervertebral components, appear as pseudosubluxation in younger ages.^{4,15,16,19,38,48} The changing facet joint orientation biomechanically contributes to a varying share of the external load (e.g., compression).

Intervertebral Disks

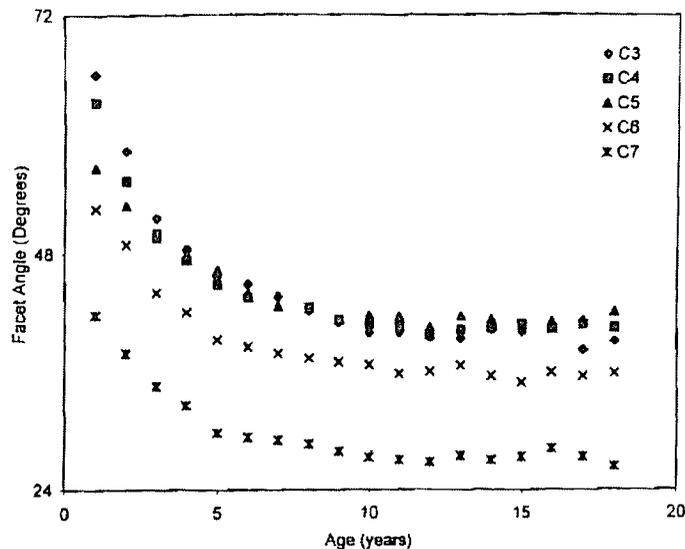
Intervertebral disks connect the vertebrae inferiorly from the axis. Nucleus pulposus, annulus fibers, and ground matrix are the principal components. These components undergo considerable developmental changes.^{28,29,50,54,97,98,100,103,124,130,132,133} Growth patterns are interrelated in the nucleus and annulus. Typically, around 1 year of age, the disk is characterized by a large nucleus with loosely embedded annular fibers rendering a less than

clear distinction between the nucleus and annulus (Fig. 21.7). At approximately 3 years of age, a clearer distinction can be appreciated, which is due to increased formation of fibers. However, the nucleus still occupies a large volume of the disk. Around 6 years of age, fibers attain higher stiffness and density, accentuating a better demarcation between the nucleus and annulus boundaries. During adolescence to adulthood, the nucleus further shrinks reaching its skeletally mature level with a concomitant development of the annulus fibrosis. The disk extends laterally beyond the centrum to form the uncinat processes during puberty coincident with the secondary vertebral ossification process. This structural variation in the disks is accompanied by the formation of uncovertebral joints.

Muscles

Neck muscles connect the ligamentous cervical spinal column with the head and torso.^{25,117}

FIGURE 21.6. Variation of the facet joint angle as a function of age. (Data adapted from Kasai et al.⁶⁰)



Although the neck muscles are numerous, they are classified based on the motions they produce. Muscles active during flexion are classified as flexors (e.g., sternocleidomastoid, longus colli). Similarly, muscles active during extension are termed extensors (e.g., splenius capitis, trapezius). During the developmental process, neck dimensions (e.g., breadth) and cross-sectional areas of the flexors and extensor muscles increase.^{59,118} However, the rate of increase is different between the two types of muscles (Fig. 21.8). The variation of neck circumference and lateral breadth as a function of age is shown in Fig. 21.9.

Age-Related Grouping and Material Properties

Based on vertebral growth and characteristic distributions in the constituents between the cartilaginous structures central to the synchondrosis and the bone itself, pediatric to adolescent structures can be broadly categorized into four groups.^{9,28,69,70,73,74,97,98,100,103,115,124,133,137} The newborn to 12-month group (I) depicts the existence of the three primary centers. The 1- to 3-year group (II) represents fusion of the posterior synchondrosis. The 3- to 6-year group

(III) denotes fusion of the bilateral anterior neurocentral synchondroses, and the 11- to 14-year (approximately puberty) group (IV) corresponds to secondary ossification and initiation of the development of the uncinate and uncovertebral anatomy (Fig. 21.10). Skeletally mature adult vertebral anatomy in the human occurs during the second decade of life. Tables 21.1 to 21.4 list the representative material properties used in the literature for the various components of the vertebrae and their interconnecting tissues for the four groups.^{68,146} These data are currently used in stress analysis-type models to understand pediatric responses and predict injury. The physiologic stress on the cervical spine changes as age progresses due to alterations in the mass of the head.

Biomechanical Studies

Quasi-Static Studies

Although biomechanical investigators have traditionally cited literature starting from the seminal work reported in 1880, with regard to the tolerance of the human neck, a study was

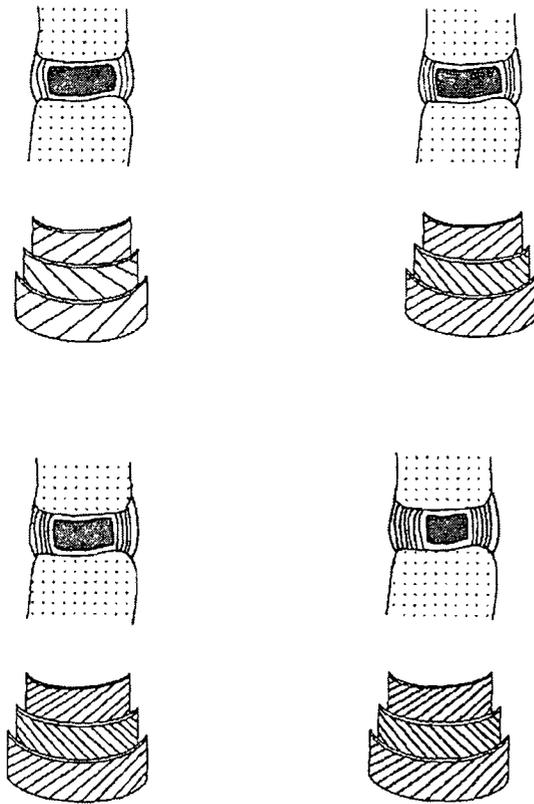


FIGURE 21.7. Schematic of the sagittal section of the intervertebral disk showing the nucleus surrounded by the annulus and the magnified view of the annulus laminae for the 1-year-old (top left), 3-year-old (top right), 6-year-old (bottom left), and adult (bottom right). Vertebral bodies above and below the disk are shown in the dotted boundaries.

conducted in 1874 using four newborn cadavers and one 2-week-old child cadaver.^{35,89} The cadavers were prepared as follows. The body was passed through an aperture cut in wood to represent the brim of a contracted pelvis, or the head was fixed below the biparietal diameter between two parallel bars. Weights were applied to a hook attached to one of the lower extremities. They were applied in increments with 30-sec duration until severed from the body. The spinal column failed rapidly with a snap followed by a marked elongation of the entire body. The severed vertebrae widely detached from adjacent segments before the

whole neck yielded, resulting in decapitation. This essentially depicts the role of cartilaginous-type structures on the failure biomechanics. The force to yield the spinal column and to decapitate the specimens increased from the newborn to the 2-week-old specimen (Fig. 21.11). For the newborn, a mean force of 470 N (± 78) for spinal column failure (middle to lower regions) and 540 N (± 101) for decapitation were reported. The 2-week-old specimen failed at 654 N and decapitation occurred at 725 N. The mean force for all five specimens was 507 N (± 107) for cervical column failure and 577 N (± 120) for decapitation.

Two cervical units, one mid-thoracic unit and one lumbar functional spinal unit from a fresh frozen 8-hour-old human cadaver spine, were loaded in axial tension at a rate of 1.25 mm/sec.⁸¹ Fixation failures resulted in the cervical and thoracic specimens. The L3-L5 specimens demonstrated initial failure of capsular ligaments and ligamentum flavum at the cephalad level followed by a tear of the intra- and supraspinous ligaments at the caudal level. Force-displacement curves indicated a peak force of 216 N at a displacement of approximately 4 mm and a distraction stiffness of 94 N/mm.

Dynamic Studies

Two studies were conducted to determine the biomechanical responses of anesthetized animals secondary to airbag loading.^{85,88,110} In the earlier study, 43 pigs (mean age: 10 weeks) and three baboons (mean age: 5.2 years) were used. The impact pulse had a 14.5g deceleration with a change in velocity (ΔV) of 56 kph or 8.4g with a $\Delta V = 33.6$ kph. The mild (less severe) pulse was more trapezoidal, which represented a car-to-car impact of longer duration with a mean deceleration of 4.3g and $\Delta V = 28.5$ kph. Seven different animal positions and 10 types of inflators were used in combination with eight types of airbags, four types of folds, and three types of covers. The animal was placed on a sled and held vertically by a tether that was released prior to airbag deployment. The buttocks were supported by the seat, floor, or foam blocks, depending on the preposition of the animal.

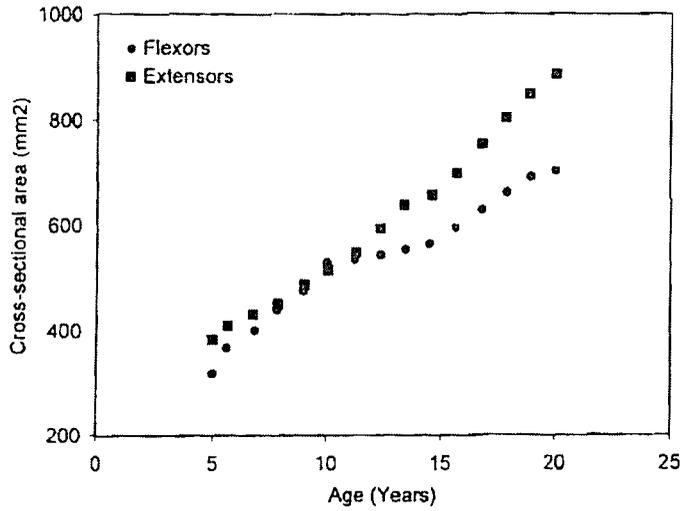


FIGURE 21.8. Variation of the cross-sectional area of the neck flexor and extensor muscles as a function of age based on magnetic resonance imaging (MRI). See text for details.

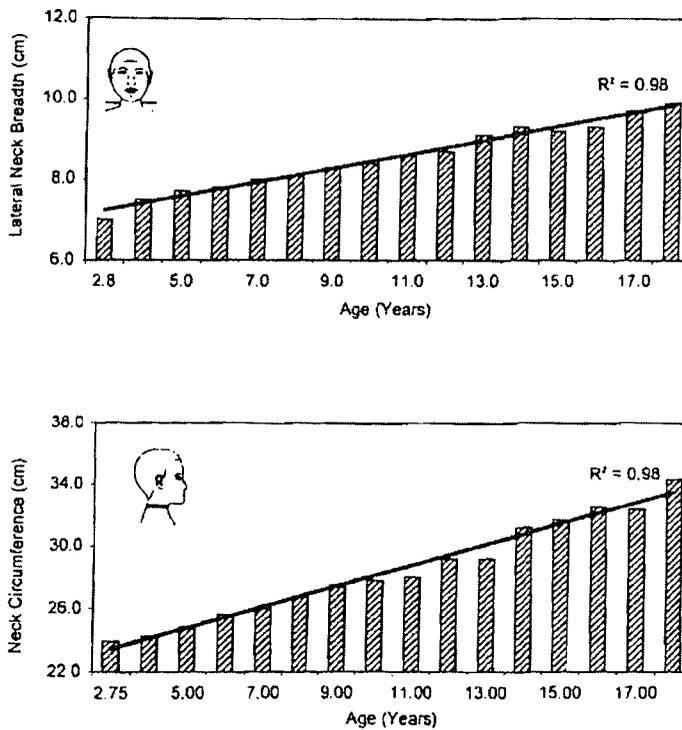


FIGURE 21.9. Variation of the neck circumference (bottom) and lateral breadth (top) as a function of age. The line shows the linear fit. (Data adapted from Snyder.¹¹⁸)

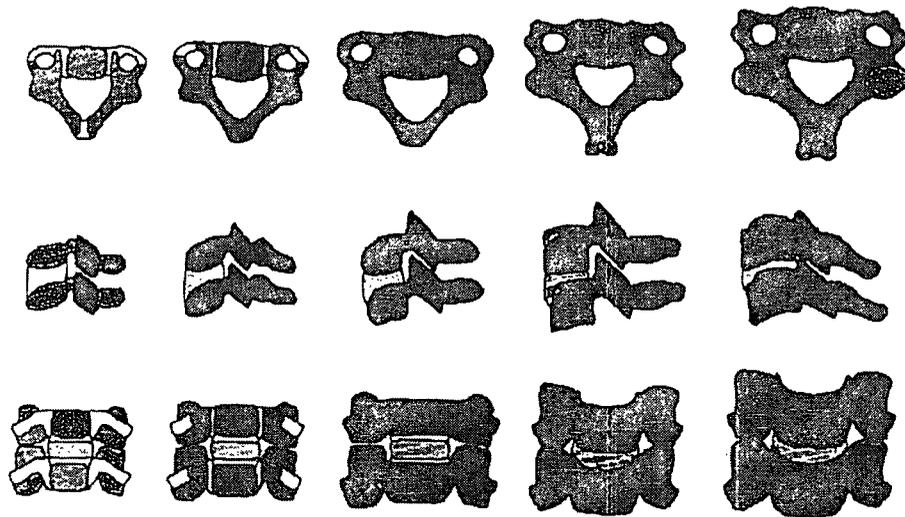


FIGURE 21.10. Typical cervical vertebrae (top row) as viewed from the top and functional units as viewed from the side (middle row) and front (bottom row). Typical growth patterns are illustrated. Starting from left, patterns represent groups I, II, III, IV, and adult spines. Cartilage is shown in lighter background and

bony components are shown in darker background. Secondary ossifications are shown in the darkest background for group IV (all three rows). In the middle row, the facet joints are expanded to illustrate changes in their orientation with skeletal development.

TABLE 21.1. Material properties applicable to the 1-year-old lower cervical spine.

Components	Young's modulus (MPa)		Poisson's ratio						
Vertebral centrum	75.0		0.29						
Growth plate	25.0		0.40						
Costal cartilage	25.0		0.40						
Posterior elements	200.0		0.25						
Posterior synchondrosis	25.0		0.40						
Neurocentral cartilage	25.0		0.40						
Disk annulus ground substance	4.2		0.45						
Disk annulus fibers	400		0.30						
Articular cartilages	10.4		0.40						
Synovial fluid (bulk modulus)	1,666.7								
Synovial membrane	12.0		0.40						
Ligaments									
Anterior longitudinal		Posterior longitudinal		Interspinous		Ligamentum flavum		Capsular	
Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)
1.4	28.4	1.0	23.2	1.3	13.5	1.9	36.7	1.8	42.9
2.7	51.9	2.0	41.1	2.7	19.5	3.7	65.9	3.9	70.3
4.1	71.8	3.0	57.0	4.0	23.6	5.6	95.7	5.8	87.5
5.4	86.9	4.0	68.6	5.4	26.3	7.5	106.9	7.7	100.6
6.8	95.7	5.0	75.8	6.7	27.9	9.4	117.8	9.7	107.8

Def., deflection.

Adapted from Kumaresan et al.⁶⁸

TABLE 21.2. Material properties applicable to the 3-year-old lower cervical spine.

Components		Young's modulus (MPa)				Poisson's ratio	
Vertebral centrum		75.0				0.29	
Growth plate		25.0				0.40	
Costal cartilage		25.0				0.40	
Posterior elements		200.0				0.25	
Neurocentral cartilage		25.0				0.40	
Annulus ground substance		4.2				0.45	
Disk annulus fibers		425				0.30	
Articular cartilages		10.4				0.40	
Synovial fluid (bulk modulus)		1,666.7					
Synovial membrane		12.0				0.40	

Ligaments									
Anterior longitudinal		Posterior longitudinal		Interspinous		Ligamentum flavum		Capsular	
Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)
1.4	30.2	1.0	24.7	1.3	14.4	1.9	39.1	1.8	45.6
2.7	55.2	2.0	43.7	2.7	20.7	3.7	70.1	3.9	74.7
4.1	76.3	3.0	60.6	4.0	25.1	5.6	101.7	5.8	92.9
5.4	92.3	4.0	72.9	5.4	27.9	7.5	113.6	7.7	106.9
6.8	101.7	5.0	80.5	6.7	29.7	9.4	125.1	9.7	114.6

Adapted from Kumaresan et al.⁶⁶

TABLE 21.3. Material properties applicable to the 6-year-old lower cervical spine.

Components		Young's modulus (MPa)				Poisson's ratio	
Vertebral centrum		75.0				0.29	
Growth plate		25.0				0.40	
Posterior elements		200.0				0.25	
Annulus ground substance		4.2				0.45	
Disk annulus fibers		450.0				0.30	
Articular cartilages		10.4				0.40	
Synovial fluid (bulk modulus)		1,666.7					
Synovial membrane		12.0				0.40	

Ligaments									
Anterior longitudinal		Posterior longitudinal		Interspinous		Ligamentum flavum		Capsular	
Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)
1.4	31.9	1.0	26.1	1.3	15.2	1.9	41.3	1.8	48.2
2.7	58.4	2.0	46.3	2.7	21.9	3.7	74.2	3.9	79.1
4.1	80.7	3.0	64.2	4.0	26.6	5.6	107.6	5.8	98.5
5.4	97.7	4.0	77.2	5.4	29.6	7.5	120.3	7.7	113.2
6.8	107.6	5.0	85.2	6.7	31.4	9.4	132.5	9.7	121.3

Adapted from Kumaresan et al.⁶⁶

TABLE 21.4. Material properties applicable to the 11-year-old lower cervical spine.

Components	Young's modulus (MPa)		Poisson's ratio						
Cortical shell	6,000.0		0.30						
Cancellous core	100.0		0.20						
End plate	150.0		0.30						
Uncinates	150.0		0.30						
Posterior elements	1,700.0		0.25						
Annulus ground substance	4.7		0.45						
Disk annulus fibers	475.0		0.30						
Articular cartilages	10.4		0.40						
Synovial fluid (bulk modulus)	1,666.7								
Synovial membrane	12.0		0.40						
Ligaments									
Anterior longitudinal	Posterior longitudinal		Interspinous		Ligamentum flavum		Capsular		
Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)	Def. (mm)	Force (N)
1.4	33.7	1.0	27.6	1.3	16.1	1.9	43.6	1.8	50.9
2.7	61.7	2.0	48.8	2.7	23.2	3.7	78.3	3.9	83.5
4.1	85.2	3.0	67.7	4.0	28.0	5.6	113.6	5.8	103.9
5.4	103.2	4.0	81.5	5.4	31.3	7.5	127.0	7.7	119.5
6.8	113.6	5.0	90.0	6.7	33.2	9.4	139.8	9.7	128.1

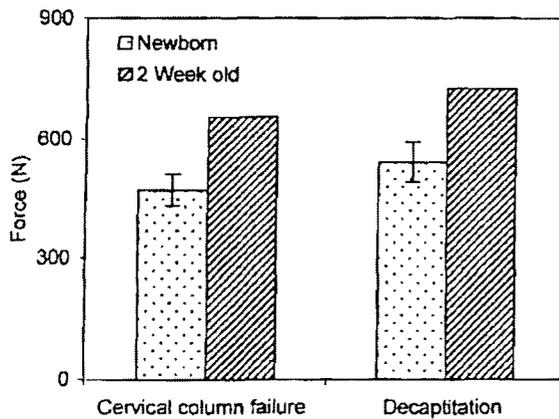


FIGURE 21.11. Biomechanical forces for spinal column failure and decapitation for the four newborn (standard deviation shown) specimens and one 2-week-old pediatric specimen. (Data adapted from Duncan.³⁵)

The following rationale was given to correlate the 10-week-old pig with the 3-year-old pediatric human. The weights of the 43 pigs (15.7 ± 1.0 kg) and three baboons (16.0 ± 1.5 kg) were similar to the 3-year-old human (14.9 kg). The thoracic and abdominal breadth dimensions of the pig (143 ± 24 mm and 144 ± 17 mm) and baboon (143 ± 16 mm and 112 ± 17 mm) were similar to the human (165 mm each). The

equivalent child age of the 15-kg pig was estimated to be 4.2 years and the baboon was estimated to be 16.3 years. The implication is that the strength of the anesthetized pig would be similar to that of the 3-year-old child. Despite these similarities, the pig has no chin protuberance for interaction because its neck attaches to the dorsal region of the skull, resulting in its snout being somewhat aligned with the

cervical column. The fore-aft range of motion of the head-neck structure of the pig is less than that of the child. This results in a smaller rearward motion to produce extension neck trauma in the pig. The neck circumference of the pig is approximately twice that of the child because of its large dorsal neck musculature. Other deficiencies include chest anthropometry (depth-to-width ratio of the pig is the inverse of that of the child) and head shape. The pigs were found to be susceptible to more injury than the baboon. The authors concluded that the pig and baboon were poor models for assessing the potential for child brain injury. This is because of the differences in head size between the quadruped animal and human.

Twenty-four of the 46 animals experienced significant neck injuries. Two animals suffered fatalities secondary to neck trauma. The most frequent neck injury was associated with hemorrhage in the occipitoatlantal joint capsules. This occurred in 20 of the 24 animals with significant (Abbreviated Injury Scale, AIS ≥ 3) neck injury.³ Three animals sustained posterior element fractures of the C5, C6, or C7 vertebra. Pathologic changes in the spinal cord occurred in four of the 24 animals with significant neck trauma. To determine the neck loads and moments, parallel testing was conducted using a Hybrid III 3-year-old dummy and its kinematics were matched with animal kinematics. A neck injury (AIS ≥ 3) risk curve based on neck tension measured with the 3-year-old dummy was derived. A force of 1,160N represented a 50% probability of injury.

In the later study, 15 paired dynamic airbag tests were conducted using 12- to 15-week-old piglets and a 3-year-old child dummy as test subjects.¹¹⁰ The child dummy was a Ford-built version of the GM child dummy, but with a different head to reduce ringing. Similar to the previous studies, for every piglet experiment a parallel matched dummy test was conducted under the same test condition. Four animals did not sustain head and neck trauma. There were four fatal neck injuries and one fatal brainstem injury. Neck injuries (including fatalities) occurred in seven of the 15 cases. Injuries were primarily concentrated at the occipitoatlantoaxial complex. Significant brain injuries

were accompanied by severe to fatal neck injuries. Upper cervical injuries were attributed to high tensile and bending stresses at the occipitoatlantal and atlantoaxial joints. Tensile loads generated at the occipitoatlantal joint were estimated by the vertical accelerations of the head multiplied by the head mass. For the piglet, tensile loads in the range of 1,500N for 11msec represented fatal neck injuries. Severe neck trauma was associated with a tension level of 2,100N for 3 to 6msec. The authors suggested the need to combine axial tension with bending moment for a composite neck injury indicator. This was accomplished using the force and moment time traces of the neck during the time of airbag deployment obtained from the matched-pair dummy test, and the combined peaks were recorded at a specific time. The constant stress line had values of 2,000N tension and 34Nm extension-moment (Fig. 21.12). Dynamic tensile studies using caprine model are being conducted by our group. Preliminary data are published elsewhere.¹⁰⁷

Tolerance

Two levels of injury tolerance based on the estimated probability of injury were reported.¹²⁰ This was based on a review of the literature, analysis of real-world pedestrian crashes, collision reconstruction, and dummy tests. To avoid irreversible injuries, a tolerance level of 818N for tension and 19Nm for flexion-moment were suggested. These values were, however, 990N and 100Nm, respectively, for a 25% probability of irreversible injury (AIS >2).

Using the principles of scaling from adult to pediatric, i.e., geometric similitude and accounting for mechanical property variations between the two groups, injury assessment reference values were obtained.⁸³ The Appendix at the end of this chapter provides the methodology. The calcaneal tendon strength data were used to determine the scaling factor for the tensile strength of the neck.¹³⁵ Implicit in this is the assumption that the developmental characteristics leading to the mechanical definitions are similar between the calcaneal tendon and the neck ligamentous tissues. The mechanical prop-

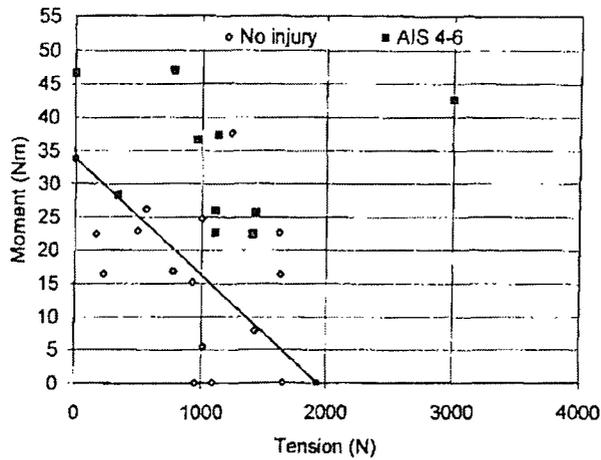


FIGURE 21.12. Moment versus axial force relationship for no injury and injury [Abbreviated Injury Scale (AIS) 4–6] cases. The constant stress line is indicated. (Redrawn from Prasad and Daniel.¹¹⁰)

TABLE 21.5. Proposed critical N_{ij} intercept values.

Dummy	Tension (N)	Compression (N)	Flexion (Nm)	Extension (Nm)
CRABI 12-month-old	2,200	2,200	85	25
Hybrid III 3-year-old	2,500	2,500	100	30
Hybrid III 6-year-old	2,900	2,900	125	40

Adapted from Kleinberger,⁸² 1993

erties of the human calcaneal tendon were approximated by a linear fit between zero (newborn) and 6 years of age.

Extrapolating the previous quasi-static data that had an average of 507 N for the newborn resulted in a value of 811 N to the 6-month-old. Data were extrapolated from the matched dynamic pig versus 3-year-old dummy airbag tests (described earlier). The scaled value for the 6-month-old was 895 N. These two values obtained from different testing protocols (static from the newborn human cadaver and dynamic from the pig-dummy paired airbag deployment study) correspond to high probabilities of injuries including fatalities. A comparison of the peak upper neck tension (injury assessment reference value) obtained from the various methods is included in Fig. 21.13. Based on dummy tests, the peak upper neck tensile force of 500 N was suggested to be the injury assessment reference value for the 6-month-old infant dummy.

Using the data set from the two previously described piglet dynamic airbag studies as a

basis, a new neck injury criterion, termed N_{ij} , has been proposed based on a combination of axial forces and moments.⁶² The criterion proposes critical limits for the four combinations of neck loading: tension or compression combined with flexion or extension (Table 21.5). N_{ij} is defined as the sum of the normalized forces and moments.

$$N_{ij} = F/F_{int} + M/M_{int}$$

where F represents axial force, M represents bending moment, and the subscript "int" denotes the critical intercept value used for normalization. In the initial analysis, the critical intercepts were obtained as follows. Statistical analysis of the earlier piglet data (1982) indicated that tension in the neck of the dummy had the strongest statistical relationship with pig injuries.⁸⁵ Little improvement in injury predictability was found when moment was added to the analysis. In contrast, the later piglet data (1984) indicated that a linear combination of tension and extension moment explained the pathologic observations.¹¹⁰ Reanalysis of all

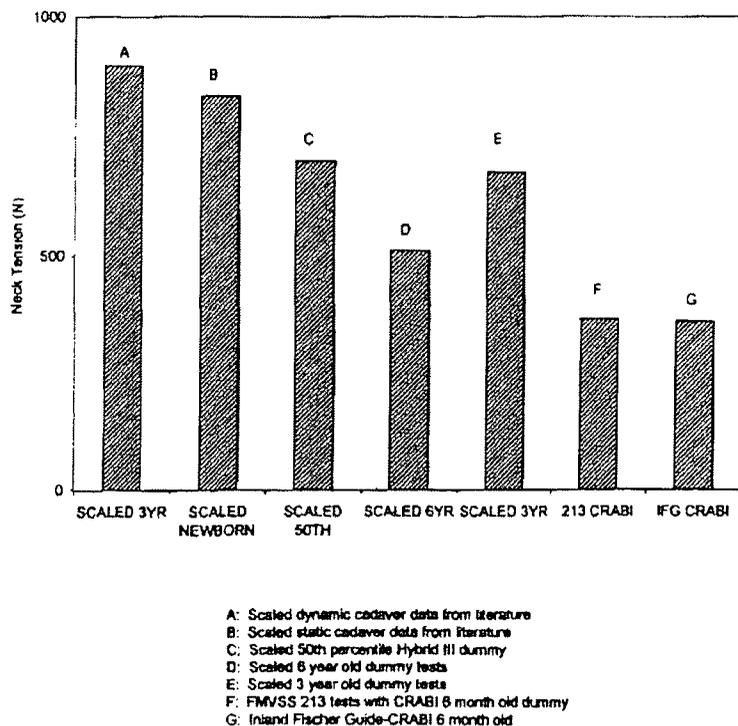


FIGURE 21.13. Comparison of peak upper neck tension injury assessment reference values obtained for the 6-month-old dummy using various methods.

The value for the 50th percentile dummy is 3,300N (not shown). (Data adapted from Melvin⁴³ and Prasad and Mertz.¹¹¹)

these data was conducted to determine the level of tension and extension moments that best predicted the injury outcomes. The result of this analysis is the basis of the proposed tension-extension requirement. For the 3-year-old dummy, the critical tension and extension intercepts were determined to be 2,500N and 30Nm. The tension and extension from the 3-year-old values were scaled to the 12-month to 6-year-old dummies. Forces and bending moments were scaled according to the second and third powers of the neck length (see Appendix). Neck circumference was used to quantify neck length because of the simplicity in obtaining such data. It was argued that since material stiffness variations were already incorporated into the dummy neck design, neck injury criteria were scaled using only geometric factors. Critical intercept values for flexion were set by maintaining the same ratio (190/57

= 3.3) between midsize adult male flexion (190Nm) and extension (57Nm); the adult values stem from human cadaver studies.^{86,87,146} Compressive tolerance was based on adult human cadaver studies.^{96,109,156} Tension tolerance was chosen to be the same as compression based on adult cadaver data.^{62,147} Scaled critical intercept values for various dummies are given (Fig. 21.14, Table 21.5). The N_{ij} limit of 1.0 corresponds to a 15% risk of serious neck injury. A limit of 1.4 corresponds to a 30% injury risk. It must be noted that the proposed intercept values and limits are only tentative. Consequently, modified numerics may be available in the near future.

Since the above determination of scaling factors did not include specific changes to the material properties of the spinal components, more recently Yoganandan et al¹⁵⁹ synthesized mechanical property data of components such

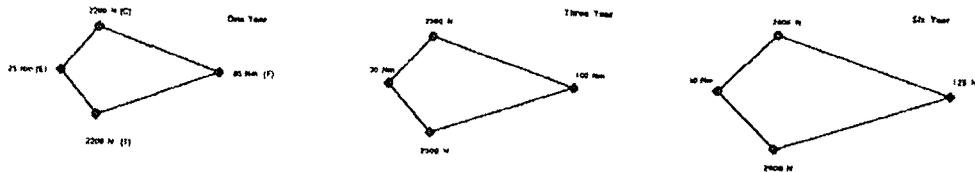


FIGURE 21.14. Proposed scaled intercept values for the 1-, 3- and 6-year-old pediatric human neck under flexion (F) and extension (E) moments, and compression (C) and tension (T) forces.

TABLE 21.6. Scale factors (as a function of loading mode) derived from combined spinal component material and geometric analysis.¹⁵⁹

Age group	Tension	Extension	Compression	Flexion
1 year old	0.26	0.22	0.26	0.23
3 years old	0.29	0.32	0.28	0.33
6 years old	0.35	0.41	0.34	0.42
Small female	0.63	0.7	0.63	0.7
Adult male	1.00	1.00	1.00	1.00

as the vertebra, ligaments, cartilage, spinal cord, muscles, and disks; information was obtained from studies in literature and in-house tests. Neck geometric data were included in the determination of scaling factors. Under each loading mode, variations in the mechanical strength of the individual components were combined with the geometric parameters for each age. For example, at a specific age, under compression, material properties of the vertebra, disk, and cartilage were averaged to obtain a materially scaled factor using the adult male as standard. The overall neck cross-sectional area factor for this age was multiplied by the above age-specific determined material factor to obtain the combined scaling factor. Similar procedures were adopted for tension, extension, and flexion. The derived scale factors using this combined spinal material and geometrical approach as a function of age and loading mode are given in Table 21.6.

Head

Developmental Anatomy

The anatomy of the head of the child is dynamic and its various aspects develop with age.^{134,146,161}

Although general features are present, the basic underlying bony characteristics do not develop until 2 years of age in most cases, and completion of growth does not occur until the second decade of life. The skull and brain are the principal components of the head.

Skull

The neurocranium houses the brain, face, and base, which are the three main parts of the skeletal structures of the adult head. The term *skull* generally refers to the entire bony structure and *cranium* refers only to the fused regions, i.e., without the mandible. The adult neurocranium is a series of irregularly shaped fused flat bones. Eight bones make up the neurocranium: frontal, two parietal, two temporal, occipital, sphenoid, and ethmoid. The sphenoid and ethmoid bones provide junction with the face and anterior base of the skull. The calvarium consists of the frontal, parietal, and occipital bones that form the bulk of the convexity on the top. Figure 21.15 illustrates the superior aspect of the adult and newborn skull. In the newborn, the occipital condyles are elongated and flat instead of the curved shape as seen in the adult. The tympanic rings form the prominent features of the base of the skull. In addition, they provide attachment for the tympanic membranes. The calvaria extends laterally and posteriorly beyond the base of the skull.

Unlike the mature adult human cranial bone, which consists of two layers of rigid cortical bone (inner and outer tables) housing the relatively deformable cancellous (diploe) layer, in the newborn the cranial bone is primarily cortical, with no diploe component. Around 3 to 6

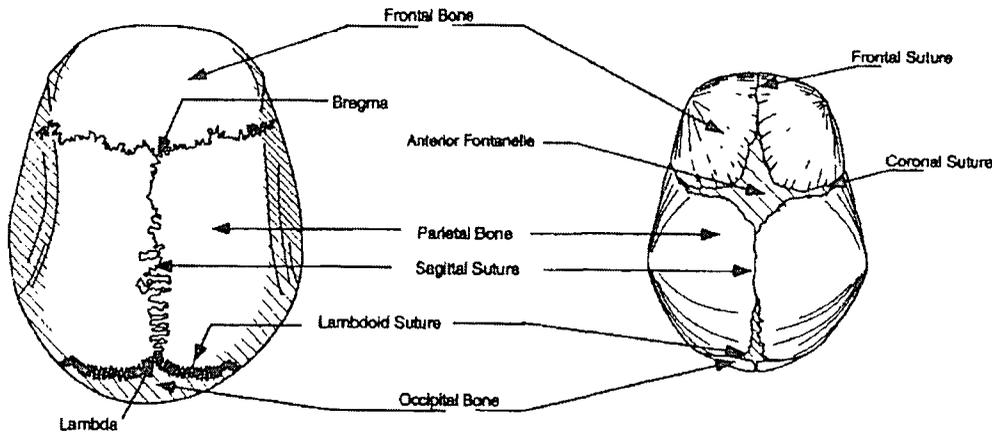


FIGURE 21.15. Superior aspect of the adult (left) and newborn (right) skull. Various bones and sutures are indicated. (Adapted from Williams.¹²⁴)

months of age, structurally, the growth proceeds to gradually transform into the sandwich composition of the adult skull. At birth, the calvarial bones are thin and ossification does not extend into the suture lines of the skull. The frontal bone is divided along the midline by the metopic suture and outlined posteriorly by the coronal suture. The parietal bones are two symmetric plates covering the lateral aspect of the skull. They are separated by the sagittal suture in the midline. Anteriorly, they are abutted by the coronal suture, posteriorly by the lambdoidal suture, and inferiorly by the squamosal suture. The occipital bone, or more specifically the squamous portion of the occipital bone, is formed by the lambdoidal suture that traverses symmetrically from the lambdoid and merges into the posterolateral fontanelle. With age, the sutures continue to diminish in size, but closure occurs at different intervals. The metopic suture closes during the sixth to eighth year of life. Complete fusion of the bony plates with obliteration of the sutures occurs around 20 years of age when the skull has reached its full definitive size.

The soft spots or fontanelles of a newborn head are large patches of fibrous tissue located between the bony plates of the skull.^{2,122,128,134,160} Fontanelles can be compared to the synchondrosis of the cervical spine (Fig. 21.16). They are

not fused in the newborn. There are six major fontanelles with variable smaller or accessory fontanelles usually located along the sagittal suture. The six fontanelles are the anterior, posterior, and the paired anterolateral and posterolateral fontanelles. The anterior fontanelle is located along the bregma or the intersection of the coronal, sagittal, and metopic sutures. This fontanelle is the largest at birth with an average diameter of 25 mm. The posterior fontanelle is located along the lambda, which is the intersection of the sagittal and lambdoidal sutures. The anterolateral fontanelles are located along the pterion, which is the intersection of the coronal and squamosal sutures. The posterolateral fontanelles lie along the asterion or the intersection of the lambdoidal and squamosal sutures. Closure of the fontanelles occurs at various times. The posterior and anterolateral fontanelles close within 2 to 3 months after birth, and the posterolateral fontanelle closes approximately at 1 year. In contrast, the anterior fontanelle closes at around 18 to 24 months of age.

Brain

We begin with the prenatal development of the brain.^{12,134} After conception, the clump of rapidly dividing cells initially begin to resemble

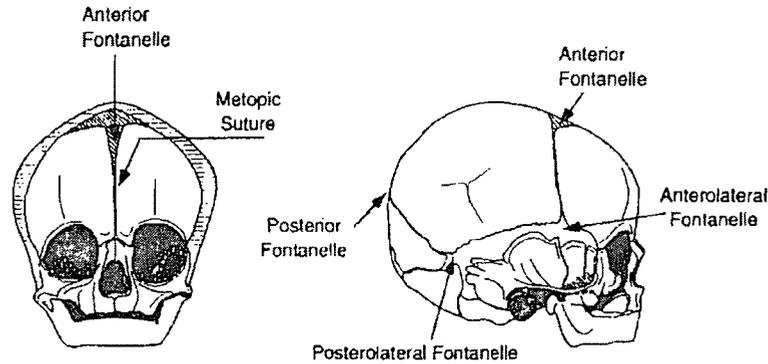


FIGURE 21.16. Front and side views illustrating the fontanelles. (Redrawn from Agur and Lee,² Tindall et al,^{12b} Williams,^{13a} and Youman.^{16d})

a more organized pattern forming the embryo. The cells of the human embryo are categorized into layers. These are the ectoderm (outer), mesoderm (middle), and endoderm (inner), named by the location of the cell layers. Along the ectoderm, a specialized region of cells forms the neural plate from which the structures of the nervous system are formed by cellular division. This process of growth is termed neural induction. As the cells along the outermost edge proliferate more rapidly, it creates an indentation or "groove" along the midline known as the neural groove. This process continues with the margins growing up and around to form the neural tube, with closure initially at the area of the neck, and proceeding in the rostral and caudal directions. The various components of the central nervous system develop from the neural tube. Once closed, the neural tube represents all components of the nervous system with the rostral portion forming cerebral hemispheres and brainstem, and the caudal portion forming the spinal cord. From the mesodermal layer comes the formation of the skeletal structures that surround and protect the nervous system. Cells from this middle layer develop into specialized regions and form paired, block-like masses known as somites. These regions are arranged around the neural tube in a symmetric pattern and develop into the vertebral column and segmental musculature. These events then form the basic blueprint

of the human skeleton and nervous system from which growth in utero will proceed until birth. Approximately, the first 5 years of human life correspond to a rapid increase in development of the brain. This is characterized by a decrease in water content with an increase in the number of glial cells and dendritic branches, and synaptic connection of neural cells and axon myelination.

Age-Related Grouping and Material Properties

As indicated above, the growth of the human head does not parallel the growth of the other structures of the human body (e.g., neck). Consequently, age-related grouping for the neck structures based on vertebral growth cannot be directly translated into the age-related grouping for the pediatric head structures. As an initial approximation, based on some of the characteristics of the developmental processes described above, it is possible to obtain an age-related grouping. Sequential closure of the fontanelles when applied to this type of a classification results in the following: newborn (zero) to 3 months corresponding to closure of the posterior and anterolateral fontanelles, 1 year corresponding to closure of the posterolateral fontanelle, and approximately 2 years

corresponding to closure of the anterior fontanelle. Other classifications are also possible. Table 21.7 includes the representative material properties used in the literature for the various components of the head in the mathematical models.^{114,126} Compared to the adult model, the same level of sophistication has not been achieved in the pediatric models. This is because data on the material properties of the pediatric human skull, suture, and brain tissues are sparse. Furthermore, unlike the neck structure, finite element modeling of the head is still very much in its infancy.

TABLE 21.7. Material properties used in human finite element model.

1-month-old			
	Young's modulus (MPa)	Density (kg/m ³)	Poisson's ratio
Cranial bone	1,300	2,150	0.28
Suture	200	1,130	0.28
Brain	2,110	—	—
Foramen magnum	100	—	—

3-month-old			
	Young's modulus (MPa)	Poisson's ratio	Stiffness (N/mm)
Cranial bone	880	0.28	—
Suture	—	0.28	189
Brain	2,110	—	—
Foramen magnum	100	—	—

Adapted from Thibault et al.¹²⁶

Adapted from Runge et al.¹¹⁴

Biomechanical Responses

While studies describing the mechanical properties of the adult human cranial bone under compression, tension, bending, and shear are available, a paucity of such information exists for the developing pediatric population.^{56,77,78,80,84,95,152} Specific to the growing population, in an earlier study, cranial bone specimens from two full-term newborns (gestation age 40 weeks) and one 6-year-old human cadaver were tested using three-point bending technique at a quasi-static rate of 0.5 mm/min.^{66,67,82} In the two newborn specimens, frontal and parietal bone samples were tested. In the 6-year-old specimen, parietal bone samples were used. Tests were conducted along an axis parallel and perpendicular to the long axis of the specimen. The maximum deflection used in these tests was 1.5 mm. Paired Student's t-tests using the data from the 6-year-old parietal specimen indicated differences in the modulus ($p < .001$) between parallel (7.38 ± 0.84 GPa) and perpendicular (5.86 ± 0.69 GPa) orientations. Significant differences ($p < .001$) in the elastic modulus were also found between parallel (3.88 ± 0.78 GPa) and perpendicular (0.951 ± 0.572 GPa) fiber orientation for the newborn (Fig. 21.17). In addition to these data, tests were conducted on four fetal cadavers with gestational age ranging from 25 to 40 weeks (Table 21.8). Significant differences ($p < .001$) in the elastic modulus were found between parallel (1.65 ± 1.17 GPa) and perpendicular (0.145 ± 0.062 GPa) orientations. In a

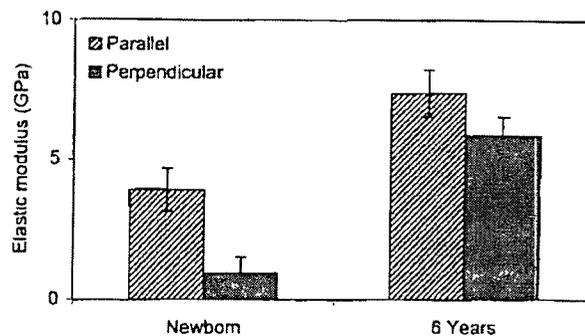


FIGURE 21.17. Comparison of the elastic modulus between the newborn and 6-year-old human cadavers in three-point bending under parallel and perpendicular orientations.

TABLE 21.8. Elastic modulus from three-point bending tests on six human cadavers.

Gestational age (week)	Elastic modulus (GPa)							
	Parallel orientation				Perpendicular orientation			
	Parietal bone	<i>n</i>	Frontal bone	<i>n</i>	Parietal bone	<i>n</i>	Frontal bone	<i>n</i>
25 ± 2	1.30 ± 0.6	8	—	—	0.12 ± 0.01	8	—	—
27 ± 2	0.94 ± 0.41	10	—	—	0.18 ± 0.03	3	—	—
28 ± 2	3.62 ± 0.46	5	—	—	0.14 ± 0.08	5	—	—
38 ± 2	4.24 ± 0.73	9	—	—	0.84 ± 0.19	8	—	—
40 ± 2	4.01 ± 1.28	3	3.05 ± 0.88	2	1.74 ± 0.59	3	1.70 ± 0.79	2
40 ± 2	3.51 ± 0.50	10	3.06 ± 0.84	10	0.57 ± 0.14	5	—	—

n, number of samples tested in each cadaver.

Adapted from McPherson and Kriewall.⁸²

TABLE 21.9. Biomechanical properties of human fetal cranial bone under three-point bending. Data obtained from 10 cadavers.

Gestational age (week)	Bone	Loading rate (mm/sec)	Stiffness (N/mm)	Elastic modulus (MPa)	
				Parallel orientation	Perpendicular orientation
20	Parietal	0.008	4.1	4,232	1,238
30	Parietal	0.008	3.3	3,478	1,044
30	Parietal	0.008	1.4	3,634	1,626
33	Parietal	0.008	2.3	3,029	1,996
34	Parietal	0.008	4.7	3,390	1,646
39	Parietal	0.008	17.5	4,301	—
40/newborn	Parietal	0.008	25.1	5,167	1,434
40/newborn	Parietal	0.008	20.4	2,961	—
40/newborn	Parietal	0.008	6.8	3,594	1,684
2-week-old	Parietal	0.008	14.1	7,360	—

Adapted from Kriewall.⁶⁶

later study, 10 human cadavers with age ranging from 20 to 42 gestational weeks were tested using three-point bending techniques at a loading rate of 0.0083 mm/sec. The bending stiffness ranged from 1.4 to 25.1 N/mm. The elastic modulus for the perpendicular orientation (1.044 to 1.996 GPa) was lower than the modulus for the parallel orientation (3.02 to 7.36 GPa). These data, on a specimen-by-specimen basis, are included in Table 21.9.

In a more recent study, cranial bone specimens were tested from human cadavers.^{114,125,126} In the initial study, 12 samples were used from four cadavers (25 and 30 weeks' gestation, and 1 week and 6 months of age). The parietal bone test specimens were extracted bilaterally parallel to the sagittal suture with approximate

dimensions of 20 to 25 mm length by 3.5 mm width. They were subjected to failure using three-point bending techniques at a loading rate of 0.042 or 42.33 mm/sec. All specimens had a constant span of 17 mm with the exception of the 6-month-old specimen, which had a span of 30 mm. For testing, the samples were oriented with the fibers perpendicular to the long axis of the bending specimen. Failure occurred in all tests under the load point on the tensile side of the specimen. Rupture and elastic moduli, and energy absorbed to failure properties (Table 21.10) demonstrated increasing tendencies with advancing age. In a later study, data were reported from three-point bending tests conducted at loading rates between 0.03 and 30 mm/sec on four subjects

TABLE 21.10. Biomechanical data from human and porcine tests.

Species	Age	Specimen type	Sample		Load rate (mm/sec)	Load type	Yield		Ultimate		Energy (100 × Nm/m ³)	Young's modulus (MPa)
			#	Size			Stress	Strain (%)	Stress	Strain (%)		
Human*	25 wks	L-Parietal	1		0.042	3-pt bend			4.5		312	71.6
	25 wks	L-Parietal	2		42.33	3-pt bend			4.0		—	43.8
	30 wks	L-Parietal	1		0.042	3-pt bend			3.1		—	95.3
	30 wks	R-Parietal	2		42.33	3-pt bend			11.2		624	444.5
	30 wks	R-Parietal	3		0.042	3-pt bend			14.9		—	618.8
	30 wks	R-Parietal	4		42.33	3-pt bend			8.9		575	407.7
	30 wks	R-Parietal	5		42.33	3-pt bend			17.0		675	455.4
	1 week	L-Parietal	1		42.33	3-pt bend			10.6		607	820.9
	6 mos	L-Parietal	1		0.042	3-pt bend			42.1		1,392	2,111.7
	6 mos	R-Parietal	2		0.042	3-pt bend			44.6		1,834	2,199.4
	6 mos	L-Parietal	3		42.33	3-pt bend			—		—	2,671.9
	6 mos	R-Parietal	4		42.33	3-pt bend			71.7		4,361	5,582.2
	Porcine*	2-3 days	Bone	11		0.042	3-pt bend			17.4 ± 2.1		1,009 ± 182
2-3 days		Suture	6		0.042	3-pt bend			12.4 ± 6.6		1,127 ± 327	194.2 ± 42.5
2-3 days		Bone	13		42.33	3-pt bend			41.4 ± 6.6		1,688 ± 338	1,371.4 ± 275.8
2-3 days		Suture	7		42.33	3-pt bend			30.8 ± 5.1		1,737 ± 415	610.3 ± 122.6
Porcine	2-3 days	Bone	6		0.042	Tension	5.3 ± 0.9	0.79 ± 0.1	10.6 ± 1.6	3.41 ± 0.69	75 ± 22	809 ± 118.9
	2-3 days	Suture	3		0.042	Tension	5.7 ± 0.9	4.22 ± 0.4	7.7 ± 0.8	6.64 ± 0.78	33 ± 5	171.5 ± 32.5
Porcine	1-yr-old	Outer table	13		0.042	Tension	6.7 ± 0.7	1.06 ± 0.9	13.5 ± 1.0	4.54 ± 0.27	25 ± 3	802.3 ± 78.8
	1-yr-old	Diploe	12		0.042	Tension	4.0 ± 0.7	1.09 ± 0.31	7.2 ± 0.7	3.02 ± 0.26	9 ± 1	543.2 ± 56.6
	1-yr-old	Outer table	10		42.33	Tension	8.1 ± 1.0	0.89 ± 0.08	18.5 ± 1.4	4.15 ± 0.42	34 ± 5	1,058.6 ± 118.4
	1-yr-old	Diploe	7		42.33	Tension	2.6 ± 0.5	0.45 ± 0.11	11.1 ± 1.0	2.77 ± 0.49	10 ± 2	858.2 ± 123.0

* Stress values correspond to rupture stress. See text for details. Stress values in MPa. Adapted from Thibault et al.¹²⁶

with ages ranging from 3 to 7-years. The specimens included parietal and occipital bones. They were approximately 20mm long by 7mm wide by 1 to 2mm thick. The material properties expressed in terms of elastic modulus showed differences between the newborn and

7-year-old specimens. Force-deflection behavior of a 3-month-old parietal bone tested at the rate of 30mm/sec is included in Fig. 21.18. A comparison of these data illustrating the age dependency in the material properties is included in Fig. 21.19. The bar chart also

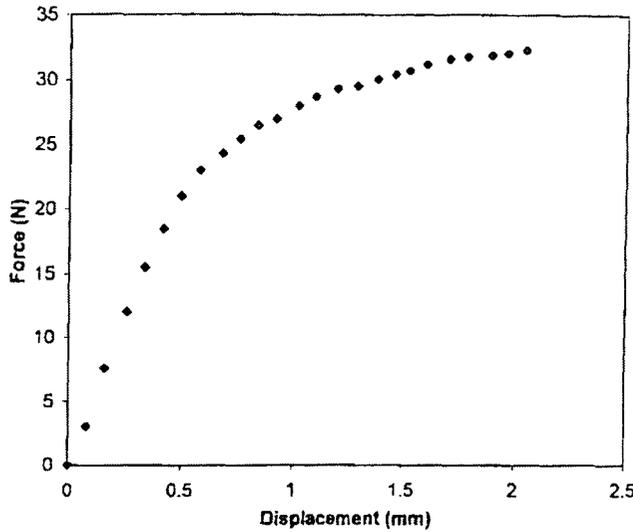


FIGURE 21.18. Force-deflection behavior of a 3-month-old parietal bone specimen tested in three-point bending at a loading rate of 30mm/sec. (Data adapted from Runge et al.¹¹⁴)

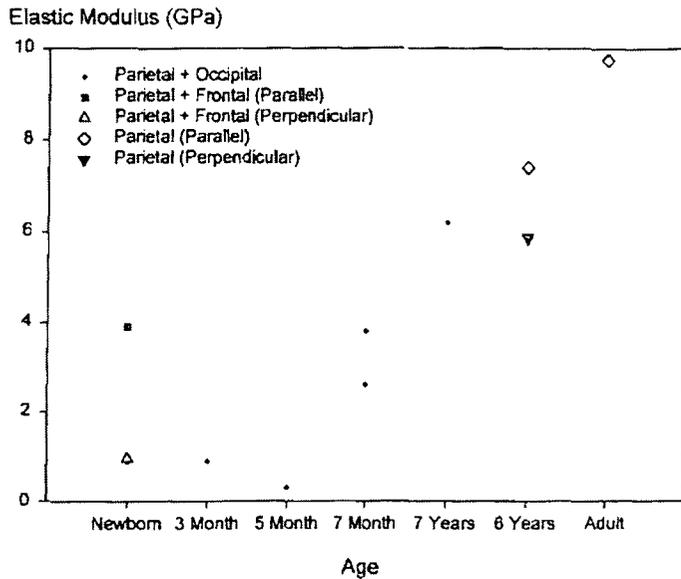


FIGURE 21.19. Elastic modulus of the cranial bone. (Adapted from Hubbard,⁵⁶ Kriewall et al,⁶⁷ McPherson and Kriewall,⁸² Runge et al,¹¹⁴ and Thibault et al.¹²⁰)

includes the embalmed adult human parietal bone specimens tested using three- and four-point bending techniques.⁵⁶

Although investigations have been conducted delineating the characteristics of the adult sutures, biomechanical studies of sutures of the human skull in the pediatric age group have received less attention.^{56,57} Coronal, sagittal, and metopic sutures from donors with age ranging from 3 to 15 months were subjected to tensile tests at an axial loading rate between 0.03 and 30 mm/sec.¹¹⁴ Force-deflection characteristics indicated a nonlinear behavior typical of biologic materials (Fig. 21.20). The stiffness was defined as the slope in the linear portion of the curve. The mean suture stiffness was 189 N/mm for the six samples.

Because of similarities in the anatomic characteristics between the porcine and human, and because of a lack of detailed information on pediatric human cadaver biomechanics, some data are presented from porcine tests. The porcine brain is similar to the human brain in terms of growth and development.^{32,125,127} The 2- to 3-day-old piglet is representative of a less than 1-month-old human newborn. The 1-year-old piglet is representative of a more than 4-year-old human. Additional rationale for the use of the porcine model is given later.

Biomechanical properties of the 2- to 3-day-old and 1-year-old porcine cranial bone and suture (Table 21.10) were determined.¹²⁵ Left or right frontoparietal bones excised from 20 porcine cadavers (2- to 3-days-old) were tested using three-point bending technique. The specimens had a span of 17 mm. For cranial bone tests, the specimens were obtained from the parietal region (parallel to the suture). For cranial bone-suture tests, the specimens were obtained from the frontoparietal region with a portion of the coronal suture penetrating the cross section. Loading rate was set at 0.042 or 42.33 mm/sec. The specimens (5 mm wide by 25 mm long) were oriented with fibers perpendicular to the long axis of the specimen. In the second series of experiments, uniaxial tension tests (3- to 5-mm gauge length) were conducted on a separate group of ten 2- to 3-day-old piglets. The parietal or frontoparietal bone specimens included the coronal suture. The loading rate was set at 0.042 mm/sec. For comparison, in the third series of experiments, similar axial tension tests were conducted on the outer table and diploe specimens from ten, 1-year-old pigs at 0.042 and 42.33 mm/sec rates of loading. Biomechanical data from these three series of experiments are presented in Table 21.10. The bending elastic modulus of the

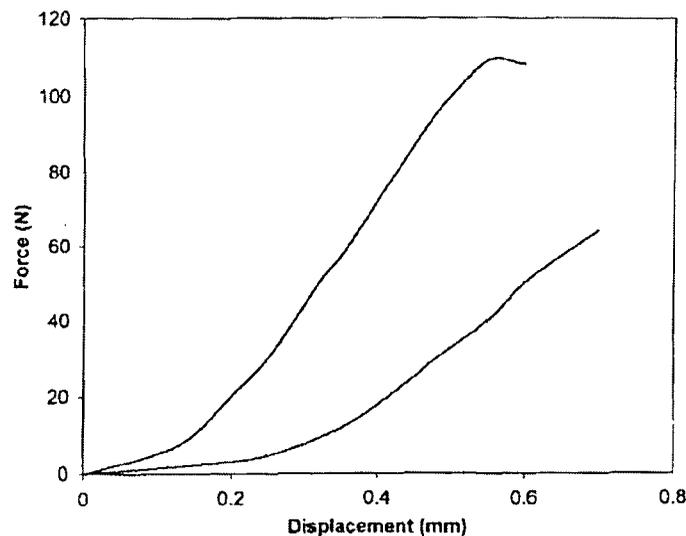


FIGURE 21.20. Tensile force-displacement curves (range shown) of the coronal suture of a 3-month-old human skull. (Adapted from Runge et al.¹¹⁴)

2- to 3-day-old piglet bone was greater ($p < .05$) than the suture at both rates of loading. In contrast, the bending elastic modulus and rupture stress of the 2- to 3-day-old piglet bone and suture were greater ($p < .05$) at 42.33 mm/sec than at 0.042 mm/sec. Significant differences ($p < .05$) were also apparent between the bone and suture in the tensile yield strain, ultimate strain, and tensile modulus. The ultimate tensile stress for the 1-year-old pig outer table and diploe, and tensile modulus for the diploe were higher ($p < .05$) at 42.33 mm/sec than at 0.042 mm/sec. Except for the tensile yield strain at 0.042 mm/sec and tensile modulus at 42.33 mm/sec, all other variables (tensile yield stress and strain, tensile ultimate stress and strain, energy, and tensile modulus) demonstrated differences ($p < .05$) between the outer table and diploe. These data indicate variations in the mechanical properties of the constituents of the skull with age.

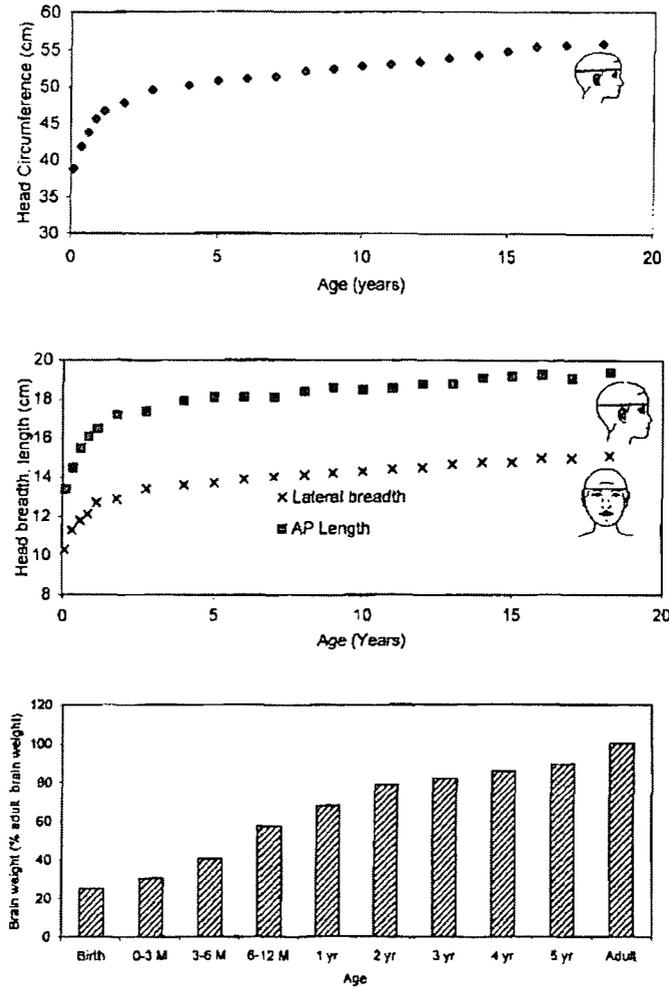
Difficulties exist with regard to characterization of the mechanical properties of soft tissue structures using autopsy material because of its quick deterioration postmortem.^{39,52,90} It is well known that mechanical properties such as stress-relaxation characteristics differ significantly between live and dead brain tissue, and the variations may be due to alterations in the parenchyma and vasculature pressure.¹⁰² Consequently, if one were to use autopsied brain tissue for the determination of the mechanical properties, time would be a critical factor. Porcine studies have been conducted to determine the mechanical response of the brain (to shear loads) within 3 hours postmortem.¹²⁵ However, practical and logistic constraints may limit the use of human cadaver brain to directly determine mechanical properties. Although it is possible to obtain tissues from operating rooms during surgery (with the approval of the Human Studies Committee), they may not all be fully usable for biomechanical material property testing because of their abnormal conditions. To circumvent this difficulty, researchers have begun to use clinical, experimental, and finite element modeling methods. This includes limited testing of pediatric brain material obtained during certain neurosurgical procedures, testing of porcine brain tissues,

testing of human skull bones, testing of porcine skull bone and sutures, and correlating experimental and modeling porcine data with the human results, thus indirectly establishing a rationale to employ age-dependent porcine brain material properties to the human.^{114,125-127} Further discussion on the characterizations of the porcine skull and brain as applied to the pediatric population is presented below. With this as a focus, the biomechanical responses of the developing porcine are included in this chapter.

Samples of frontal cerebrum (free from sulci) from 2- to 3-day-old piglets ($n = 12$) and 1-year-old pigs ($n = 12$) were tested within 3 hours postmortem.¹²⁷ The disk-shaped specimen was approximately 1 to 2 mm in thickness and 10 to 12 mm in diameter. Each specimen was subjected to oscillatory simple shear strain amplitudes of 2.5% or 5% from 20 to 200 Hz.⁵ The shear modulus was found to be different ($p < .05$) between the 2- to 3-day-old and the 1-year-old pig brains, and the complex shear modulus of both groups increased with strain rate.

The developmental characteristics of the human head expressed in terms of its breadth, length, circumference, and body and brain weight are included in Fig. 21.21.^{27,33,118,123,134} Structural differences have also been quantified in the form of stiffness between the adult and child skull.¹¹⁹ Using previously reported data on the anterior-to-posterior force-deflection properties from twelve adult unembalmed human cadaver heads, age-dependent stiffness can be obtained.^{78,79} These data indicate that the stiffness is a nonlinear function of age attaining 75% of the adult value between 6 and 9 years, 90% at 13 years, and full adult value at 20 years of age (Fig. 21.22). The numeric changes in stiffness represent the differences in maturation processes of the skull bones. In deriving this relationship, it is assumed that the ratio of stiffness of the skull of the newborn to the adult is equivalent to the ratio of compressive strength of the femur of the newborn to the adult human.¹³⁵ A comparative anatomy of the adult and the newborn human skull is given in Figs. 21.15 and 21.16.

FIGURE 21.21. Variation of the head circumference (top), head breadth and length (middle), and brain weight expressed as a percentage of adult brain weight (bottom) as a function of age. (Adapted from Snyder et al.¹¹⁹)



Tolerance

The widely used tolerance, head injury criterion (HIC), was developed in the 1970s. The basis of the HIC lies in the use of the Wayne State University tolerance curve.^{8,45,116,131,146} This was developed by dropping embalmed human cadaver foreheads onto unyielding flat surfaces. In its final form, the tolerance curve was developed by combining the results from a wide variety of pulse shapes, cadavers, animals, human volunteers, clinical research, and injury mechanisms. Skull fracture and/or concussion were used in the failure criterion except for long-duration human volunteer tests wherein

no apparent injuries were reported. The current HIC limit of 1,000 applies to the midsize male.^{1,8} The criterion is given by the following equation:

$$HIC = \max \left[\left\{ \frac{1}{t_2 - t_1} \right\} \int_{t_1}^{t_2} a(t) dt \right]^{2.5}$$

where t_1 and t_2 are arbitrary times during the acceleration phase, and $a(t)$ is the resultant acceleration response. Attempts have been made to establish pediatric head injury tolerance using adult data for the HIC and angular acceleration thresholds (see Table 21.12 in the

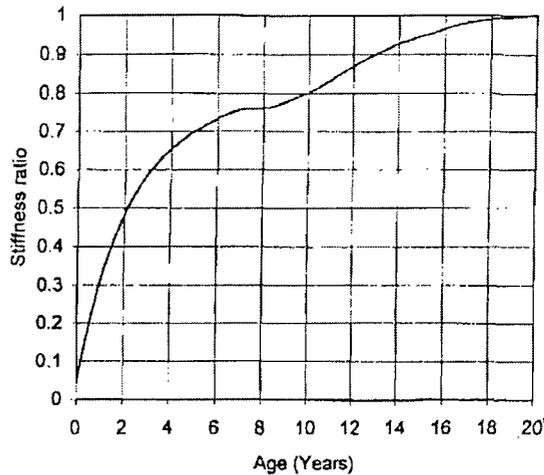


FIGURE 21.22. Variation of skull stiffness from the newborn to the adult (20 years of age). Data indicated as the ratio of the stiffness at a particular age to the average adult stiffness. (Data adapted from Snyder et al.¹¹⁹)

Appendix). A widely used procedure for the angular/rotational acceleration is based on the mass of the brain.¹⁰¹ This relation was based on noncontact, inertial loading tests using primates.

$$\ddot{\theta}_H = \ddot{\theta}_A [M_A/M_H]^{2/3}$$

where subscripts H and A refer to the human and animal, M is the mass of the brain, and the double dot represents rotational acceleration. The underlying assumption is that the density and material property characteristics between the animal (model) and the human (prototype) are equal. This equation results in a value of 1,700 rad/sec/sec for the angular acceleration threshold for adult human. Using this approach and assuming the mass of the infant brain to be 0.5 kg, pediatric head injury tolerances have been obtained.³⁴ Because developmental and anatomic differences exist between the pediatric and the adult skull and brain (see earlier sections), this equation was modified in later studies.¹²⁷ To incorporate the age-related material property changes in the brain tissues, recent research has indicated that, at low shear strains, the above mass-related equation can be modified as follows.

$$\ddot{\theta}_C = \ddot{\theta}_H [M_H/M_C]^{2/3} \{G'_C/G'_H\}$$

The rotational acceleration threshold of the child (represented by subscript C) is obtained by multiplying the rotational acceleration threshold of the adult human (represented by subscript H) by the product of the ratio of the adult brain mass to the child brain mass raised to the two-thirds power and the ratio of the storage modulus of the child to the storage modulus of the adult human. The use of this modified scaling relation reduces the rotational acceleration threshold. The validity of this relationship, however, is unknown at large strain levels (beyond 5%). Further research in this area is needed. It should be reiterated that this is applicable only to noncontact, inertial load applications. More recent human volunteer studies conducted using boxers in Europe have recorded head angular acceleration levels exceeding 13,600 rad/sec/sec.¹⁰⁴ Further research is needed to establish tolerance criteria for all ages.

Recognizing the need to account for both material and geometric variations, as an initial step injury assessment reference values for the 6-month-old infant dummy were obtained by combining geometric and material scaling data between the adult and pediatric groups.⁴³ Similar to the procedures of using calcaneal tendon strength to determine the scaling of the neck tensile strength (described earlier), the skull bone modulus of elasticity from literature was used to determine the modulus of elasticity ratio for the skull of the 6-month-old infant dummy.⁴² The mechanical properties of the human skull bone between the newborn and the 6-year-old child were approximated by a linear fit. Using the principles of scaling discussed in the earlier section and detailed in the Appendix, injury assessment reference values developed for the adult midsize male (Hybrid III) and 6-year-old and 3-year-old dummies were scaled to estimate the corresponding values for the 6-month-old infant dummy. The resulting values for the HIC and head acceleration are shown in Figs. 21.23 and 21.24. The HIC and head acceleration values directly obtained from tests using the 6-month-old

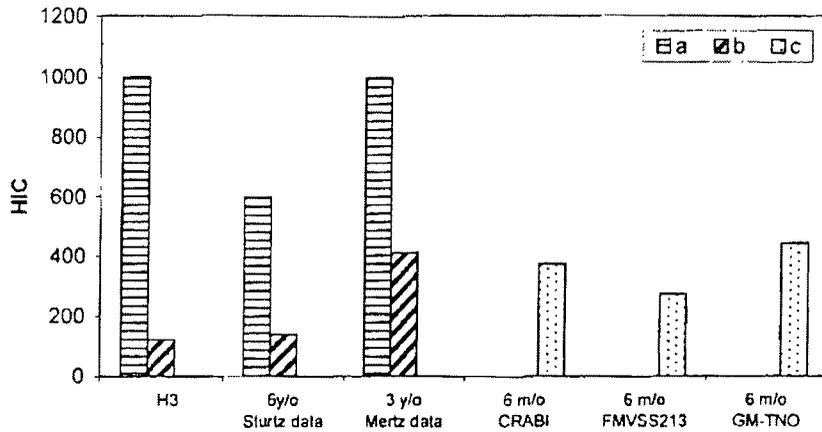


FIGURE 21.23. Estimated head injury criteria for the 6-month-old dummy. (a) Corresponds to values for that particular dummy (example Hybrid III = 1,000). (b) Scaled values applicable to the 6-month-old dummy [example, head injury criterion (HIC) of 1,000 scaled from Hybrid III results in a HIC of 121 for the 6-month-old dummy]. (c) Values directly obtained from tests using the 6-month-old dummy. (Data adapted from CFR,¹ Melvin,⁸³ Mertz et al,⁸⁵ Mertz and Weber,⁸⁶ and Sturtz.¹²⁰)

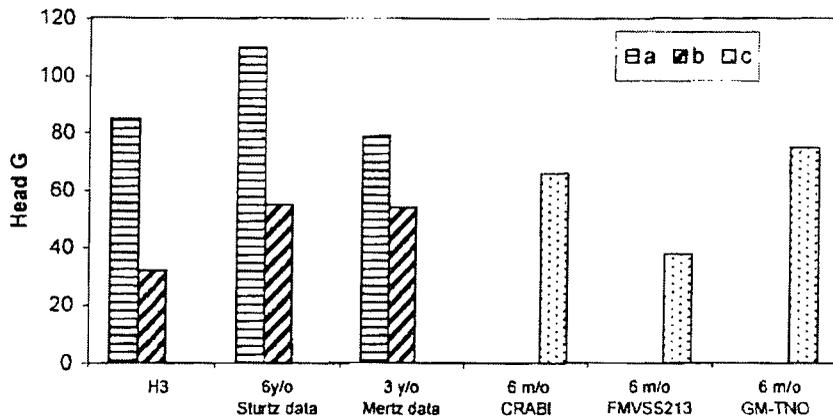


FIGURE 21.24. Head acceleration for the 6-month-old dummy. Data are presented in a manner similar to that in Fig. 21.23 (Data adapted from CFR,¹ Melvin,⁸³ Mertz et al,⁸⁵ Mertz and Weber,⁸⁶ and Sturtz.¹²⁰)

infant dummy (CRABI) in the Federal Motor Vehicle Safety Standard (FMVSS) 213 sled tests with standard bench seat, and Inland Fisher Guide sled tests with instrument panels using the CRABI 6-month-old and GMTNO 6-month-old dummies, are included in these figures. Since the head acceleration level of 32g obtained by scaling from the midsize male

dummy was significantly lower than the head accelerations estimated from the scaling of the other dummies, it was decided to exclude these data. The head acceleration values scaled from the 3- (54g) and 6-year-old (55g) dummies were found to be closer and lie between the values obtained with the FMVSS 213 (38g) and Inland Fisher Guide (66g) sled tests.⁸³ The average of

these four test values resulted in the recommendation of an injury assessment reference value of 54g for head acceleration. For the HIC, it was decided to exclude the scaled Hybrid III (HIC = 121) and scaled 6-year old dummy values (HIC = 138) because they were considerably different than the value obtained from the other tests (Fig. 21.23).

Based on these types of analyses, injury assessment reference values were developed for the CRABI 6-month-old dummy with a deploying passenger airbag.⁶³ The recommended values are peak resultant head acceleration of 50g associated with a 22-msec HIC of 390; peak resultant chest acceleration of 50g; and peak upper neck tension and shear forces, and flexion and extension moments of 500 and 470 N, and 16.4 and 5 Nm, respectively. It was stated that the 390 value for HIC should not be permitted for shorter time duration pulses with values above 50g. Using the skull bone modulus as a governing parameter for material scaling, HIC values of 121, 275, and 525 were obtained for the 12-month-old, 3-year-old, and 6-year-old dummies, respectively.⁶² Another proposed method is to compute the HIC for the pediatric group based on the assumption that the pediatric skull deformation is controlled by the properties of the cranial sutures instead of skull bones. Using the calcaneal tendon as a surrogate for suture stiffness, HIC limits for the 12-month-old, 3-year-old, and 6-year-old dummies are 660, 900, and 1,000, respectively (see Table 21.12 in the Appendix, which provides tolerance-related data for the various child dummies).

Although efforts have been advanced using mathematical and other physical models to simulate brain injury mechanisms, to date no widely accepted specific tolerance criteria exist as a function of age.^{41,42} In a recent unpublished study, a series of simulations was performed to investigate the importance of skull maturation process on the impact response of pediatric heads.⁶⁴ A simple adult head model was used along with three different 3-year-old pediatric models. The first pediatric model was obtained by simple geometric scaling of the adult model. The second model included more appropriate skull stiffness properties, i.e., geometric and

material scaling. The third model included unfused sutures, i.e., geometric and material scaling with open sutures. The simulations consisted of each head model impacting a 45-degree inclined plane with a forward initial velocity of 7.5 m/sec. Compared to the adult skull response, the geometrically scaled 3-year-old model indicated a higher acceleration peak occurring at an earlier time. The level of effective stress within the brain did not change significantly. In the geometrically and materially scaled model, the acceleration peak dropped below the adult level, while the stress in the brain increased. Addition of sutures did not change the peak deceleration value, although the time of occurrence of the peak was delayed. The effective stress (within the brain) in this model increased by approximately 10% compared to the pediatric model with geometric and material scaling (Fig. 21.25). These results underscore the need to experimentally obtain the mechanical properties of the various components of the human head as a function of age. In addition, although preliminary in nature, these analyses demonstrate the limitations of using simple geometric scaling for predicting head injury in the pediatric population.

Thorax (Chest)

Pediatric thoracic structures also undergo a process of developmental changes with increasing age. Skeletal components such as thoracic and lumbar spine vertebrae develop similarly to typical cervical vertebrae. The rib cage ossifies from cartilaginous origin. The size and properties of the internal organs and soft tissues also develop to reach adult maturity. An in-depth presentation of these developments as a function of age is not given because of space limitations. The reader is referred to the literature. With regard to the tolerance of pediatric human thoracic injury, using the principles of geometric and material scaling (see earlier discussion), chest acceleration levels for pediatric dummies were derived.⁶³ Figure 21.26 includes a bar chart representation of the various values. More recently, a Combined Thoracic Index

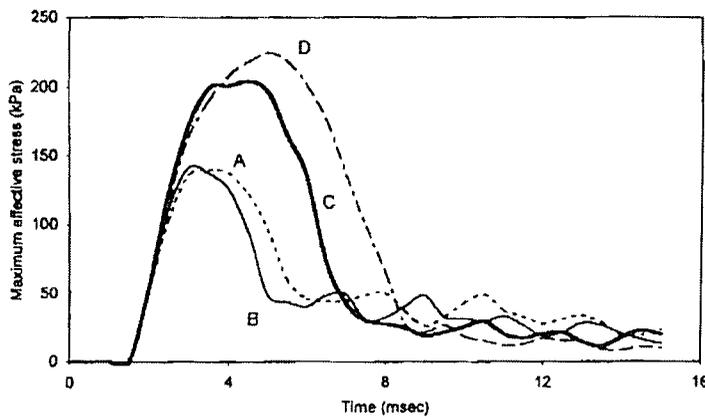
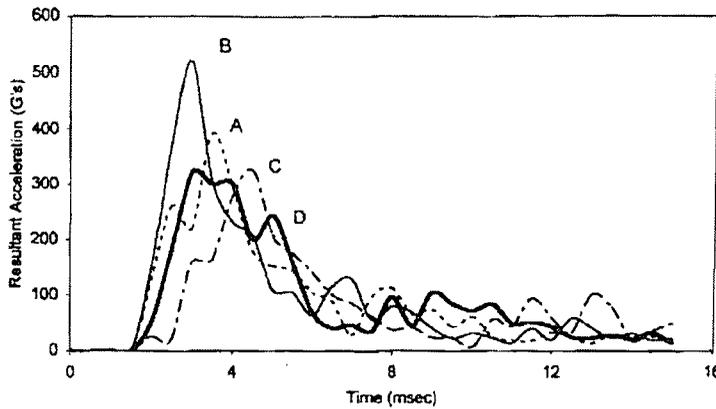


FIGURE 21.25. Comparative responses of adult and 3-year-old heads with varying assumptions used in the mathematical model. (A) Adult skull. (B) three-year-old skull with geometric scaling. (C) Three-year-old skull with geometric and material scaling. (D) three-year-old skull with geometric and material scaling and open suture simulation.

(CTI) criterion was suggested. The criterion was derived from 71 highly instrumented adult human cadaver tests conducted under varying belt and airbag combinations and at various ΔV .^{30,59,91,153,158} Detailed statistical analysis of data, i.e., deflections from chest bands (contours of chest deflection as a function of time and location), accelerations from accelerometers placed on the thoracic spinous processes, and velocities and viscous criteria computed from chest deflections, were conducted by dividing the injury pathology into AIS < 3 (no

injury) and AIS ≥ 3 (injury) groups. In the final analysis, the criterion was based on chest deflection and spine acceleration, the two measurements routinely made with the Hybrid III dummy in crash tests.¹ It is given by the relation:

$$CTI = A_{max} / A_{int} + D_{max} / D_{int}$$

where A_{max} and D_{max} denote the maximum chest/spine acceleration and chest deflection, and A_{int} and D_{int} denote the corresponding maximum allowable intercept values. Adult-

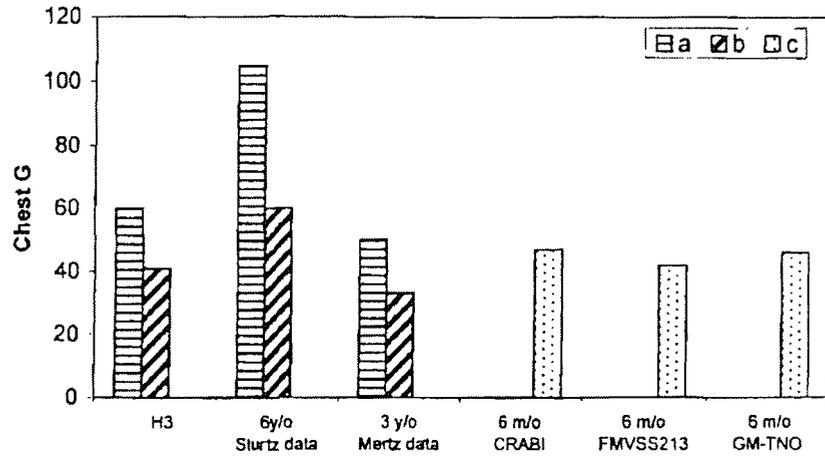


FIGURE 21.26. Estimated chest acceleration values for the 6-month-old dummy. Data were presented in a manner similar to that in Fig. 21.24. (Data adapted from CFR,¹ Melvin,⁸³ Mertz et al,⁸⁵ Mertz and Weber,⁸⁶ and Sturtz.¹²⁰)

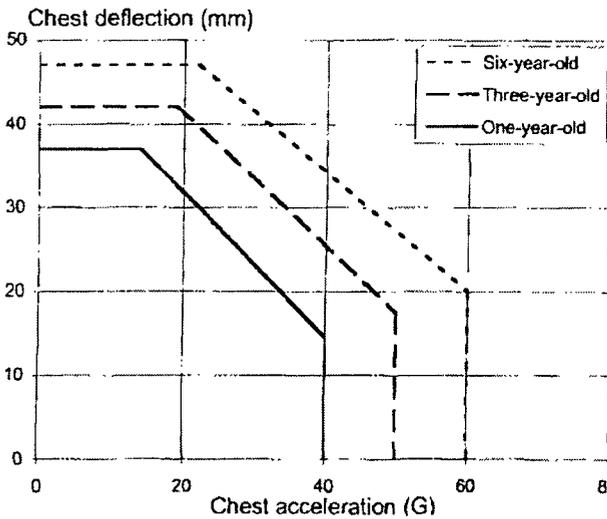


FIGURE 21.27. Proposed chest trauma index for various dummies.

to-pediatric scaling was accomplished as follows. Deflection was scaled according to chest depth, and acceleration was scaled according to geometric and material scaling. The critical intercept and limiting values used in the computation of CTI are given (Fig. 21.27, Table 21.11). As indicated earlier, it must be noted that these proposed intercept values and limits are only tentative. Consequently,

TABLE 21.11. Critical intercept and limiting values for Combined Thoracic Index (CTI).

Parameter	6-year-old	3-year-old	12-month-old
D_{int} (mm)	63	57	49
A_{int} (G)	85	70	55
D_{lim}	47	42	37
A_{lim}	60	50	40

Adapted from Kleinberger et al.⁸²

revised numbers may be available in the near future.

Extremities

Like the head and spinal column, extremities develop with advancing age. Because of space limitations, it is not possible to describe the developmental processes. The reader is referred to the literature. As an example, the femoral shaft is chosen for demonstrating the effects of age on biomechanical properties. The femur responds with increasing strength from birth to

adulthood. Tests have revealed that children have a low modulus of elasticity, lower ash content, and absorb more energy than the adult femur.^{31,135} Variation of femur bending strength with age is depicted in Fig. 21.5. With regard to the shear strength tolerance of the femur, isolated femur testing from child cadavers ranging from 5 days to 15 years has been conducted.²⁴ In younger children, the perichondrial complex is shown to be a major contributor to the shear strength of the perichondrial complex-epiphyseal plate combination (Fig. 21.28). Established tolerance criteria for the femur as a function of age are not available.

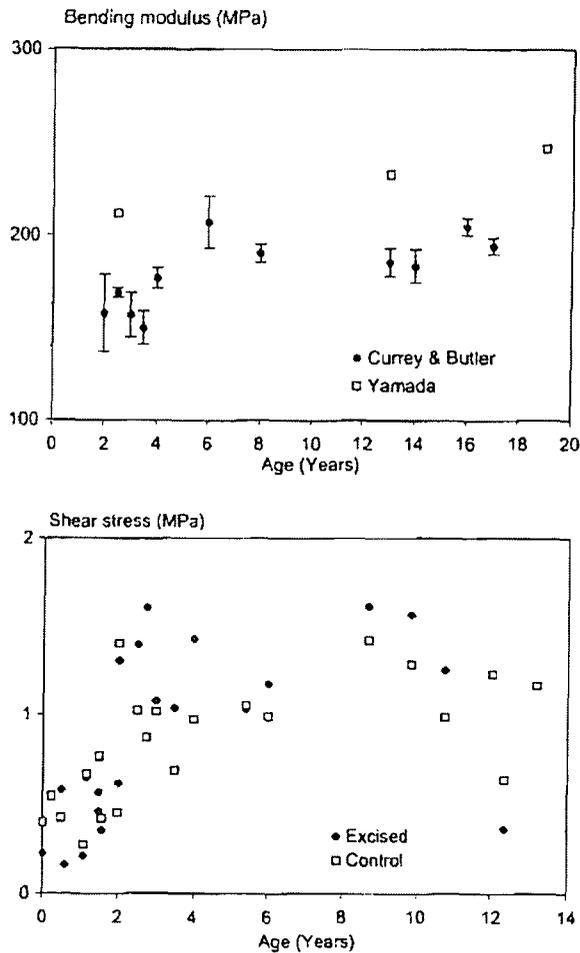


FIGURE 21.28. Variation of femur strength as a function of age. (Data adapted from Currey and Butler³¹ and Yamada.¹³⁵)

Summary

The pediatric structural components of the human body develop and attain maturity through various processes. This chapter focused on the neck, head, thorax, and extremity structures. Emphasis was placed on neck and head structures. While not exhaustive, developmental processes such as ossification were described. Detailed material properties for the various components of these structures were given as a function of age so that modeling efforts can be advanced to understand pediatric biomechanics. Since experimental age-specific data are very limited, and since stress analysis-type mathematical models offer unique capabilities to investigate the biomechanical issues, these data will be of value. If and when experimental output becomes available, it will be possible to update and/or validate age-specific models using these data. Attention must be paid in future research to obtaining experimental data (e.g., material properties) of pediatric structures. Although tolerance criteria are presented for other structures, because of space constraints such detailed coverage was not given to the thorax and extremity regions in this chapter. These data should act as a stimulus to further our pediatric biomechanical knowledge.

Acknowledgments. This study was supported in part by Department of Transportation (DOT) National Highway Traffic Safety Administration (NHTSA) grant DTNH22-93-Y-17028 and the Department of Veterans Affairs Medical Research.

Appendix

The type of scaling commonly used in vehicular applications is dimensional analysis. For mechanical systems in which thermal and electrical effects can be ignored, this technique allows the unknown physical responses of a given system to be estimated from the known responses of a similar system. This is accom-

plished by establishing three fundamental scaling factors. They are based on ratios between the fundamental properties that characterize the two systems. For structural analysis, the three fundamental ratios are length, mass density, and modulus of elasticity or stiffness. The scaling ratios for other variables are based on the fundamental ratios. The three fundamental nondimensional ratios⁶³ are:

$$\text{Length scale ratio: } \lambda_L = L_1/L_2 \quad (1)$$

$$\text{Mass density ratio: } \lambda_D = \rho_1/\rho_2 \quad (2)$$

$$\text{Modulus of elasticity ratio: } \lambda_E = E_1/E_2 \quad (3)$$

Subscripts 2 and 1 refer to the subjects to be scaled to (prototype) and from (model), respectively. The scale factors for all other physical quantities associated with the impact response of the system can be obtained from these three dimensionless ratios.^{63,65}

When scaling data between adult subjects, it is generally assumed that the moduli of elasticity and mass densities are equal for both subjects. The scale factors for these quantities are equal to unity. The implication of this assumption is that all physical quantities can be scaled as functions of the basic length scale ratio, λ_L , assuming geometric similitude. When scaling data from adults to children, or between children of various ages, differences in the modulus of elasticity (or stiffness) must be considered to account for the structural immaturity in children. Assuming the mass density to be constant for all subjects ($\lambda_D = 1$), the following scale factors are obtained:

$$\text{Length: } \lambda_L = L_1/L_2 \quad (4)$$

$$\text{Mass: } \lambda_m = (\lambda_L)^3 \quad (5)$$

$$\text{Modulus of elasticity: } \lambda_E = E_1/E_2 \quad (6)$$

$$\text{Time: } \lambda_T = \lambda_L/(\lambda_E)^{0.5} \quad (7)$$

$$\text{Acceleration: } \lambda_A = \lambda_E/\lambda_L \quad (8)$$

$$\text{Force: } \lambda_F = (\lambda_L)^2 \lambda_E \quad (9)$$

$$\text{Moment: } \lambda_M = (\lambda_L)^3 \lambda_E \quad (10)$$

$$\text{Head injury criteria: } \lambda_{HIC} = (\lambda_E)^2/(\lambda_L)^{1.5} \quad (11)$$

TABLE 21.12. Scale factors.

	6-year-old	3-year-old	12-month-old	6-month-old	Newborn
Head length	0.89	0.87	0.82	0.77	0.63
Neck length	0.66	0.62	0.59	0.57	—
Chest length	0.59	0.53	0.49	0.42	0.33
Head mass	0.72	0.66	0.55	0.46	—
Bone modulus	0.67	0.47	0.32	0.29	0.24
Tendon strength	0.96	0.85	0.70	0.66	0.63
Head					
HIC GS	1.26	1.32	1.49	1.69	2.52
GB	0.54	0.27	0.14	0.12	0.11
GT	1.10	0.89	0.66	0.65	0.79
Angular acceleration					
MS	1.23	1.32	1.49	1.68	—
GB	0.85	0.62	0.48	0.49	0.61
GT	1.21	1.12	1.04	1.11	1.59
Neck tension—GT	0.41	0.32	0.24	0.21	0.13
Chest acceleration	1.19	0.93	0.65	0.68	0.74

GS, geometric scaling only.

MS, mass scaling only.

GB, geometric scaling with material scaling based on bone modulus.

GT, geometric scaling with material scaling based on calcaneal tendon modulus.

Midsize adult values: head circumference 57.1 cm, neck circumference 38.3 cm, head weight 4.55 kg, chest circumference 101.0 cm, HIC = 1,000, head angular acceleration 1,700 rad/sec/sec, neck tension 3,300 N, chest acceleration 60 g.

Adapted from CFR,¹ Kleinberger et al.,² Melvin,³ and Ommaya et al.^{4a}

$$\text{Angular velocity: } \lambda_{\omega} = 1/\lambda_T = \lambda_E^{0.5}/\lambda_L \quad (12)$$

$$\text{Angular acceleration: } \lambda_{\alpha} = \lambda_E/(\lambda_m)^2 = \lambda_E/\lambda_L^2 \quad (13)$$

When applying the different scale factors to anthropomorphic test devices (dummies), it is necessary to determine whether or not the material scale factor has been incorporated into the design of the dummies. Table 21.12 includes the fundamental length scale factors for the head, neck, and chest, material property scale factors based on the skull bone elastic modulus and calcaneal tendon strength, and the resulting neck tension, HIC, head and chest acceleration scale factors using the above relations.^{63,83}

References

- Code of Federal Regulations, 49 CFR 400-999, pp. 1111, 1995.
- Agur AMR, Lee ML. *Grant's atlas of anatomy*, 9th ed. Williams & Wilkins, Baltimore, 1991.
- AIS. *The Abbreviated Injury Scale*. American Association for Automotive Medicine, Arlington Heights, IL, 1990.
- Allen BL Jr, Ferguson RL. Cervical spine trauma in children. In: Bradford D, Hensinger R, eds. *The pediatric spine*. Thieme, New York, pp. 89-104, 1985.
- Arbogast KB, Thibault KL, Margulies SS. A high-frequency shear device for testing soft biological tissues. *J Biomech* 1997;30(7):757-759.
- Backaitis SH, ed. *Biomechanics of impact injury and injury tolerances of the head-neck complex*, vol PT43. Society of Automotive Engineers, Warrendale, PA, 1993.
- Backaitis SH. *Biomechanics of impact injury and injury tolerances of the thorax-shoulder complex*, vol PT-45. Society of Automotive Engineers, Warrendale, PA, 1995.
- Backaitis SH, Mertz HJ, eds. *Hybrid III: the first human-like crash test dummy*, vol PT44. Society of Automotive Engineers, Warrendale, PA, 1994.

9. Bailey D. Normal cervical spine in infants and children. *Radiology* 1952;59:712-719.
10. Begeman P, Aekbote K, Levine R, King A. Human ankle response in internal and external rotation. Proceedings of the 4th Annual Injury Prevention Through Biomechanics Symposium, Detroit, MI, May 1994.
11. Begeman PC, Aekbote K. Axial load strength and some ligament properties of the ankle joint. Proceedings of the 6th Injury Prevention Through Biomechanics Symposium, Detroit, MI, May 1996.
12. Behrman RE, Vaughan VC III. Developmental pediatrics: growth and development. In: Nelson W, ed. *Nelson textbook of pediatrics*, 13th ed. WB Saunders, Philadelphia, pp. 6-35, 1987.
13. Bick E, Copel J. Longitudinal growth of the human vertebra: contribution to human osteogeny. *J Bone Joint Surg* 1950;32A(4):803-814.
14. Bick EM. Vertebral growth: its relation to spinal abnormalities in children. *Clin Orthop* 1961;21:43-48.
15. Bonadio WA. Cervical spine trauma in children: part I: general concepts, normal anatomy, radiographic evaluation. *Am J Emerg Med* 1993;11(2):158-165.
16. Bonadio WA. Cervical spine trauma in children: part II: mechanisms and manifestations of injury, therapeutic considerations. *Am J Emerg Med* 1993;11(3):256-278.
17. Boreadis AG, Gershon-Cohen J. Luschka joints of cervical spine. *Radiology* 1956;66:181-187.
18. Carpenter EB. Normal and abnormal growth of the spine. *Clin Orthop* 1961;21(1):49-55.
19. Cattell H, Filtzer D. Pseudosubluxation and other normal variations in cervical spine of children. *J Bone Joint Surg* 1965;47A(7):1295-1309.
20. Cavanaugh JM, Nyquist GW, Goldberg SJ, King AI. Lower abdominal tolerance and response. Proceedings of the 30th Stapp Car Crash Conference, San Diego, CA, October 1986.
21. Cavanaugh JM, Walilko T, Walbridge A, Huang Y, King AI. An evaluation of TTI and ASA in SID side impact sled tests. Proceedings of the 38th Stapp Car Crash Conference, Fort Lauderdale, FL, October 1994.
22. Cavanaugh JM, Walilko TJ, Malhotra A, Zhu Y, King AI. Biomechanical response and injury tolerance of the pelvis in twelve sled side impacts. Proceedings of the 34th Stapp Car Crash Conference, Orlando, FL, November 1990.
23. Chandraraj S, Briggs C. Multiple growth cartilages in the neural arch. *Anat Rec* 1991;230:114-120.
24. Chung M, Batterman S, Brighton C. Shear strength of human femoral capital epiphyseal plate. *J Bone Joint Surg* 1976;58A(1):94-103.
25. Clark CR, Ducker TB, Dvorak J, et al. *The cervical spine*, 3rd ed. Lippincott-Raven, Philadelphia, 1998.
26. Compere E, Tachdjian M, Kernahan W. Luschka joints: their anatomy, physiology and pathology. *Orthopaedics* 1959;1:159-168.
27. Coursin DB. Malnutrition, brain development, and behavior: anatomic, biochemical, and electrophysiologic constructs. In: Brazier MA, ed. *Growth and development of the brain*. Raven Press, New York, 1975.
28. Coventry M, Ghormley R, Kernohan J. Intervertebral disc: its microscopic anatomy and pathology. Part I. Anatomy, development, and physiology. *J Bone Joint Surg* 1945;27(1):105-112.
29. Coventry M, Ghormley R, Kernohan J. Intervertebral disc: its microscopic anatomy and pathology. Part II. Changes in the intervertebral disc concomitant with age. *J Bone Joint Surg* 1945;27(2):233-247.
30. Crandall JR, Pilkey WD, Klopp GS, et al. A comparison of two and three point belt restraint systems. Advances in Occupant Restraint Technologies: Joint AAAM-IRCOBI Special Session, Lyon, France, 1994.
31. Currey JD, Butler G. Mechanical properties of bone tissue in children. *J Bone Joint Surg* 1975;57A(6):810-814.
32. Dickerson J, Dobbing J. Prenatal and postnatal growth and development of the central nervous system of the pig. *Proc R Soc Lond* 1966;166B:384-395.
33. Dobbing J, Sands J. Quantitative growth and development of human brain. *Arch Dis Child* 1973;48:757-767.
34. Duhaime AC, Gennarelli TA, Thibault LE, Bruce DA, Margulies SS, Wiser R. The shaken baby syndrome—a clinical pathological, and biomechanical study. *J Neurosurg* 1987;66:409-415.
35. Duncan J. Laboratory note: on tensile strength of fresh adult foetus. *Br Med J* 1874;2:763-764.
36. Eppinger RH. Prediction of thoracic injury using measurable experimental parameters. Sixth International Conference on Experimental Safety Vehicles, Washington, DC, 1976.

37. Eppinger RH, Augustyn K, Robbins DH. Development of a promising universal thoracic trauma prediction methodology. Proceedings of the 22nd Stapp Car Crash Conference, Ann Arbor, MI, October 1978.
38. Evans D, Bethem D. Cervical spine injuries in children. *J Pediatr Orthop* 1985;9:563-568.
39. Fitzgerald E, Freeland A. Viscoelastic response of intervertebral discs at audio-frequencies. *Med Biol Eng* 1970;9:459-478.
40. Ford D, McFadden K, Bagnall K. Sequence of ossification in human vertebral neural arch centers. *Anat Rec* 1982;203:175-178.
41. Gennarelli TA. The spectrum of traumatic axonal injury. *Neuropathol Appl Neurobiol* 1996;22:509-513.
42. Gennarelli TA, Meaney DF. Mechanisms of primary head injury. In: Wilkins R, Rengachary S, eds. *Neurosurgery*, vol 2, 2nd ed. McGraw Hill, New York, pp. 2611-2621, 1996.
43. Gilsanz V, Gibbens D, Roe T, et al. Vertebral bone density in children: effect of puberty. *Radiology* 1988;166(3):847-850.
44. Gooding C, Neuhauser E. Growth and development of vertebral body in presence and absence of normal stress. *AJR* 1965;93:388-394.
45. Gurdjian ES. *Impact Head Injury, Mechanistic, Clinical and Preventive Correlations*. Charles C Thomas, Springfield, IL, 1975.
46. Gurdjian ES, Lissner HR, Evans FG, Patrick LM, Hardy WG. Intracranial pressure and acceleration accompanying head impacts in human cadavers. *Surg Gynecol Obstet* 1961;113:185-190.
47. Haas SL. Growth in length of vertebrae. *Arch Surg* 1939;38:245-249.
48. Hadley M, Zabramski J, Browner C, ReKate H, Sonntag V. Pediatric spinal trauma. Review of 122 cases of spinal cord and vertebral column injuries. *J Neurosurg* 1988;68:18-24.
49. Hall MC. *Luschka's joint*. Springfield, IL: Charles C Thomas, 1965.
50. Hallen A. Collagen and ground substance of human intervertebral disc at different ages. *Acta Chem Scand* 1962;16(3):705-710.
51. Hayashi K, Yabuki T. Origin of the uncus and of Luschka's joint in the cervical spine. *J Bone Joint Surg* 1985;67A(5):788-791.
52. Hayes W, Bodine A. Flow-independent viscoelastic properties of articular cartilage matrix. *J Biomech* 1978;11:407-419.
53. Hinck V, Hopkins C, Savara B. Sagittal diameter of the cervical spinal canal in children. *Radiology* 1962;79:97-108.
54. Hirsch C, Schajowicz F, Galante J. Structural changes in the cervical spine: a study on autopsy specimens in different age groups. *Acta Orthop Scand* 1967;S109.
55. Hodgson VR, Thomas LM. *Breaking strength of the human skull vs. impact surface curvature*. Springfield, VA: US Department of Transportation, 1971.
56. Hubbard RP. Flexure of layered cranial bone. *J Biomech* 1971;4:251-263.
57. Jaslow CR. Mechanical properties of cranial sutures. *J Biomech* 1990;23(4):313-321.
58. Kallieris D, Stein KM, Mattern R, Morgan R, Eppinger RH. The performance of active and passive driver restraint systems in simulated frontal collisions. Proceedings of the 38th Stapp Car Crash Conference, Ft. Lauderdale, FL, October 1994.
59. Kasai T. Growth of neck muscle in normal children at MR imaging. Personal Communication, 1998.
60. Kasai T, Ikata T, Katoh S, Miyake R, Tsubo M. Growth of cervical spine with special reference to its lordosis and mobility. *Spine* 1996;21(18):2067-2073.
61. Kleinberger M. Personal communication, 1999.
62. Kleinberger M, Sun E, Eppinger R, Kuppa S, Saul R. *Development of improved injury criteria for the assessment of advanced automotive restraint systems*. Washington, DC: NHTSA, 1998.
63. Kleinberger M, Yoganandan N, Kumaresan S. Biomechanical considerations for child occupant protection. 42nd AAAM Conference, Charlottesville, VA, October 1998.
64. Klopp G, Crandall J, Hall G, Pilkey W, Hurwitz S, Kuppa S. Mechanisms of injury and injury criteria for the human foot and ankle in dynamic axial impacts to the foot. IRCOBI Conference, Hanover, Germany, September 1997.
65. Knutsson F. Growth and differentiation of postnatal vertebra. *Acta Radiol* 1961;55:401-408.
66. Kriewall TJ. Structural, mechanical, and material properties of fetal cranial bone. *Am J Obstet Gynecol* 1982;143:707-714.
67. Kriewall TJ, McPherson GK, Tsai A. Bending properties and ash content of fetal cranial bone. *J Biomech* 1981;14:73-79.
68. Kumaresan S, Yoganandan N, Pintar F. Age-specific pediatric cervical spine biomechanical

- responses: three-dimensional nonlinear finite element models. Proceedings of the 41st Stapp Car Crash Conference, Orlando, FL, November 1997.
69. Kumaresan S, Yoganandan N, Pintar FA. Adult and pediatric human cervical spine finite element analyses. *ASME Adv Bioeng* 1997;35(BED):515-516.
 70. Kumaresan S, Yoganandan N, Pintar FA. Biomechanical responses of pediatric cervical spine using nonlinear finite element approach. *ASME Adv Bioeng* 1999;42(BED):143-144.
 71. Kumaresan S, Yoganandan N, Pintar FA. Finite element modeling approaches of human cervical spine facet joint capsule. *J Biomech* 1998;31:371-376.
 72. Kumaresan S, Yoganandan N, Pintar FA. Methodology to quantify the uncovertebral joint in the human cervical spine. *J Musculoskeletal Res* 1997;1(2):1-9.
 73. Kumaresan S, Yoganandan N, Pintar FA. Pediatric neck modeling using finite element analysis. *Int J Crashworthiness* 1997;2(4):367-377.
 74. Kumaresan S, Yoganandan N, Pintar FA. Summary of pediatric biomechanical responses. *ASME ADV Bioeng* 1999;42(BED):765-766.
 75. Maiman DJ, Yoganandan N. Biomechanics of cervical spine trauma. In: Black P, ed. *Clinical neurosurgery*, vol 37. Williams & Wilkins, Baltimore, pp. 543-570, 1991.
 76. Markuske H. Sagittal diameter measurements of bony cervical spinal canal in children. *Pediatr Radiol* 1977;6:129-131.
 77. McElhaney JH, Fogle JL, Melvin JW, Haynes RR, Roberts VL, Alem NM. Mechanical properties of cranial bone. *J Biomech* 1970;3:495-512.
 78. McElhaney JH, Roberts VL, Hilyard JF, eds. *Handbook of human tolerance*. Japan Automobile Research Institute, Tokyo, 1976.
 79. McElhaney JH, Stalnaker RL, Roberts VL. Biomechanical aspects of head injury. In: King WF, Mertz HJ, eds. *Human impact response*. Plenum Press, New York, pp. 85-112, 1973.
 80. McElhaney JH, Stalnaker RL, Estes MS. Dynamic mechanical properties of scalp and brain. Proceedings of the 6th Rocky Mountain Bioengineering symposium, Denver, Co, April 1970.
 81. McGowan D, Voo L, Liu Y. Distraction failure of immature spine. *ASME Adv Bioeng* 1993;24(BED):24-25.
 82. McPherson G, Kriewall T. Elastic modulus of fetal cranial bone: first step toward an understanding of biomechanics of fetal head molding. *J Biomech* 1979;13(1):9-16.
 83. Melvin JW. Injury assessment reference values for the CRABI 6-month infant dummy in a rear-facing infant restraint with airbag deployment. SAE Congress and Exposition, Detroit, MI, February 1995.
 84. Melvin JW, Evans FG. A strain energy approach to the mechanics of skull fracture. Proceedings of the 15th Stapp Car Crash Conference, Coronado, CA, November 1971.
 85. Mertz H, Driscoll G, Lenox J, Nyquist G, Weber D. Responses of animals exposed to deployment of various passenger inflatable restraint system concepts for a variety of collision severities and animal positions. 9th International Technical Conference on Experimental Safety Vehicles, Kyoto, Japan, 1982.
 86. Mertz HJ. The kinematics and kinetics of whiplash. Ph.D. Dissertation, Wayne State University, 1967.
 87. Mertz HJ, Patrick LM. Strength and response of the human neck. Proceedings of the 15th Stapp Car Crash Conference, Coronado, CA, November 1971.
 88. Mertz HJ, Weber DA. Interpretations of the impact responses of a 3-year-old child dummy relative to child injury potential. 9th International Technical Conference on Experimental Safety Vehicles. Kyoto, Japan, 1982.
 89. Messerer O. *Über Elasticität und Festigkeit der Menschlichen Knochen*. Stuttgart, Germany, 1880.
 90. Metz HJ, McElhaney JH, Ommaya A. A comparison of the elasticity of live, dead, and fixed brain tissue. *J Biomech* 1970;3(4):453-458.
 91. Morgan RM, Eppinger RH, Haffner MP, Yoganandan N, Pintar FA, Sances A Jr. Thoracic trauma assessment formulations for restrained drivers in frontal impact. Proceedings of the 38th Stapp Car Crash Conference, Ft. Lauderdale, FL, October 1994.
 92. Morgan RM, Eppinger RH, Hennesey BC. Ankle joint injury mechanism for adults in frontal automotive impact. Proceedings of the 35th Stapp Car Crash Conf, San Diego, CA, November 1991.
 93. Morgan RM, Marcus JH, Eppinger RH. Side impact—the biofidelity of NHTSA's proposed ATD and efficacy of TTI. Proceedings of the

- 30th Stapp Car Crash Conf, San Diego, CA, October 1986.
94. Myklebust JB, Pintar FA, Yoganandan N, et al. Tensile strength of spinal ligaments. *Spine* 1988; 13(5):526-531.
95. Nahum AM, Melvin JW, eds. *Accidental injury: biomechanics and prevention*. Springer-Verlag, New York, 1993.
96. Nightingale RW, McElhane JH, Camacho DL, Kleinberger M, Winkelstein BA, Myers BA. The dynamic responses of the cervical spine: buckling, end conditions, and tolerance in compressive impacts. Proceedings of the 41st Stapp Car Crash Conference, Orlando, FL, November 1997.
97. O'Rahilly R, Benson D. Development of vertebral column. In: Bradford D, Hensinger R, eds. *The pediatric spine*. Thieme, New York, pp. 3-17, 1985.
98. Oda J, Tanaka H, Tsuzuki N. Intervertebral disc changes with aging of human cervical vertebra—from neonate to 80s. *Spine* 1988; 13(11):1205-1211.
99. Ogden J, Ganey T, Sasse J, Neame P, Hilbelink D. Development and maturation of the axial skeleton. In: Weinstein S, ed. *The pediatric spine: principles and practice*. Raven Press, New York, pp. 1959, 1994.
100. Ogden JA, Grogan DP, Light TR. Postnatal development and growth of musculoskeletal system. In: Albright J, Brand R, eds. *The scientific basis of orthopaedics*. Appleton & Lange, Norwalk, CT, pp. 526, 1987.
101. Ommaya A, Yarnell P, Hirsch A, Harris E. Scaling of experimental data on cerebral concussion in sub-human primates to concussion threshold in man. 10th Stapp Car Crash Conference, November 1967.
102. Ommaya AK. Mechanical properties of tissues of the nervous system. *J Biomech* 1968;1:127-138.
103. Peacock A. Observations on postnatal structure of intervertebral disc in man. *J Anat* 1956;86(part 2):162-179.
104. Pincemaille Y, Trosselle X, Mack P, Tarriere C, Breton F, Renault B. Some new data related to human tolerance obtained from volunteer boxers. Proceedings of the 33rd Stapp Car Crash Conference, Washington, DC, October 1989.
105. Pintar F, Yoganandan N, Eppinger RH. Response and tolerance of the human forearm to impact loading. Proceedings of the 42nd Stapp Car Crash Conference, Tempe, AZ, November 1998.
106. Pintar FA, Schlick MB, Yoganandan N, Maiman DJ. Instrumented artificial spinal cord for human cervical pressure measurement. *Bio Med Mater Eng* 1996;6(2):219-229.
107. Pintar FA, Yoganandan N. Head-neck tension biomechanical models for pediatric and small female populations. 43rd AAAM Conference, Barcelona, Spain, in press.
108. Pintar FA, Yoganandan N, Voo L. Effect of age and loading rate on human cervical spine injury threshold. *Spine* 1998;23(18):1957-1962.
109. Pintar FA, Yoganandan N, Voo LM, Cusick JF, Maiman DJ, Sances A Jr. Dynamic characteristics of the human cervical spine. *SAE Trans* 1995;104(6):3087-3094.
110. Prasad P, Daniel R. Biomechanical analysis of head, neck, and torso injuries to child surrogates due to sudden torso acceleration. Proceedings of the 28th Stapp Car Crash Conference, Chicago, IL, November 1984.
111. Prasad P, Mertz HJ. The position of the United States Delegation to the ISO Working Group 6 on the use of HIC in the automotive environment. Society of Automotive Engineers, Warrendale, PA, 1985.
112. Roaf R. Vertebral growth and its mechanical control. *J Bone Joint Surg* 1960;42B:40-59.
113. Rothman RH, Simeone FA. *The spine*, vol 1, 3rd ed. WB Saunders, Philadelphia, 1992.
114. Runge CF, Youssef A, Thibault KL, Kurtz SM, Magram G, Thibault LE. Material properties of human infant skull and suture: experiments and numerical analysis. 9th Injury Prevention through Biomechanics Symposium, Detroit, MI, May 1998.
115. Saito T, Yamamuro T, Shikata J, Oka M, Tsutsumi S. Analysis and prevention of spinal column deformity following cervical laminectomy I: Pathogenetic analysis of postlaminectomy deformities. *Spine* 1991;16(5):494-502.
116. Sances A Jr, Yoganandan N. Human head injury tolerance. In: Sances A Jr, Thomas DJ, Ewing CL, Larson SJ, Unterharnscheidt F, eds. *Mechanisms of head and spine trauma*. Aloray, Goshen, NY, pp. 189-218, 1986.
117. Sherk HH, Dunn EJ, Eismont FJ, et al. *The cervical spine*, 2nd ed. JB Lippincott, Philadelphia, 1989.
118. Snyder RG. Anthropometry of infants, children, and youths to age 18 for product safety design. University of Michigan, Ann Arbor, 1977.
119. Snyder RG, Foust DR, Bowman BM. Study of impact tolerance through free-fall investigation. University of Michigan, Ann Arbor, 1977.

120. Sturtz G. Biomechanical data of children. Proceedings of the 24th Stapp Car Crash Conference, Troy, MI, October 1980.
121. Swearingen JJ. Tolerances of the human face to crash impact. Office of Aviation Medicine, Federal Aviation Agency, Oklahoma City, 1965.
122. Tanner J. *Growth at adolescence*. Blackwell Scientific, Oxford, 1962.
123. Tanner J, Whitehouse R, Takaishi I. Standards from birth to maturity for height, weight, height velocity and weight velocity. *Arch Dis Child* 1966;41:454-471.
124. Taylor J. Growth of human intervertebral disc. *J Anat* 1970;107:183-184.
125. Thibault KL. Pediatric head injuries: the influence of brain and skull mechanical properties. University of Pennsylvania, 1997.
126. Thibault KL, Kurtz SM, Margulies SS. Effect of the age-dependent properties of the braincase on the response of the infant brain to impact. *AMSE Adv Bioeng* 1997;36(BED):137-138.
127. Thibault KL, Margulies SS. Age-dependent material properties of the porcine cerebrum: effect on pediatric head injury criteria. *J Biomech* 1998;31:1119-1126.
128. Tindall G, Cooper P, Barrow D. *The practice of neurosurgery*. Williams & Wilkins, Baltimore, 1996.
129. Tulsi R. Growth of human vertebral column: osteological study. *Acta Anat* 1971;79:570-580.
130. Verbout A. Development of the vertebral column. *Adv Anat Embryol Cell Biol* 1985;90: 1-100.
131. Versace J. A review of the severity index. Proceedings of the 15th Stapp Car Crash Conference, Coronado, CA, November 1971.
132. Walmsley R. Development and growth of the intervertebral disc. *Edinb Med J* 1953;60(8): 341-364.
133. Weinstein S. *Pediatric spine: principles and practice*. Raven Press, New York, 1994.
134. Williams PL. *Gray's anatomy*. Churchill Livingstone, New York, 1995.
135. Yamada H. *Strength of biological materials*. Williams & Wilkins, Baltimore, 1970.
136. Yoganandan N, Haffner M, Pintar FA. Facial injury: a review of biomechanical studies and test procedures for facial injury assessment. *J Biomech* 1996;29(7):985-986.
137. Yoganandan N, Kumaresan S, Pintar FA. Pediatric cervical spine biomechanics using finite element models. International Research Council on the Biomechanics of Impact, Goteborg (Sweden), September 1998.
138. Yoganandan N, Morgan RM, Eppinger RH, Pintar FA, Sances A Jr, Williams A. Mechanism of thoracic injury in a frontal impact. *J Biomech Eng* 1996;118:595-597.
139. Yoganandan N, Morgan RM, Eppinger RH, Pintar FA, Skrade DA, Sances A Jr. Thoracic deformation and velocity analysis in frontal impact. *J Biomech Eng* 1995;117:48-52.
140. Yoganandan N, Myklebust JB, Ray G, Sances A Jr. Mathematical and finite element analysis of spinal injuries. *CRC Rev Biomed Eng* 1987; 15(1):29-93.
141. Yoganandan N, Pintar F. Biodynamic response of the human body in frontal impact. In: Leondes C, ed. *Biomechanic systems, techniques and applications*. Gordon & Breach, Amsterdam, The Netherlands, 6-1 to 6-34, 2000.
142. Yoganandan N, Pintar FA. Biomechanics of the cranio-cervical region. In: Boeker D, ed. *Cranio-cervical junction—anatomy, physiology, therapy*. Biermann Verlag, GMBH, Köln, Germany, 2-14, 1999.
143. Yoganandan N, Pintar FA. Cervical vertebral and facet joint kinematics under whiplash. *J Biomech Eng* 1998;120:305-308.
144. Yoganandan N, Pintar FA, Boynton M, et al. Dynamic axial tolerance of the human foot-ankle complex. *SAE Trans* 1996;105:1887-1898.
145. Yoganandan N, Pintar FA, Kumaresan S, Haffner M, Kuppa S. Impact biomechanics of the human thorax-abdomen complex. *Int J Crashworthiness* 1997;2(2):219-228.
146. Yoganandan N, Pintar FA, Larson SJ, Sances A Jr, eds. *Frontiers in head and neck trauma: clinical and biomechanical*. IOS Press, Amsterdam, The Netherlands, 1998.
147. Yoganandan N, Pintar FA, Maiman DJ, Cusick JF, Sances A Jr, Walsh PR. Human head-neck biomechanics under axial tension. *Med Eng Physics* 1996;18(4):289-294.
148. Yoganandan N, Pintar FA, Reinartz J, Sances A Jr. Human facial tolerance to steering wheel impact: a biomechanical study. *J Safety Res* 1993;24(2):77-85.
149. Yoganandan N, Pintar FA, Sances A Jr. Biodynamics of steering wheel induced facial trauma. *J Safety Res* 1991;22:179-190.
150. Yoganandan N, Pintar FA, Sances A Jr, Maiman DJ. Strength and motion analysis of the human head-neck complex. *J Spinal Disord* 1991;4(1): 73-85.
151. Yoganandan N, Pintar FA, Sances A Jr, et al. Steering wheel induced facial trauma. *SAE Trans* 1989;97(4):1104-1128.

152. Yoganandan N, Pintar FA, Sances A Jr, et al. Biomechanics of skull fracture. *J Neurotrauma* 1995;12(4):659-668.
153. Yoganandan N, Pintar FA, Skrade D, Chmiel W, Reinartz JM, Sances A Jr. Thoracic biomechanics with airbag restraint. Proceedings of the 37th Stapp Car Crash Conference, San Antonio, TX, November 1993.
154. Yoganandan N, Pintar FA, Skrade D, Sances A Jr. Evaluation of thoracic trauma in frontal impact. 14th International Conference on Enhanced Safety of Vehicles, Munich, Germany, May 1994.
155. Yoganandan N, Pintar FA, Wilson CR, Sances A Jr. In vitro biomechanical study of female geriatric cervical vertebral bodies. *J Biomed Eng* 1990;12(2):97-101.
156. Yoganandan N, Sances A Jr, Maiman DJ, Myklebust JB, Pech P, Larson SJ. Experimental spinal injuries with vertical impact. *Spine* 1986;11(9):855-860.
157. Yoganandan N, Sances A Jr, Pintar FA, et al. Traumatic facial injuries with steering wheel loading. *J Trauma* 1991;31(5):699-710.
158. Yoganandan N, Skrade D, Pintar F, Reinartz J, Sances A Jr. Thoracic deformation contours in a frontal impact. Proceedings of the 35th Stapp Car Crash Conference, San Diego, CA, November 1991.
159. Yoganandan N, Kumaresan S, Pintar FA. Factors for pediatric scaling based on experimental data from spine structures. (Unpublished.)
160. Youman J. *Neurological surgery*. WB Saunders, Philadelphia, 1996.
161. Yousefzadeh D, El-Khoury G, Smith W. Normal sagittal diameter and variation in pediatric cervical spine. *Pediatr Radiol* 1982;14(2):319-325.



Injury biomechanics for aiding in the diagnosis of abusive head trauma

Mary Clyde Pierce, MD^{a,b,*}, Gina E. Bertocci, PhD, PE^c,
Rachel Berger, MD, MPH^{a,b}, Ev Vogeley, MD, JD^{a,b}

^aDepartment of Pediatrics, Children's Hospital of Pittsburgh, 3705 Fifth Avenue, Pittsburgh, PA 15213, USA

^bDepartment of Pediatrics, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

^cDepartment of Rehabilitation Science and Technology and Department of Bioengineering,
University of Pittsburgh, 5034 Forbes Tower, Pittsburgh, PA 15260, USA

The neurosurgeon caring for the potentially abused infant or child is asked to look at the injuries and determine what happened, when it occurred, and if the provided history is consistent with the findings. Unlike most injury scenarios, where the patient presents with a known cause of injury, the history given for an infant or child who has suffered from inflicted trauma contains little or no true information about what actually occurred. The stated cause is often deceptive, but may contain elements of the truth that make the story seem more plausible. In some cases, the person bringing the child for care may be unaware that an injury has even occurred, but seeks care because of concerns about the infant's symptoms, such as vomiting, increased sleeping, or irritability. Because accidental and abusive trauma can present similarly, it is imperative that the physician caring for the head-injured infant consider the stated mechanism and all of the clinical findings before reaching any conclusions as to cause. If the injury type and severity do not match the expected injury potential of the stated cause, additional information should be sought to better define the extent of injury and the details of how this occurred. Because the true mechanism of injury is rarely known in abuse cases, the physician must use all available information to determine best the manner of injury. The story is to

be reconstructed from the physical findings, clinical presentation, and time course of the patient. Diagnosing abuse in the young child or infant requires piecing together a puzzle of facts without assumptions.

A missed diagnosis of abusive head trauma can be catastrophic because the young infant or child who is being abused has a very high likelihood of suffering further insult that may result in morbidity and even mortality [1-4]. Equal in harm to the family and the child is when a truly accidental head injury is diagnosed as an abusive event. Careful and expert consideration is required to decrease the likelihood of either accusing innocent families of abuse, or misdiagnosing a child who has suffered abusive trauma, placing the patient back into a potentially harmful environment. To improve accuracy and expert opinion concerning the manner and timing of the injury, a biomechanical approach is the key. An understanding of the biomechanics of specific injuries and specific injury mechanisms provides a more objective approach for evaluating consistency between the stated cause of injury and the actual clinical findings. This understanding may also help guide therapy, interventions, and anticipation of the potential for neurologic deterioration. A biomechanical profile for evaluating head injuries in young infants has been outlined by Duhaime et al [5] and Hymel et al [6] has proposed a biomechanical approach for assessing potential abusive head trauma.

Inconsistency between the history and observed injuries is one of the red flags used to differentiate accidental and nonaccidental head trauma

* Corresponding author.

E-mail address: piercem@chplink.chp.edu (M.C. Pierce).

Pierce, M.C. et al.
(Head trauma)



(i.e., a minor history accompanied by major or multiple injuries). In some cases this discrepancy may be based on learned assumptions rather than experience and medical reason. It is critical to keep an open mind to the injury potential for various mechanisms, while gathering more data in a nonaccusatory manner. A biomechanical approach to evaluating the head-injured patient can provide a more scientific and objective framework for diagnosing a discrepancy between history and injury. Evaluating this inconsistency and determining that a head injury is or is not consistent with the stated history requires (1) an understanding of what types of biomechanical forces are necessary to result in specific types of head injuries, (2) what the mechanical injury potential is from the reported cause, and (3) what the expected clinical presentation and course are for a given mechanism and resultant injury. These key areas are the focus of this article, which provides an overview of concepts in injury biomechanics as they pertain to the clinical evaluation of an infant or young child who may have an inflicted head injury, and reviews experimental modeling of head injury. The reader is referred elsewhere for further information [7-10,95].

Evaluating the potentially abused infant or child

The physician must begin by obtaining a detailed history of the event and carefully defining all the cranial and noncranial injuries. A detailed history of the event with a focus on the biomechanical explanation of the cranial injury is essential. The history should be taken with sufficient detail to allow for biomechanical reconstruction of the incident. If the historian reports, for example, that the child fell down a flight of stairs, it is essential to ask how many steps, of what the steps are made, and on what type of surface the child landed. It is also important that the history is taken before providing the historians with any information about the patient's injuries. Documentation of the initial history is also important because in abusive trauma, the history often changes as the perpetrator learns more about the injuries and begins to fabricate a more realistic history of the event. The physician must next define the cranial injuries and then classify them as either primary or secondary. Although the presence of secondary injury has a profound effect on treatment and outcome, only the primary cranial injuries can be explained by biomechanics. If the patient requires an operative procedure, the

operating room is the place where the neurosurgeon is able to add a unique perspective to the biomechanical puzzle. In some cases of abusive injury, the only evidence of impact is that seen by the neurosurgeon in the operating room. In addition, the presence of new or old blood, and xanthochromia in the hemorrhage may provide important clues to the timing of the injury event.

To determine plausibility of a given set of injuries resulting from a stated cause, all of the injuries must be identified and taken into consideration. Can a single event explain all of the medical findings including time course of clinical presentation? Key aspects for consideration when evaluating the injury potential of a given mechanism are whether the stated cause generated sufficient energy to cause all of the discovered injuries, and whether the types of injuries match the biomechanics of the event.

Biomechanics of specific types of head injuries and other injuries commonly encountered in abuse

Brain injury can be caused both by forces directly applied to the brain and through indirect forces transmitted to the head through the neck. The primary head injuries from contact forces occur at impact. Contact forces cause focal injuries but are not the cause of diffuse brain injuries, such as a concussion [7]. Indirect forces, or inertial accelerations, are typically applied to body regions (e.g., torso) other than the head and often result in a whiplashing effect. Accelerations may be linear, rotational, or a combination of the two. Both direct and indirect forces applied to the head can result in accelerations, or changes in velocity, which lead to stress or strain within the brain. Gennarelli and Thibault [7] note that "strain is best understood as the amount of deformation that the tissue undergoes as a result of mechanical loading," and because "biological tissues are viscoelastic, their tolerance to strain changes with the rate at which the mechanical load is applied." Melvin et al [11] summarized the various mechanisms of brain injury as follows: (1) direct contusion from skull deformation, (2) brain contusion from motion relative to the internal skull surface, (3) reduced blood flow caused by pressure or infarction, (4) indirect contusion of the brain opposite the side of impact, (5) tissue strain produced by relative motion of the brain with respect to the skull or hemisphere, and (6) rupture or tearing of the blood vessels between the brain and dura mater.

External abrasions and contusions

Scalp and face findings add an important piece to assessing the plausibility of a given mechanism and provide information concerning contact forces or phenomena. Scalp abrasions, swelling, and bruising result from direct contact forces and should be identified and documented before any surgical interventions. The absence of scalp and subgaleal swelling does not preclude impact, and in some instances no impact can be found [12, 13]. A soft surface may dissipate the force of impact to the head, resulting in brain injury without visible signs of trauma [14,15]. In a study by Duhaime et al [14], all patients who died from abusive head trauma had evidence of blunt head trauma, but in several of the cases evidence of impact was found only at autopsy on reflection of the scalp. It is also important to document the number of planes the bruising reflects (i.e., forehead and back represent two different directions of impact force). Does the reported mechanism account for multiple directions of force or is the described event more unidirectional? Bruising about the ear or pinna is a relatively common finding in severely injured children where abusive trauma is the cause. This can be an important observation not only for helping validate or refute the history of injury, but for evaluating potential injuries associated with ear bruising, such as described by Hanigan et al [16]. The constellation of unilateral ear bruising, ipsilateral cerebral edema, and hemorrhagic retinopathy is described as the tin ear syndrome. Hanigan et al [16] hypothesize that the rotational acceleration produced by blunt trauma to the ear produces these findings. In their report, all of the children with these findings had poor outcomes. Bruising of the ear can be an important indicator of a more serious intracranial process [16]. Likewise, the presence of a subgaleal hematoma often indicates a skull fracture is present [17,18]. Bruising and abrasions elsewhere on the body should also be documented. These findings reflect points of contact and are helpful for injury reconstruction, biomechanical consideration of the event, and assessing the plausibility of the history. Soft tissue findings may also point to other, occult or nonobvious injuries.

Skull fractures

Skull fractures result from impact, or direct contact forces when energy is absorbed and injury

threshold is exceeded. Local inbending of the skull occurs with impact. This results in compression and tension strains on the inner and outer tables, respectively. The fractures begin in the inner table and take the path of least resistance. It is the thickness of the skull that determines fracture propagation length and direction [7,19]. Linear fractures result from a broader contact force, such as a floor, whereas depressed skull fractures result from a smaller, more focused impacting energy, such as the edge of a coffee table, or a small object, such as a hammer. Basilar fractures result from propagated stress waves [7]. Aging of skull fractures is difficult, but the presence of soft tissue swelling clinically or by CT may help indicate that the injury is acute [18]. Fractures of the skull occur in both accidental and abusive trauma. To help with differentiation, several papers have studied specific characteristics of skull fractures resulting from accidental versus abusive trauma. Multiple fractures, bilateral fractures, and fractures crossing sutures were more commonly associated with abuse cases [20]. The parietal or occipital bones were the site most often involved in abuse cases [21,22] and a depressed occipital fracture was found to be highly suspicious for abuse [23]. Simple linear fractures can occur from short distance falls, and are common in both accidental and abusive trauma [24]. Most of the fractures in Duhaime's et al [14] study on shaken baby syndrome were in the occipital or parieto-occipital region and "more complex injuries were associated with greater mechanical impact forces generated from higher falls" [5].

Cerebral contusions

Cerebral contusions are the result of direct contact forces and can occur directly beneath the site of impact, or away from the site of impact (contra coup) as a result of propagated energy [25]. The injuries are focal and occur most commonly in the frontal and temporal poles and on the inferior surfaces because of bony prominences. Gliding contusions are focal hemorrhages in the cortex and subjacent white matter. They are believed to result from acceleration-deceleration forces and are a common finding in cases of diffuse axonal injury (DAI) [25]. Contusions and subarachnoid hemorrhages are also seen from inflicted head injury, as identified by MRI [5]. In accidental cases, focal contusions are found in "more significant falls resulting in focal parenchymal contusions or focal subarachnoid hemorrhages." Of interest, those

children whose injuries resulted from accidental trauma did well clinically in general [5].

Subdural hematomas

Subdural hematoma (SDH) "is the most common and distinctive type of central nervous system injury detected on imaging studies in abuse" and in cases of inflicted head injury, occur more commonly in the occipital region and posterior interhemispheric fissure [3,5,17,26]. A subdural can occur from contact forces with resultant subdural bleeding occurring directly underneath the impact site. Epidurals, uncommon in child abuse, also occur from contact forces directly under site of impact, and are often associated with a skull fracture. Epidurals, and some subdurals resulting from direct contact, are associated with good clinical outcomes if diagnosed before secondary brain injury [27]. Most SDHs, however, occur from inertial or indirect angular acceleration-deceleration forces [28] that cause disruption of bridging veins or other surface vessels. Bridging veins are sensitive to high strain rate conditions; "SDH result from high-strain accelerations that produce short duration, high strain rate loading" [7,29]. With rapid angular deceleration of the head, the brain continues to rotate in relation to the skull and dura, resulting in strain forces on surface vessels. Once threshold is exceeded, tearing and resultant SDH occur. Depending on magnitude, rate, and duration of the acceleration-deceleration forces, injury threshold for diffuse brain injury may also be met [5,7,30]. In fact, SDHs have a high association with bad outcomes in traumatic brain injury victims. This morbidity and mortality association is caused in part by injury to cerebral parenchyma from the same acceleration-deceleration forces that caused the SDH [31].

Primary diffuse brain injury

"Concussion is the beginning of the clinically apparent continuum of primary diffuse brain injury" [6] and loss of consciousness at the time of injury is an indicator of primary diffuse brain injury [6,32]. Forces applied to the head that result in rotation of the brain cause diffuse brain injuries, and at the moment of impact, diffuse damage to the white matter can occur [7,15,32,33]. Contact forces do not directly cause diffuse brain injury [7], although the contact strains may set the head in motion, resulting in acceleration-deceleration forces sufficient to cause diffuse brain injury [7,25]. Concussions and DAI are caused by

inertial or acceleration-deceleration forces injurious to the brain parenchyma by producing strains within the brain tissue. The acceleration loading causes sufficient strains within the brain so that the brain tissue itself is injured [7]. When the patient is severely or fatally injured, nerve fibers in the white matter are torn at the time of the impact [33,34]. Direction of acceleration and impact occurrence are important variables affecting injury potential. Greater damage occurs in animal models with movement in the coronal plane, or lateral head motion [35]. Duhaime's et al [14,15] model of a shaken infant showed that the magnitude of angular deceleration is 50 times greater with impact. Angular acceleration forces, when associated with impact, reach calculated injury threshold for SDH, concussion, and DAI.

In the infant with brain injury, it is important to determine if the infant lost consciousness at the time of the injury, and if so, for how long. If the mechanism from history is more translational in force production, such as a short distance fall, and yet the injuries are consistent with angular acceleration (i.e., SDH, loss of consciousness, or DAI) further investigation is critical. Did the fall generate greater biomechanical forces than assumed or expected, or are there additional findings inconsistent with accidental trauma?

Secondary injury and brain edema

The causes of brain swelling are not always clear, but can occur secondary to the primary insult. Swelling may be focal, unilateral, or diffuse [9,25]. Focal swelling is typically adjacent to a contusion as a result of direct impact injury; unilateral cerebral edema usually occurs in association with an ipsilateral SDH [25]. Diffuse swelling can result from diffuse structural damage or increased cerebral blood flow, or swelling can be a reflection of the added injury of hypoxia and ischemia. The precise cause of cerebral edema in any given case is not always clear, and in abusive head trauma multiple mechanisms may coexist. Swelling can occur immediately after major blunt force trauma, or soon after when significant brain injury has occurred [36,37]. Brain swelling may also take several hours or days to evolve, adding to the challenge of patient management and injury diagnosis.

In the severely injured child, apnea, hypoxia, and ischemia are often present, and contribute to the deleterious effects of the initial insult. The role of hypoxia and ischemia in causing severe

brain swelling is especially important to consider when an infant has been harmed as a result of a violent outburst. It is not uncommon for medical attention to be sought only after the caretakers fear the child has died, or after someone else comes onto the scene or into the home, initiating an emergency response call. This can delay care for hours, or possibly even days, thereby worsening secondary injury.

Other organ systems injured

In the infant presenting with a head injury, other systems should also be evaluated for injury. Liver or spleen bruising or lacerations and bony injuries, such as rib, metaphyseal, and long bone fractures, are relatively common findings in the battered infant with a brain injury. It is important to identify all injuries for the well-being of the patient and for consideration of the mechanism and the expected injury potential. The absence of any other injuries can also provide helpful information for evaluating the consistency between the stated cause and the clinical findings. Additional radiologic testing to detect additional occult, extracranial injuries should be performed in any child less than 2 years of age, any child who is developmentally delayed and cannot reveal a history of abuse, and any child who is comatose [38]. Findings on a skeletal survey can add important biomechanical information [5] about the injury including the point of impact, the force with which the impact occurred, and the direction of impact. A skeletal survey can also reveal the presence of previous, unreported trauma. Laboratory evaluation can often add additional evidence. The presence of increased liver or pancreatic enzymes in a patient without a history of hypoxia or hypotension suggests abdominal trauma [39,40] even in the presence of a normal abdominal CT scan. Injuries to the pancreas, retroperitoneal duodenum, and liver and spleen occur with forceful, concentrated direct blows to the epigastrium. When this constellation of injuries is discovered, abuse must be strongly considered. Other sites of injury add an important element when considering feasibility of a mechanism causing a specific injury or set of injuries.

Subtle or occult injuries of forensic significance

Injuries are often defined by whether or not they require intervention. Such injuries as a bruise on a cheek [41,42] or an abrasion on an arm have little consequence for treatment or outcome in

accidental trauma and are rarely even considered in the list of significant injuries after trauma. When the cause of injury is unknown, unclear, or abusive, however, defining injuries becomes a staged procedure in which injuries are defined as any change in the normal structure of the body at a clinical, radiographic, intraoperative, or pathologic-histologic level. Even mild laboratory abnormalities need to be considered injuries even if they do not warrant intervention.

When trying to identify cases of abuse and prevent wrongful accusations in cases of truly accidental injury, gathering as much information as is possible is critical. These findings may have no actual clinical or operative significance, but may be pivotal in differentiating abusive from nonabusive trauma.

Prior injuries

There may be multiple injury events when a child is in an abusive environment, and prior injuries may have occurred when infants and young children present with abusive head trauma. In Ewing-Cobbs et al [3], almost half of children with abusive head trauma had evidence of prior undiagnosed head injury on CT scan compared with none of the children with accidental injury. The affect of prior head injury as it relates to a new head injury is unclear but the possibility is raised that the biomechanics of repeat injury are different. Old injuries raise concerns for the child's future safety and may point to an ongoing, abusive environment. Each prior injury and its reported cause must also be considered when evaluating the current injury and mechanism.

Mechanical injury potential of common injury events and clinical observations

To differentiate between abuse and nonabusive head trauma, it is necessary to have an understanding of the biomechanics of mechanism and injury potential of injury where the cause is not abuse. One of the most common described mechanisms of injury in abuse cases is a fall, such as a fall from a couch, bed, or crib, or a fall down stairs. There is a vast body of literature addressing the topic of injuries from fall, associated injuries and mechanisms, and the expected injury potential of a given mechanism. An understanding of the biomechanics of accidental falls aids the neurosurgeon in evaluating the expected injury potential of a given history. This section highlights some of these

articles on injury mechanism, and the clinical and experimental outcomes.

In part, the laws of physics govern the types of injury occurring in free falls. Heights of fall and material properties of the impact surface are the primary factors influencing free-fall injury severity. The fall victim's weight and impact landing position are also key to predicting free-fall outcome. The height of a free fall plays a key role in the injury outcome. Impact force, which is a function of body mass, fall height, and stopping distance, also is a critical factor in resultant free-fall injuries. Stopping distance is largely a function of impact surface properties. In particular the stiffness or energy absorbency of the surface influences stopping distance. Impacting on a soft surface, such as mud, results in a change in velocity over a longer period of time, which reduces the level of deceleration to what the falling body is exposed. Conversely, falling onto a stiff, rigid surface, such as concrete, results in a shorter stopping distance and ultimately a higher level of deceleration. Higher levels of deceleration are translated into greater injury severity. Impact energy, which is directly related to body mass and impact velocity, is also a factor influencing free-fall outcome. The distribution of impact force may also play a key role in free-fall injury. Forces that are distributed over a larger portion of the body, as opposed to a concentrated region, serve to diminish impact force in a fall.

Falls are one of the most common causes of traumatic death in children under the age of 15 years [43]. Based on a 1986 study of children who were hospitalized because of brain injury in San Diego County, California, it is estimated that nearly 30% of childhood injury deaths resulted from head injury. An estimated 34,100 children age 0 to 4 years in the United States suffered traumatic brain injury. Of these, half were related to falls [44]. This high incidence of falls was also confirmed as the primary cause of injury in children age 0 to 12 years [45]. In the study by Gallagher et al [45], falls were also found to be responsible for the greatest proportion of head injuries in all children. Krauss et al [44] reported in a 1990 study that 56% of serious brain injuries in children younger than 1 year were associated with abuse. Billmire and Myers [24] found that 36% of head injuries were associated with abuse in infants 11 months and younger. Chadwick et al [46] examined fatal outcome of injury in 317 infants and children who presented with a history of having fallen. Their study found that in fatal falls of less than 4 ft, the history was incorrect.

Duhaime et al [5] studied 100 consecutively admitted children with head injuries, age 2 years and younger, to determine injury mechanism and injury types. Findings indicate that most injuries were the result of falls, and that fall heights were found to influence injury types. Most household falls were found to be neurologically benign. Results also revealed that 24% of cases were associated with inflicted trauma and 32% were suspected abuse. This study found that intradural hemorrhages were more likely to be associated with motor vehicle accidents and abusive injury; abuse was the most common cause of mortality. Reece and Sege [47] conducted a retrospective review of 287 children 6.5 years and younger with head injuries to determine the injury mechanism and type of injury. Mechanisms were classified as either definite abuse or accident. Eighteen percent of the cases were classified as accidental, with a mortality rate of 2%, whereas 19% were classified as definite abuse, with mortality rates of 13%. Subarachnoid hemorrhage, SDH, and retinal hemorrhages were more prevalent in the definite abuse cases. Falls were responsible for 58% of the accident cases. In actual falls less than 4 ft, 8% had SDH, 2% had subarachnoid hemorrhages, and none had retinal hemorrhages. In contrast, in abuse cases falsely reported as falls from 4 ft or less, 38% had SDHs, 38% had subarachnoid hemorrhages, and 25% had retinal hemorrhages.

Falls from heights

In one of the earliest examples of injury science, presented in 1942, DeHaven [48] investigated falls from heights of 50 to 150 ft. In the eight cases examined, all subjects survived. DeHaven [48] found that the primary causes of injury were attributable to impact surface and localization of force. Distributing the impact force over time and space can help to ameliorate injury risk. He concluded that "structural provisions to reduce impact and distribute pressure can enhance survival and modify injury"; falls onto yielding surfaces can serve to reduce injury. Richter et al [49] studied vertical deceleration injuries associated with both intentional (suicide) and unintentional high falls, which averaged 7.2 m in height. In 101 subjects, head injuries occurred in 27% of the cases. In this study, severity of injury was correlated with fall height, type of impact surface, and impact position of the body. Striking the ground headfirst corresponded with only a 50% survivability rate. Warner and Demling report

that children most frequently tend to impact in the headfirst position in falls [50]. Head injuries are common in pediatric falls [51]. The mortality rate from falls of four stories or less in children (2% to 20%), however, is lower than that in adults (50%). Warner and Demling postulate that the improved survivability in children is likely caused by their lower body mass, which reduces deceleration levels, and their reduced body stiffness because of a greater proportion of cartilage to bone [50, 94]. Lallier et al [52] studied falls in children (mean age 7.4 year) from a height of 10 ft or greater. Major injuries included head trauma in 39% of the cases. Head injuries occurred more frequently in younger children. The authors conclude that the greater surface area of the head relative to the body and higher center of gravity of younger children make them more susceptible to head injury in falls. Overall, head injury was the most frequent reason for hospitalization. Williams [53] prospectively evaluated falls witnessed by a noncaregiver. His study concluded “that infants and children are relatively resistant to injuries from free falls, and falls of less than 10 feet are unlikely to produce serious life-threatening injury.”

Short-distance falls are among the most common types of accidents to occur in children. Short falls are also one of the most common falsely reported mechanism of injury in cases of child abuse and inflicted head injury. Plunkett [54] reviewed 18 fall-related head injury fatalities involving equipment, such as swings or platforms. Ages ranged from 12 months to 13 years, with falls ranging from 2 to 10 ft. Distance of fall was defined as the distance of the closest body part rather than center of mass from the ground at the beginning of the fall. If the specific distance could not be determined, a range of possible minimum and maximum height was given. Whether the event was or was not witnessed by a noncaregiver was recorded and in one case, the injury event was videotaped. Plunkett [54] concluded that falls from less than 10 ft may cause fatal head injury, that such injuries may not always be associated with immediate symptoms, and that a history of a short-distance fall in a seriously head-injured patient should not be dismissed. Because of the complexity of each injury event, the recommendation is made that a biomechanical analysis be made for any incident in which the severity of the injury seems to be inconsistent with the history [54].

A 1977 study conducted by Foust et al [55] investigated impact tolerances of the head and

lower extremities in free-fall accidents occurring in children, women, and the elderly populations. This study used a combination of techniques and investigative strategies to create an epidemiologic database to analyze injury patterns and define an association between biomechanical measures and injury severity. These researchers relied on scene investigations, medical record assessment, theoretical biodynamic calculations, experimental free falls using anthropometric test devices, and validated two-dimensional computer simulation models to predict key biomechanical impact measures, which have previously been correlated with injury risk. It was found that for given accident conditions, children were generally injured less severely than adults exposed to the same conditions. Simulation models were used to predict whole-body energy, impact velocity, and momentum. Body impact position was also found to have influence on injury risk and resulting injuries. For rigid impact surfaces, researchers showed a relation between types and severity of injury, and age and fall distance. Positive relations between overall abbreviated injury scale and injury severity scale with impact velocity were also found for free falls. Correlations between injury severity level and whole body biomechanical measures were observed only for headfirst-type impact accidents. For children 18 months and younger, a fall distance of 4 to 10 ft was determined to be the threshold for skull fracture. Headfirst falls from greater than 10 ft onto a rigid surface were predicted to result in skull fracture or concussion and at least Abbreviated Injury Scale-2 (AIS-2) injuries for adults and children [55].

Injuries associated with falls from beds

MacGregor [56] evaluated 8343 pediatric emergency department records and determined that 85 were associated with falls from beds. Twenty seven of 85 children sustained a head injury during falls from a bed. Of these 27 cases, there were no skull fractures and no evidence of intracranial bleeding. In a study conducted by Lyons and Oates [57], 207 children who had fallen from either a bed or crib were evaluated to determine type of injury and injury severity. Lyons and Oates [57] sought to answer the question of how likely is it that a fall from a low object, such as a bed, would lead to severe injury. Of the 207 cases, only one head injury (skull fracture) was identified. They concluded that falls from short distances, such as beds and cribs, do not lead to multiple or clinically

significant injuries. Additional studies by Nimityongskul and Anderson [58] and Helfer et al [59] evaluating falls from beds support the concept that falls from short distances generally do not result in severe head injuries.

Stairway injuries in children

Joffe and Ludwig [60] sought to describe clinical findings in children 11 years and younger who presented to the emergency department and had fallen down stairs. Injury rarely occurred to more than one body part, and no patient had life-threatening injuries. Chiaviello et al [61] also studied stairway-related injuries. Injuries to more than one body part did not occur. Significant stairway-related injuries were reported in 22% of patients, especially if the fall occurred with the caregiver. Significant injuries included subdurals and skull fractures. There were no deaths. Bertocci et al [62] used computer modeling of a stair fall in a 3 year old and found that the stair number, slope, and surface had an important influence on biomechanical measures associated with injury risk.

Falls will continue to be one of the most common types of injury mechanisms in children and will also be common in false reports of injury in the abused child. Assumptions of what is or is not possible must be replaced with careful and detailed analysis of all of the clinical and social findings, and the biomechanical principles behind both the event and the injuries. If the patient's clinical course is poor, resulting in either a fatal or severely disabled outcome, the history of a minor mechanism is in question [33]. Determining that a mechanism is truly minor, however, requires biomechanical consideration of the injury environment and event. Underlying brain injury is almost always minimal when the mechanism is a low-height fall. This and other studies support the conclusion that significant injuries require greater biomechanical forces and that the type of force determines the particular type of associated injury [5]. The possibility of a serious or even fatal head injury does exist, however, from a short-distance fall and the history of such cannot be assumed to be incorrect [54]. Much work still needs to be done to define injury tolerance better in children and to gain an improved understanding of the types and magnitude of biomechanical forces generated by common childhood injury scenarios and their associated injury potentials. Specifically, the influence of initial conditions, protective reflexes, surface impact, and fall dynamics are integral in evaluating injury potential.

Clinical presentation and course for a given injury and mechanism

Expected behavior and symptoms

When evaluating the stated cause of injury, the history must be analyzed from a biomechanical point of view. Factors to be considered include the following:

1. Is the described behavior of the child during and after the trauma event consistent with the clinical findings?
2. What are the developmental capabilities of the child?
3. Did the child lose consciousness?
4. Was there a lucid interval?
5. Are there other injuries that negate the possibility of the child behaving normally after the injury, such as a transverse fracture of the femur, or liver laceration?
6. Was there evidence of a hypoxic-ischemic event or a delay in seeking care?
7. Were the clinical symptoms of serious injury subtle or even occult, and the delay because of lack of recognition of a problem?
8. Do the facts of the story as told by the caregiver remain consistent, or does the story change to try and retrofit any newly discovered injuries?

It is critical that medical personnel not jump to conclusions and that additional information is gathered as needed to make sense of or refute the history and injuries from a biomechanical perspective, and to ensure safety for the child and family.

Other contributing factors

Multiple primary trauma events must be considered as a possibility in the infant presenting with a severe head injury. Further complicating the evaluation is the common occurrence of repeat primary traumatic events over days, weeks, or even months. Repetitive slapping, throwing, or shaking the infant may result in multiple, subclinical concussive events. This adds to the complexity of determining what happened and when it occurred. Is one seeing the results of a single event, or the results of multiple, repetitive insults?

Experimental head injury models

Many combinations of forces and accelerations can result from a traumatic event that may

lead to brain injury. Accordingly, there have been many studies in support of developing brain injury criteria, most of which are based on the input forces or accelerations delivered to the head. Various, sometimes conflicting, theories have been postulated to predict the type of head injury that may result from given forces or accelerations imparted to the head. Most of the studies can be classified based on either head impact or head acceleration models. Acceleration models can be grouped further by either rotational or translational accelerations of the head.

Direct-impact head injury models

Direct-impact head injury studies can be grouped into those that permit free movement of the head following impact and those that constrain head movement on impact. Early experiments conducted by Gurdjian et al [63] in anesthetized primates used a piston impactor to investigate impact force, head accelerations, and resulting injuries in primates. In these experiments, the neck was used as the only means of constraint, allowing for a complex dynamic response of the head making it difficult to determine a cause and effect relationship. Injury severity, however, was found to be a function of impact velocity, impactor material properties, impactor mass, and impactor contact area [11]. Subsequent studies attempted to constrain head motion to a single plane during and following impact so that reproducibility in injury could be attained [64–66].

Additional direct-impact studies were also conducted in cats and dogs using a captive bolt head impactor to study cerebral contusion and edema [36,67–69]. Efforts have also been made to study direct head impact in rats; success has been limited, however, because convulsion is a common response in rats and may affect interpretation of resulting injuries [8,70,71]. In 1991, Goldman et al [72] used a pendulum impact model to investigate mild head injury in rats associated with direct impact. In these studies, Goldman et al [72] attempted to prevent skull fracture by resting the skull on an energy-absorbing pad. A key outcome of these experiments was a description of the relationships between severity of injury, body mass, skull strength, and impact loading parameters in the rat.

Acceleration-based head injury models

Early studies by Holburn [73] using photoelastic models determined that rotational accelerations could produce concussive head injury.

Gurdjian et al [74] postulated that head rotation, which leads to brain movement relative to the skull, could lead to injury. Ommaya et al [75] conducted studies in primates to describe the effects of head acceleration in 1966. These studies found that whiplash-type motion, resulting from indirect head forces, could lead to concussion. Ommaya et al [75] also studied direct impact and rotation of the head, demonstrating that concussion could also result from such mechanical input. Through this work it was determined that direct rotational acceleration levels, which were half that necessary to produce indirect rotational loading concussions, could produce concussions.

Ommaya and Genneralli [76] conducted subsequent experimental studies comparing the effects of rotational and translational accelerations. These experiments showed that diffuse injuries resulted only from rotational acceleration. Loss of consciousness was also more readily produced in this series of experiments at high levels of rotational acceleration than at high levels of translational acceleration [76,77]. In a subsequent series of controlled experiments, Abel et al [78] investigated the relationship between tangential acceleration in the sagittal plane and the presence of SDHs. A tangential acceleration of 714 g was defined as the threshold for SDHs. Genneralli et al [35] isolated and studied the effects of rotational deceleration in 1982. Their experiments expanded on previous studies through the additional factor of time exposure to deceleration. In general, their findings predicted that concussion and diffuse brain injury could result from decreasing levels of deceleration when time exposure was increased. Both the levels of deceleration and the duration of exposure to this deceleration are keys to predicting injury risk. Experiments have shown that two factors, acceleration and the time over which the acceleration occurs (time exposure), influence head injury risk and severity. Large rotational accelerations occurring over short periods of time typically result in SDH, whereas longer exposures to acceleration are often associated with DAI [7]. Lee et al [79] and Lee and Haut [80] conducted experiments that describing the relationships between tangential and rotational accelerations and the threshold for SDH in rhesus monkeys. In 1982, Gennarelli et al [35] studied the biomechanics of SDHs by investigating the effects of coronal plane head acceleration. Margulies et al [81] visualized the effects of coronal acceleration by using simulated silicone gel human and baboon brain models. These models allowed

for estimation of critical shear strain and a threshold coronal plane rotational acceleration for DAI in humans.

Repetitive head acceleration generated through indirect forces is also of interest, especially as it relates to the concept of "shaken baby syndrome." In a cat animal model, Nelson et al [82] and Barron et al [83] found that repetitive nonimpact accelerations were capable of producing immediate death, extended coma, or delayed mortality.

Injury criteria

Over the past 40 years, a great deal of effort has been devoted to the investigation of head injury mechanisms and the development of injury criteria. The most widely used criteria, to date, is the Head Injury Criteria (HIC), which was proposed in 1972 by the National Highway Transportation Safety Administration (NHTSA). The HIC have been developed to assess head injury risk associated with direct-impact events. The origins of the HIC are based on experiments conducted by Lissner et al [84] in 1960. In these experiments, embalmed cadaver heads were impacted on rigid surfaces to determine the accelerations which were associated with the onset of skull fractures. Because concussions were found to be present in 80% of skull fractures, test results could infer the onset of concussion. This work was compiled to represent the Wayne State Tolerance Curve (WSTC), which characterizes the relationship between acceleration, acceleration duration, and onset of concussion-fracture. The WSTC was later modified to include a wider range of acceleration exposures and to include findings associated with animal and human volunteer experiments [85]. Gadd [86] further enhanced the WSTC by adding data from long duration exposures and other primate sled tests, which resulted in the Gadd Severity Index. In 1971, Versace [50] proposed the HIC, which was subsequently slightly modified to include long duration human subject testing and was adopted by NHTSA in 1972. The HIC is based on a time weighting of the resultant translation head acceleration. The Federal Motor Vehicle Safety Standards limits the HIC value to 1000 for a mid-sized male test dummy.

Recently, NHTSA attempted to adapt the HIC established for mid-sized men to various sized crash victims. To do so, scaling factors must account for both geometric and material property differences. Material properties specific to the cranial sutures were chosen as the key scaling property reflecting

resistance to skull fracture in children. Accounting for these factors, the NHTSA Notice of Proposed Rulemaking proposed HIC values for children (Table 1). The probability of skull fracture ($AIS \geq 2$) associated with the proposed NHTSA HIC limits is estimated to be 47% across all test dummies [87,88].

Despite its wide usage, the HIC have important limitations. One limitation is the fact that the HIC are based solely on translational acceleration associated with impact. In most head injury events, it is common to have combined loading, which consists of both rotational and translational accelerations. To account for such combined loading, a model accounting for the complex mechanical response of brain tissue is required.

Other efforts related to the development of head injury tolerance levels include the work by Ommaya et al [89,90] to determine threshold levels of nonimpact angular acceleration and velocity. Nonimpact angular acceleration has been associated with DAI ranging from concussion to coma. Ommaya's experiments were based on primate testing, which were then scaled to establish an injury threshold for adult humans. The threshold reflected the relationship between angular acceleration, angular velocity, duration of exposure, and presence of concussion. Ommaya's head angular injury criteria were then further scaled to children using head mass and length scaling factors [91].

As shown in Table 2, children are able to tolerate a higher level of angular acceleration and velocity before onset of DAI. Working with Ommaya's et al data, Sturtz [92] formulated angular acceleration injury criteria for 6 and 3 year olds based on both impact and nonimpact events. Sturtz criteria also differentiated between duration exposures (Table 3). As shown in Table 3, as the duration of acceleration exposure increases the tolerance to acceleration decreases. Additionally, younger children are able to tolerate a higher level of acceleration before onset of DAI.

Margulies and Thibault [93] also conducted primate studies and developed physical and analytic models to establish injury thresholds associated

Table 1
HIC values for children

Test dummy	Mid-sized male	6 year old	3 year old	1 year old
Proposed HIC Limit	1000	1000	900	660

Table 2
Ommaya's head angular acceleration scaled injury criteria

	Angular velocity limit (rad/sec)	Angular acceleration limit (rad/sec ²)
Adult	30	<1700
6 year old	33	<2106
3 year old	34	<2255
1 year old	37	<2524

with angular acceleration and velocity. Using a mass scaling factor, they also described the relationship between angular acceleration, angular velocity, and the onset of DAI for infants.

Summary

Much of what is understood as potential for injury is based in what has been observed clinically. This knowledge base is critical for decision making but has inherent and important limitations. Experimental studies investigating the influence of environmental factors, such as height of fall and surface type on injury potential, add important information, but also have inherent limitations. Important trends and predictions of probable injury can be studied but inference to a specific child's injuries is difficult because of unaccounted for contributing factors of injury risk. Such factors include muscle contraction, protective reflexes, and specific tissue response to trauma forces. Additional biomechanical research is needed to bridge the gap between clinical observations and experimental predictions.

The specific and unique perspective of the neurosurgeon is a critical piece in differentiating accidental and nonaccidental head injury with experience and reason as the basis of the conclusion. Do the physics of the injury match the mechanistic principals of the described injury event? Could all of the injuries result from the event? Is it plausible that these set of injuries occurred from the described event based on the

Table 3
Sturtz scaled angular acceleration tolerances (rad/sec²)

Duration	Impact type	Adult	6 year old	3 year old
10 ms	Indirect	7020	7390	8140
3 ms	Indirect	70,200	73,900	81,400
10 ms	Direct	71,732	1823	2008
3 ms	Direct	7900	8300	9100

physician's experience and the current scientific understanding of injury biomechanics? Do the mechanical forces of the reported mechanism and injuries match?

To determine that an explanation is plausible requires consideration of all the facts and injuries, consideration of the described behavior, and consistency with the neurologic status. These facts of the case are compared with medical knowledge and the learned experience of the neurosurgeon. The answer to the question "is it possible?" is based on clinical experience and objective reasoning. Rather than a black box question and answer based in unrealistic probability, the answer is based on the facts of the case and physical principles that govern biomechanics and resultant injuries.

References

- [1] Alexander R, Crabbe L, Sata Y, et al. Serial abuse in children who are shaken. *Am J Dis Child* 1990; 144:58–60.
- [2] Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and non-inflicted traumatic brain injury in young children. *Pediatrics* 1998;102:300–7.
- [3] Ewing-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Childs Nerv Syst* 2000;16:25–34.
- [4] Jenny C, Hymel KP, Ritzen A, et al. Analysis of missed cases of abusive head trauma. *JAMA* 1999; 281:621–6.
- [5] Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics* 1992;90:179–85.
- [6] Hymel KP, Bandak FA, Partington MD, et al. Abusive head trauma? A biomechanics-based approach. *Child Maltreatment* 1998;3:116–28.
- [7] Gennarelli TA, Thibault LE. Biomechanics of head injury. In: Wilkins RH, Rengachary SS, editors. *Neurosurgery*. New York: McGraw-Hill; 1985. p. 1531–6.
- [8] Govons SR, Govons RB, Vanluss WD, et al. Brain concussion in the rat. *Exp Neurol* 1972;34:121–8.
- [9] Le Roux PD, Choudhri H, Andrews BT. Cerebral concussion and diffuse brain injury. In: Cooper PR, Golfinos JG, editors. *Head injury*. 4th edition. New York: McGraw-Hill; 2000. p. 175–99.
- [10] Bandak FA. On the mechanics of impact neurotrauma: a review and critical synthesis. *J Neurotrauma* 1995;12:635–49.
- [11] Melvin J, Lighthall H, Kazunari U. Brain injury biomechanics. In: Nahum A, Melvin J, editors. *Accidental injury—biomechanics and prevention*. New York: Springer-Verlag; 1993. p. 268–91.

- [12] Alexander R, Sato Y, Smith W, et al. Incidence of impact trauma with cranial injuries ascribed to shaking. *Am J Dis Child* 1990;144:724-6.
- [13] Gilliland M, Folberg R. Shaken babies—some have no impact injuries. *J Forensic Sci* 1996;41:114-6.
- [14] Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome: a clinical, pathological, and biomechanical study. *J Neurosurg* 1987;66:409-15.
- [15] Duhaime AC, Christian CW, Rorke LC, et al. Nonaccidental head injury in infants—the “shaken-baby” syndrome. *N Engl J Med* 1998;338:1822-9.
- [16] Hanigan WC, Peterson RA, Njus G. Tin ear syndrome: rotational acceleration in pediatric head injuries. *Pediatrics* 1987;80:618-22.
- [17] Kleinman PK, Barnes PD. Head trauma. In: Kleinman PK, editor. *Diagnostic imaging of child abuse*. 2nd edition. St. Louis; Mosby; 1998. p. 285-342.
- [18] Kleinman PK, Spevak MR. Soft tissue swelling and acute skull fractures. *J Pediatr* 1992;121:737-9.
- [19] Gurdjian ES, Webster JE, Lissner HR. The mechanism of skull fracture. *Radiology* 1950;54:313-39.
- [20] Meservy CJ, Towbin R, McLaurin RL, et al. Radiographic characteristics of skull fractures resulting from child abuse. *Am J Neuroradiol* 1987;149:173-5.
- [21] Hahn YS, Raimondi AJ, McLone DG, et al. Traumatic mechanisms of head injury in child abuse. *Childs Brain* 1983;10:229-41.
- [22] Merten DF, Osborne DRS. Craniocerebral trauma in the child abuse syndrome: radiologic observations. *Pediatr Radiol* 1984;14:272-7.
- [23] Hobbs CJ. Skull fracture and the diagnosis of abuse. *Arch Dis Child* 1984;59:246-52.
- [24] Billmire ME, Myers PA. Serious head injury in infants: accident or abuse? *Pediatrics* 1985; 75:340-2.
- [25] Graham DI, Gennarelli TA. Pathology of brain damage after head injury. In: Cooper PR, Golfinos JG, editors. *Head injury*. 4th edition. New York: McGraw-Hill; 2000. p. 133-53.
- [26] Hymel KP, Rumack CM, Hay TC. Comparison of intracranial computed tomographic (CT) findings in pediatric abusive and accidental head trauma. *Pediatr Radiol* 1997;27:743-7.
- [27] Shugerman R, Paez A, Grossman D, et al. Epidural hemorrhage: is it abuse? *Pediatrics* 1996;97:664-8.
- [28] Ommaya AK, Yarnell P. Subdural haematoma after whiplash injury. *Lancet* 1969;2:237-9.
- [29] Gennarelli TA, Thibault LE. Biomechanics of acute subdural hematoma. *J Trauma* 1982;22:680-6.
- [30] Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage: an experimental study. *JAMA* 1968;204:285-9.
- [31] DiMaio DJ, DiMaio VJM. Trauma to the skull and brain: craniocerebral injuries. In: *Forensic pathology*. Boca Raton, FL: CRC Press; 1993. p. 139-69.
- [32] Adams JH, Mitchell DE, Graham DI, et al. Diffuse brain damage of immediate impact type: its relationship to ‘primary brain-stem damage’ in head injury. *Brain* 1977;100:489-502.
- [33] Adams JH, Doyle D, Ford I, et al. Diffuse axonal injury in head injury: definition, diagnosis and grading. *Histopathology* 1989;15:49-59.
- [34] Gennarelli TA, Thibault LE, Graham DI. Diffuse axonal injury: an important form of traumatic brain damage. *Neuroscientist* 1998;4:202-15.
- [35] Gennarelli TA, Thibault LE, Adams JH, et al. Diffuse axonal injury and traumatic coma in the primate. *Ann Neurol* 1982;12:564-74.
- [36] Tornheim PA, Linwicz BH, Hirsch CS, et al. Acute responses to blunt head trauma: experimental mode and gross pathology. *J Neurosurg* 1983;59:431-8.
- [37] Willman KY, Bank DE, Senac M, et al. Restricting the time of injury in fatal inflicted head injuries. *Child Abuse Negl* 1997;21:929-40.
- [38] American Academy of Pediatrics Section on Radiology. *Diagnostic imaging of child abuse*. *Pediatrics* 2000;105:1345-8.
- [39] Cameron CM, Lazoritz S, Calhoun AD. Blunt abdominal injury: simultaneously occurring liver and pancreatic injury in child abuse. *Pediatr Emerg Care* 1997;13:334-6.
- [40] Coant PN, Kornberg AE, Brody AS, et al. Markers for occult liver injury in cases of physical abuse in children. *Pediatrics* 1992;89:274-8.
- [41] Labbe J, Caouette G. Recent skin injury in normal children. *Pediatrics* 2001;108:271-6.
- [42] Sugar NF, Taylor JA, Feldman KW. Bruises in infants and toddlers: those who don’t bruise rarely bruise. *Arch Pediatr Adolesc Med* 1999;153:399-403.
- [43] Smith M, Burrington J, Woolf A. Injuries in children sustained in free falls: analysis of 66 cases. *J Trauma* 1975;15:987-91.
- [44] Krauss JF, Rock A, Hemyari P. The causes, impact and preventability of childhood injuries in the US: brain injuries among infants, children, adolescents and young adults in the US. *Am J Dis Child* 1990; 144:684-91.
- [45] Gallagher S, Finison K, Guyer B. The incidence of injuries among 87,000 Massachusetts children and adolescents: results of the 1980-81 statewide childhood injury prevention program surveillance system. *Am J Public Health* 1984;74:1340-7.
- [46] Chadwick DL, Chin S, Salerno C, et al. Deaths from falls in children: how far is fatal? *J Trauma* 1991;31:1353-5.
- [47] Reece RM, Sege R. Childhood head injuries: accidental or inflicted? *Arch Pediatr Adolesc Med* 2000;154: 11-5.
- [48] DeHaven H. Mechanical analysis of survival in falls from heights of fifty to one hundred fifty feet. *War Medicine* 1942;2:586-96.
- [49] Richter D, Hahn MP, Ostermann PA, et al. Vertical deceleration injuries: a comparative study of injury patterns of 101 patients after accidental and intentional high falls. *Injury* 1996;27:655-9.
- [50] Versace J. A review of the severity index. *Proceedings of the 15th Stapp Car Crash Conference SAE Paper No. 710881*, 1971.

- [51] Barlow B, Niemirska M, Gandhi R. Ten years of experience with falls from a height in children. *J Pediatr Surg* 1983;18:509–11.
- [52] Lallier M, Bouchard S, St-Vil D, et al. Falls from heights among children: a retrospective review. *J Pediatr Surg* 1999;34:1060–3.
- [53] Williams RA. Injuries in infants and small children resulting from witnessed and corroborated free falls. *J Trauma* 1991;31:1350–2.
- [54] Plunkett J. Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 2001;22:1–12.
- [55] Foust D, Bowman B, Snyder R. Study of human impact tolerance using investigations and simulations of free falls. 21st Stapp Car Crash Conference Proceedings. SAE No. 770915. 1977.
- [56] MacGregor DM. Injuries associated with falls from beds. *Inj Prev* 2000;6:291–2.
- [57] Lyons TJ, Oates RK. Falling out of bed: a relatively benign occurrence. *Pediatrics* 1993;92:125–7.
- [58] Nimityongskul P, Anderson LD. The likelihood of injuries when children fall out of bed. *J Pediatr Orthop* 1987;7:184–6.
- [59] Helfer RE, Slovis TL, Black M. Injuries resulting when small children fall out of bed. *Pediatrics* 1977;60:533–5.
- [60] Joffe M, Ludwig S. Mechanical analysis of survival in falls from heights of 50 to 150 feet—stairway injuries in children. *Pediatrics* 1988;82:457–61.
- [61] Chiaviello CT, Christoph RA, Bond GR. Stairway-related injuries in children. *Pediatrics* 1994;94:679–81.
- [62] Bertocci GE, Pierce MC, Deemer E, et al. Computer simulation of stair falls to investigate scenarios in child abuse. *Arch Pediatr* 2001;155:1008–14.
- [63] Gurdjian ES, Lissner HR, Webster JF, et al. Studies on experimental concussion. *Neurology* 1954;4:674–81.
- [64] Gosch HH, Gooding E, Schneider RC. The lexan calvarium for the study of cerebral responses to acute trauma. *J Trauma* 1970;10:370–6.
- [65] Langfitt TW, Tannanbaum HM, Kassell NF. The etiology of acute brain swelling following experimental head injury. *J Neurosurg* 1966;24:47–56.
- [66] Shatsky SA, Evans DE, Miller F, et al. High-speed angiography of experimental head injury. *J Neurosurg* 1974;41:523–30.
- [67] Tornheim PA, McLaurin RL, Thorpe JF. The edema of cerebral contusion. *Surg Neurol* 1976;5:171–5.
- [68] Tornheim PA, McLaurin RL, Sawaya R. Effect of furosemide on experimental traumatic cerebral edema. *Neurosurg* 1979;4:48–52.
- [69] Tornheim PA, McLaurin RL. Acute changes in regional brain water content following experimental closed head injury. *J Neurosurg* 1981;55:407–513.
- [70] Nilsson B, Ponten U, Voigt G. Experimental head injury in the rat. Part I: Mechanics, pathophysiology, and morphology in an impact acceleration trauma. *J Neurosurg* 1978;47:241–51.
- [71] Ommaya AK, Geller A, Parsons LC. The effects of experimental head injury on one-trial learning in rats. *Int J Neurosci* 1971;1:371–8.
- [72] Goldman H, Hodgson V, Moorehead M, et al. Cerebrovascular changes in a rat model of moderate closed-head injury. *J Neurotrauma* 1991;8:129–44.
- [73] Holbourn AHS. Mechanics of head injury. *Lancet* 1943;2:438–41.
- [74] Gurdjian ES, Webster JE, Lissner HR. Observations of the mechanism of brain concussion, contusion, and laceration. *Surg Gynecol Obstet* 1955;101:680–90.
- [75] Ommaya AK, Hirsch AE, Flamm ES, et al. Cerebral concussion in the monkey: an experimental model. *Science* 1966;153:211–2.
- [76] Ommaya AK, Gennarelli TA. Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. *Brain* 1974;97:633–54.
- [77] Gennarelli TA, Thibault LE. Experimental production of prolonged traumatic coma in the primate. In: Villiani R, editor. *Advances in neurotraumatology*. Amsterdam: Excerpta Medica; 1983. p. 31–3.
- [78] Abel JM, Gennarelli TA, Segawa H. Incidence and severity of cerebral concussion in the rhesus monkey following sagittal plane angular acceleration. 22nd Stapp Car Crash Conference, Society of Automotive Engineers 1978;22:35–53.
- [79] Lee MC, Melvin JW, Ueno K. Finite element analysis of traumatic subdural hematoma. 31st Stapp Car Crash Conference. Society of Automotive Engineers 1987;31:67–77.
- [80] Lee MC, Haut RC. Insensitivity of tensile failure properties of human bridging veins to strain rate: implications in biomechanics of subdural hematoma. *J Biomech* 1989;22:537–42.
- [81] Margulies SS, Thibault LE, Gennarelli TA. Physical model simulations of brain injury in the primate. *J Biomech* 1990;23:823–36.
- [82] Nelson LR, Auen EL, Bourke RS, et al. A new head injury model for evaluation of treatment modalities. *Neurosci Abstr* 1979;5:516.
- [83] Barron KD, Auen EL, Dentinger MP, et al. Reversible astroglial swelling in a trauma-hypoxia brain injury in cat. *J Neuropath Exp Neurol* 1980;39:340.
- [84] Lissner HR, Lebow M, Evans FG. Experimental studies on the relation between acceleration and intracranial pressure changes in man. *Surg Gynecol Obstet* 1960;111:329–38.
- [85] Gurdjian ES, et al. Concussion—mechanism and pathology. Proceeding of the 7th Stapp Car Crash Conference, 1963.
- [86] Gadd CW. Use of a weighted impulse criterion for estimating injury hazard. 10th Stapp Car Crash Conference. Society of Automotive Engineers 1966;10:164–74.
- [87] Hertz. A note on the head injury criteria (HIC) as a predictor of the risk of skull fracture. 37th Annual Proceedings of the Association for the Advancement of Automotive Medicine, 1993.

- [88] Prasad P, Mertz H. The position of the United States delegation to the ISO working group 6 on the use of HIC in the automotive environment. SAE Government/Industry Meeting and Exposition, SAE paper No. 851246.
- [89] Ommaya AK, et al. Scaling of experimental data on cerebral concussion in subhuman primates to concussion threshold for man. 11th Stapp Car Crash Conference Proceedings, SAE No. 670906, 1967.
- [90] Ommaya AK, Hirsch AE. Tolerance for cerebral concussion from head impact and whiplash in primates. *J Biomech* 1971;4:13-31.
- [91] Klinich KD, Saul RA, Auguste G, et al. Techniques for developing child dummy protection reference values. Washington, DC: National Highway Transportation Safety Administration; 1996.
- [92] Sturtz G. Biomechanical data of children. 24th Stapp Car Crash Conference Proceedings, SAE No. 801313, 1980.
- [93] Margulies SS, Thibault LE. A proposed tolerance criterion for diffuse axonal injury in man. *J Biomech* 1991;25:917-23.
- [94] Warner K, Demling R. Pathophysiology of free-fall injury. *Ann Emerg Med* 1986;15:1088-93.
- [95] Case ME, Graham MA, Handy TC, Jentzen JM, Monteleone JA. The National Association of Medical Examiners Ad Hoc. Committee on Shaken Baby Syndrome. *Am J Forensic Med Pathol* 2001;22:112-22.