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– literature review

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Summary

This report documents a literature study which attempts to answer whether recoil-induced mild traumatic brain injury (mTBI) is a side-effect of firearm training. Recoil-induced mTBI has not been observed in any of the published work that we have evaluated. This may be either because the recoil force is not strong enough to induce mTBI, or because the number of shots associated with each work was too small. It is, however, worth noting that members of the Canadian armed forces have verbally reported symptoms of mTBI during repeated long-range rifle firing.

Based on the recorded head motion in the literature, it seems that if recoil-induced mTBI exists, then it is likely a consequence of rotational motion. On physical grounds, this makes sense as the brain's shear modulus is much lower than its bulk modulus, i.e., the brain deforms more during rotation than translation. A smaller sample of the recorded rotational head motion overlaps with the lower mTBI thresholds.

In the end, it is unclear if the reported mTBI symptoms arise due to shock loading, if they are undiagnosed mTBI from other exercises whose symptoms gets triggered by the recoil load, or if the mTBI was induced purely by repeated firing of long-range rifles. Experiments with longer shootings sessions, in combination with theoretical studies where the brain is recoil loaded by repeated firings may shed more light on this matter.

Sammendrag

I dette litteraturstudiet forsøker vi å finne ut om våpenrekyl kan forårsake mild hjernerystelse (mild traumatic brain injury – mTBI) i millitært personell.

Det har ikke blitt observert rekylindusert mild hjernerystelse i noen av artiklene vi har sett igjennom. Dette kan enten være fordi rekylkraften ikke er sterk nok til å forårsake mild hjernerystelse, eller så har det ikke blitt utført nok skudd i de aktuelle studiene. Det er likevel verdt å merke seg at soldater i den kanadiske hæren muntlig har rapportert om symptomer på mild hjernerystelse under lange økter med skytetrening.

Basert på den dokumenterte dynamikken virker det som at hvis rekylindusert mild hjernerystelse eksisterer, så er det en konsekvens av rotasjon heller enn bevegelse langs en akse (translasjon). At rotasjoner er farligere enn translasjoner, kan forklares ved at hjernens skjærmodul (sensitivitet mot skjærkrefter) er vesentlig mindre enn dens bulkmodul (sensitivitet mot kompresjonskrefter). Fysisk betyr det at hjernen deformeres enklere under rotasjoner enn translasjoner. Et lite utvalg av de dokumenterte rotasjonsdataene overlapper med de minste terskelverdiene som kan forårsake milde hjernerystelser.

Det er uklart om disse symptomene stammer fra sjokkbelastning, om det er snakk om udiagnostisert mild hjernerystelse med symptomer som dukker opp på grunn av rekylbelastningen, eller om den milde hjernerystelsen ble indusert av våpenets rekyl. Eksperimenter som tar for seg lengre skyteøkter, kombinert med teoretiske studier hvor hjernen belastes av rekyl fra reperte skudd kan tenkes å avklare dette.

Contents

1 Introduction

Mild traumatic brain injury (mTBI) is caused by hazards that military personnel frequently encounter, such as blast overpressure (breacher syndrome), shock waves (blast or firearms), and falls (CQB, field training). The symptoms are similar to a concussion and may affect how the soldier feels, thinks, acts, and sleeps. The most common symptoms are [1]:

- Physical.
  - Sensitivity to light and/or noise.
  - Balance problems and/or dizziness.
  - Tiredness.
  - Headaches.
  - Nausea or vomiting.
  - Vision problems.
- Thinking and remembering.
  - Reduced attention span and concentration.
  - Feeling foggy and groggy.
  - Problems with short- and/or long-term memory.
  - Trouble thinking cohesively.
- Social or emotional.
  - Anxiety and/or nervousness.
  - Irritability.
  - Feeling more emotional.
  - Sadness.
- Sleep.
  - Sleeping less or more than usual.
  - Trouble falling asleep.

mTBI may also lead to long-term neurological damage, which significantly decreases the soldiers or veterans quality of life. Since the symptoms are general mTBI is challenging to diagnose, especially so if an on-site physician is unavailable.

Recently it has been proposed by the Defence Research and Development Canada (DRDC), that repeated exposure to recoil also may cause mTBI. In this literature study we will evaluate the work that has been done, to attempt to understand this issue more deeply. Concretely, we will compare the measured head motion of soldiers to known mTBI thresholds in the sports literature. We emphasize that the field of recoil-induced mTBI is still in its infancy, so it is hard to draw rigorous conclusions without performing more testing.

2 Quantifying head injury

Head injury in the form of mTBI which does not originate from overpressure and shock waves typically originate from head motion. A head motion measurement typically consists of both linear and rotational acceleration as a function of time. In order to characterize different head motions and distinguish them from each other, several metrics have been developed. In this chapter we will summarize some of them. An overview over some of the existing injury criteria is shown in Tab. 2.1. The best criteria for characterising mTBI due to recoil needs to consider six-degrees of freedom, directional dependence, and loading duration.

Table 2.1 An overview over the various brain injury criteria that are often correlated with head injuries such as mTBI. The ones (or a variation) marked with an asterisk (*) are employed herein. The table is adapted from [2].

Injury criteria	Directional dependence	Loading duration
3DOF translation-only kinematic criteria		
Peak translational acceleration*	No	No
Wayne state tolerance curve (WSTC)*	No	Yes
Severity index (SI)	No	Yes
Head Injury criterion*, $\Delta t = 15$ ms (HIC_{15})*	No	Yes
Head Injury criterion, $\Delta t = 36$ ms (HIC_{36})	No	Yes
3DOF rotation-only kinematic criteria		
Peak rotational acceleration*	No	No
Rotational Injury Criterion (RIC)	Yes	Yes
Peak change in rotational velocity magnitude	No	No
Power rotational head injury criterion (PRHIC)	Yes	Yes
Brain Injury Criterion (BrIC)*	Yes	No
6DOF translation and rotation kinematic criteria		
Head Impact Power (HIP)	Yes	Yes
Generalized Acceleration Model for Brain Injury Threshold (GAMBIT)	No	No
Principal Component Score (PCS)	No	No
6DOF translation and rotation brain finite-element criteria		
Principal strain, corpus callosum*	Yes	Yes
Principal strain, whole brain*	Yes	Yes
Cumulative Strain Damage Measure ($CSDM_{15}$)*	Yes	Yes
Cumulative Strain Damage Measure ($CSDM_{25}$)*	Yes	Yes
Minimum pressure (P_{min})	Yes	No
Maximum pressure (P_{max})	Yes	No

2.1 Translational metrics

In this section we will present the relevant metrics that only take into account translational motion. While this is very useful in high-strain rate scenarios such as car crashes, they are less useful in more delicate cases such as firing rifles. Therefore the translational metrics presented here are useful in predicting the onset of severe brain injuries. Unfortunately, the metrics do display uncertain and varied thresholds for milder injuries such as concussions and mTBI. The main disadvantage of the translational metrics is that they completely ignore rotational motion. In modern approaches, the translational metrics must be supplemented by rotational metrics. Nevertheless, the translational metrics represent a useful starting point when analysing head motion.

2.1.1 Wayne state tolerance curve

In federal motor vehicle safety standards the so-called Wayne State Tolerance Curve (WSTC) is used to determine whether an acceleration is dangerous or not. The curve was first published by Gurdjian et. al. [3] in the 1960s. In the original dataset, human cadaver heads were subjected to linear accelerations and it was recorded whether linear skull fracture occurred. These points represent high-acceleration and short duration impact. The WSTC is often critized due to the small amount of data points, unclear definition of effective acceleration, old instrumentation, the curve does not differentiate between skull fracture and brain damage, and that impact location and direction were not varied. Nevertheless it is an important historical first step. The original curve as well as an extension, including rotational acceleration and more datapoints, are shown in Fig. 2.1. The WSTC curve indicates that the head can survive high accelerations for short durations. For long durations even relatively small accelerations can cause injury.

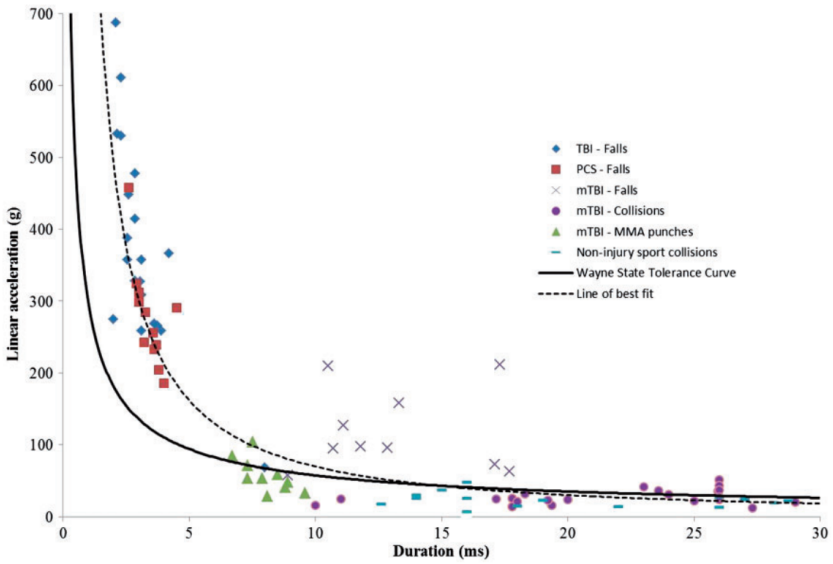


Figure 3. Linear acceleration magnitude/duration relationships for Hybrid III reconstructions of impacts resulting in brain injury and no-brain injury.

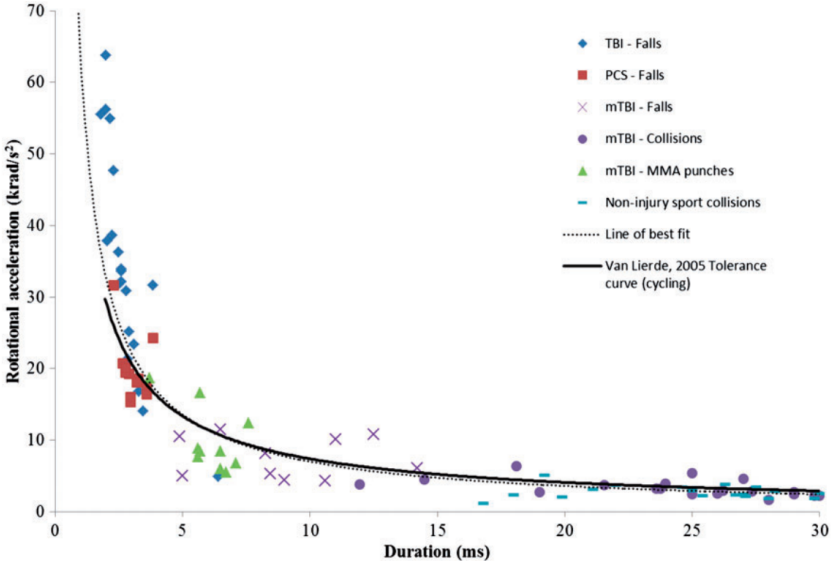


Figure 4. Rotational acceleration magnitude/duration relationships for Hybrid III reconstructions of impacts resulting in brain injury and no-brain injury.
mTBI: minor traumatic brain injury; MMA mixed martial art..

Figure 2.1 Wayne State University Curves taking into account both translations and rotations. The curve is reprinted from [4].

2.1.2 Gadd severity index

Gadd [5] proposed the severity index (GSI or SI) of the form

$$(\mathrm{G})\mathrm{SI} = \int_0^t a(t)^{2.5}\mathrm{d}t \tag{2.1}$$

where the exponent 2.5 comes from the slope of the orignal WSTC curve when plotted as a log-log plot. Here $a(t)$ and t represents the linear acceleration of the head's centre of mass in multiples of g and the duration of the impact in miliseconds. In the original work the threshold value for serious internal head injury is set to 1000.

2.1.3 Head injury criterion (HIC)

The National Highway Traffic Safety Administration (NHTSA) used the GSI to define the head injury criterion (HIC) as

$$\mathrm{HIC}_{t_2-t_1} = \max_{t_2-t_1}\left\{(t_2-t_1)\left[\frac{\int_{t_1}^{t_2} a(t)\mathrm{d}t}{t_2-t_1}\right]^{2.5}\right\}. \tag{2.2}$$

Here t_1 and t_2 are the initial and final times (in seconds) which are chosen to maximize the HIC. The length of the time interval $t_2 - t_1$ is typically chosen to be either 15 ms or 36 ms depending on the length of the primary loading in the car crash. The measurements are obtained from mounting an accelerometer at the center of mass of a crash dummy's head, and then applying crash forces. The HIC is one of (if not the) most common metric used to characterize head motion. An injury probability curve as a function of HIC was determined in [6, 7] and is displayed in Fig. 2.2.

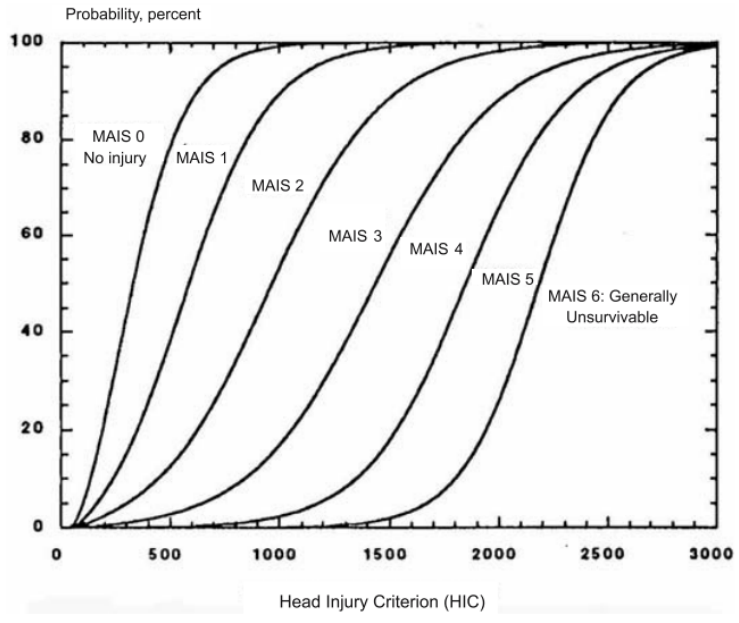


Figure 1. Probability of head injuries of different severities for given HIC values.

Figure 2.2 Injury probability as a function of HIC, as shown in [7]. MAIS stands for the maximum abbreviated injury scale.

2.2 Rotational metrics

In this section we introduce the brain injury criterion (BrIC) metric that was used in the literature to characterise rotational motion in the context of recoil-induced mTBI. In hindsight, it might have been better if the literature focused on the so-called power rotational head injury criterion (PRHIC), since the latter takes into account both directional and time dependence.

2.2.1 Early work

An overview of the rotational injury tolerance values was given by Ommaya in 1985 [8]. Ommaya's experiments focused on the onset of concussion in rhesus monkeys. The animal tolerances were translated into human tolerances by a mass scaling relationship between angular accelerations and brain mass. For humans the angular velocity and acceleration tolerances were determined to be $20 - 30\,\mathrm{rad/s}$ and $1800\,\mathrm{rad/s^2}$ respectively. The employed mass-scaling relationship is no longer thought to be mathematically sound, as noted by Ommaya himself: "*It should be reemphasized that this information (rotational tolerances) is considered to be reliable for the Rhesus, sketchy for the chimpanzee, and completely speculative for man.*". Furthermore it is suggested to revise the tolerances when human accident data become available, which they recently have through head measurements in different sports.

2.2.2 Brain injury criterion (BrIC)

Since its original inception in 2011, the BrIC has been modified [9] to the form

$$\mathrm{BrIC} = \sqrt{\left(\frac{\omega_x}{\omega_{x,c}}\right)^2 + \left(\frac{\omega_y}{\omega_{y,c}}\right)^2 + \left(\frac{\omega_z}{\omega_{z,c}}\right)^2}. \tag{2.3}$$

Here $\boldsymbol{\omega} = (\omega_x, \omega_y, \omega_z)$ and $\boldsymbol{\omega}_c = (\omega_{x,c}, \omega_{y,c}, \omega_{z,c})$ are the maximum angular velocity and critical angular velocity around the specified axes respectively. The specific critical angular velocities are given in Tab. 2.2 and were determined by measuring the head motion of collegiate american football players. Note that the while the original BrIC incorporated angular accelerations, the modified version does not. The angular acceleration was removed in the modified version because "*Angular acceleration did not correlate well to any physical parameter...*". In the paper, it is recommended to use the MPS bases risk curves because they have better correlation with the data. The risk

Table 2.2 The critical angular velocities associated with brain injury. The two sets of critical angles come from applying two different measures: the maximum principal strain (MPS) and the cumulative strain damage measure (CSDM). The table has been adapted from [9].

Critical max ang. vel.	Rad/s (CSDM based)	Rad/s (MPS based)	Rad/s (Average)
ω_x	66.20	66.30	66.25
ω_y	59.10	53.80	56.45
ω_z	44.25	41.50	42.87

curves associated with the BrIC are shown in Fig. 2.3. In the figures the head injury is categorised according to the abbreviated injury scale (AIS). The case of mTBI is not explicitly mentioned, but

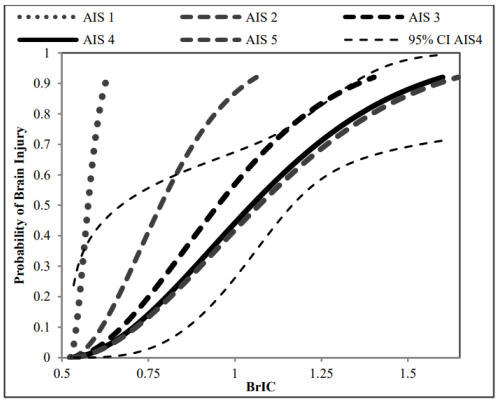


Figure 18. BrIC based on CSDM and formulation given by equation 4 (average critical angular velocities from Table 3).

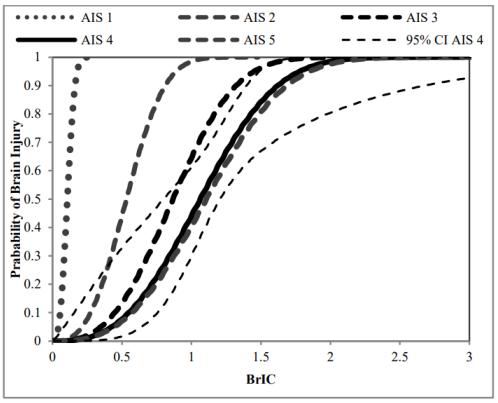


Figure 20. BrIC based on MPS and formulation given by equation 4 (average critical angular velocities from Table 3).

Figure 2.3 The probability of sustaining a head injury as a function of BrIC. The head injury is categorised according to the abbreviated injury scale (AIS). The curve is reprinted from [9].

the symptoms of an AIS 1 injury seems close to the symptoms of mTBI. Concretely the symptoms of an AIS 1 injury are [10, 11]:

- Cerebral injury with headache or dizziness, but no loss of consciousness.
- Whiplash complaint with no anatomical or radiological evidence.
- Abrasions and contusions of ocular apparatus (lids, conjunctivae, cornea, uveal injuries).
- Vitreous or retinal hemorrhages.
- Fractures and/or dislocation of teeth.

2.3 Finite-element metrics

With the advent of finite element simulations it has become possible to calculate the strains on a brain model given specific loadings. The combination of kinematic, rotational, and output of finite element codes have great potential for analyzing whether specific head motion results in various kinds of brain damage such as mTBI and concussions. Currently a lot of work is being done to attempt to determine metrics that correlate well with brain damage and their associated thresholds. Two very common metrics are the Maximum Principal Strain (MPS) and the Cumulative Strain Damage Measure (CSDM). Other metrics are the intracranial pressure, strain rate, and the product of strain and strain rate.

The MPS has consistently been associated with observed concussions, but there is large variation in the proposed thresholds. For example, for the corpus callosum the some sources have reported the MPS threshold to be in the interval $0.15 - 0.31$. As the field has matured, the literature has gradually started to report the 95th percentile MPS, as this is a measure that is less sensitive to single elements with artificially high strains due to numerical issues.

The CSDM is defined as the total volume of a specific brain region that experiences strains larger than a specific threshold, typically 0.15 or 0.25. Unfortunately, the correlation between CSDM and concussion is not as robust as compared with MPS, as the CSDM turns out to be very

sensitive to the choice of threshold.

At the time of writing it is the MPS_{95} that appears to be the most frequently used metric in finite element codes, but there is not consensus for the threshold for the onset of mTBI or concussions. Some MPS thresholds reported in the literature are given in Tab. 2.3.

Table 2.3 An overview over some of the reported MPS thresholds to cause mTBI. The table was adapted from section 2.6.3.1 in T. Seburrun's thesis [12].

Brain region.	Injury probability	Threshold	references
Entire brain	Median MPS concussed calue	0.16	xx
Grey matter	50% risk	0.26	xx
Thalamus	50%risk	0.13	xx
White matter	50% risk	0.26	xx
Midbrain	Mean concussed value	0.108	xx
Cerebrum	Median MPS concussed value	0.18	xx
Cerebellum	Median MPS concussed value	0.09	xx
Brainstem	Median MPS concussed value	0.14	xx
Corpus callosum	Median MPS concussed value	0.13	xx
Corpus callosum	50% risk	0.15	xx
Corpus callosum	50% risk	0.21	xx

3 Specific mTBI data from american football

The literature on recoil-induced mTBI is at the time of writing scarce, as the field is still in its infancy. However, if it is a measurable effect it will be induced by significant head motion. To get a sense of the magnitudes required, it may be useful to look at some concrete mTBI data in sports. Concretely, mTBI due to head motion has been throughougly characterised in american football. Typically a dataset consists of recorded or reconstructed head motion of football players, and a team-specific physician's assessment of whether the player has mTBI.

The results of a famous dataset from the National Football League is openly available and can be found in Pellmann et. al. [13]. In this work mTBI was defined as "*. . . a traumatically induced alteration in brain function manifested by an alteration of awareness or consciousness, including but not limited to a loss of consciousness, "ding," sensation of being dazed or stunned, sensation of "wooziness" or "fogginess, seizure, or amnesic period, and by symptoms commonly associated with postconcussion syndrome, including persistent headaches, vertigo, light-headedness, loss of balance, unsteadiness, syncope, near-syncope, cognitive dysfunction, memory disturbances, hearing loss, tinnitus, blurred vision, diplopia, visual loss, personality change, drowsiness, lethargy, fatigue, and inability to perform usual daily activities.*" The head motion data was obtained between 1996 and 2001 through video recordings. The head-motion curves were extracted from the recordings indirectly by subjecting test dummies to the same type of motion. Examples of data are shown in Fig. 3.1.

The loading was recorded for a total of 25 ms in total. For the translational motion the peak acceleration for the concussed and non-concussed player were approx. 80 g and 50 g respectively. The rotational acceleration for the concussed and non-concussed player were approx. 4700 rad/s^2 and 3000 rad/s^2 respectively.

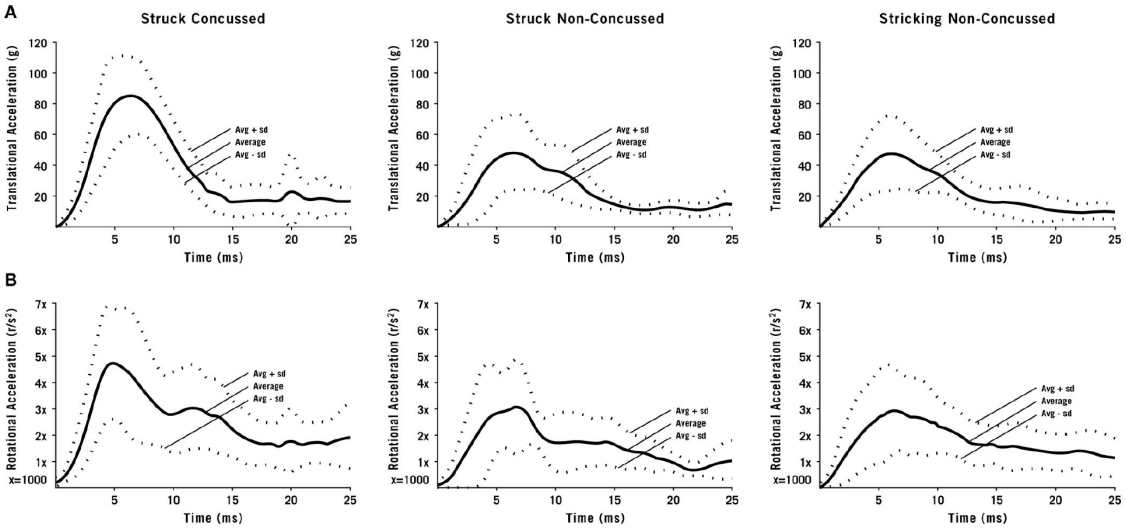


Figure 3.1 Head translation (A) and rotational (B) accelerations (average and standard deviation) for concussed (mTBI) and nonconcussed struck players and striking players, as determined from reconstructed game impacts. The Fig. is reprinted from [13].

An overview of the recorded NFL data is given in Fig. 3.2. In the cases were mTBI was

recorded the ranges of SI, HIC, Peak translation acceleration, and Peak rotational acceleration were 94 – 866, 77 – 730, 48 – 135 g, 2615 – 9678 rad/s^2.

TABLE 3. Peak biomechanical responses for struck and striking players in 31 National Football League game impact reconstructions[a]

	Struck players								Striking players						
Case	MTBI	Velocity (m/s)	SI	HIC	Peak translational acceleration (g)	Peak velocity change (m/s)	Peak rotational acceleration (rad/s^2)	Peak rotational velocity (r/s)	MTBI	SI	HIC	Peak translational acceleration (g)	Peak velocity change (m/s)	Peak rotational acceleration (rad/s^2)	Peak rotational velocity (r/s)
7	Yes	6.9	120	93	61	4.6	6266	28.1	No	65	51	50	2.2	2832	9.8
9	Yes	10.3	843	600	134	10.1	7428	27.4	No	275	217	79	2.3	6719	18.7
38	Yes	9.5	736	554	118	9.7	9678	50.8	No	157	127	60	4.0	5205	28.2
39	Yes	10.9	656	522	129	8.4	5921	36.1	No	60	43	44	2.3	4487	10.4
57	Yes	8.8	253	206	77	6.0	6514	37.0	No	48	38	32	4.1	4151	33.2
67	Yes	8.1	756	632	135	8.0	5957	13.8							
69	Yes	10.3	177	153	61	5.0	4381	19.9	No	55	50	38	3.1	2620	23.0
71	Yes	10.3	658	510	123	7.3	5400	35.0	No	512	434	102	6.6	5541	32.4
77	Yes	9.9	226	185	80	5.2	5148	36.4	No	65	53	35	4.2	2714	25.5
84	Yes	9.4	276	222	82	6.3	9193	80.9	No	96	78	45	4.4	3169	26.5
92	Yes	11.1	630	508	107	10.0	6878	44.2	No	204	164	60	5.6	6070	43.0
98	Yes	9.6	351	301	91	6.2	7548	43.4	No	241	187	84	4.8	4487	38.5
113	Yes	7.0	163	140	59	5.1	3965	12.8	No	101	75	61	3.7	3700	31.2
118	Yes	10.7	492	378	101	9.6	7017	42.9	No	122	73	56	3.7	3687	23.4
123	Yes	6.3	866	730	121	8.3	4727	30.3							
124	Yes	11.4	380	282	81	7.5	7138	34.8	No	105	73	56	3.1	4086	16.1
125	Yes	11.7	817	633	113	9.1	7716	63.3	No	132	111	47	4.2	3366	28.1
133	Yes	6.0	648	557	113	8.4	5012	16.0							
135	Yes	10.0	751	566	138	8.6	7540	41.0	No	230	179	81	3.8	5005	29.3
148	Yes	6.6	117	99	48	5.1	3476	23.9	No	47	37	33	3.9	2466	26.5
155	Yes	9.1	418	341	100	6.6	6940	37.0	No	76	61	45	4.2	4217	29.5
157	Yes	10.8	545	472	103	8.1	6750	33.5	No	215	180	79	5.0	4662	15.7
162	Yes	5.5	94	77	52	4.2	2615	18.4	No	34	30	29	3.2	1672	17.2
164	Yes	10.8	451	370	124	6.0	9590	26.6	No	243	202	89	5.1	6136	30.8
181	Yes	11.7	423	382	93	7.1	8011	36.5	No	402	333	85	7.3	6613	55.8
Concussed															
Average		9.3	474	381	98	7.2	6432	34.8							
SD		1.9	252	197	28	1.8	1813	15.2							
48	No	9.7	155	130	57	4.7	5617	42.4	No	44	37	32	3.2	2939	28.0
59	No	5.3	205	138	82	5.6	5387	26.9	No	32	26	32	2.3	2087	13.1
142	No	3.1	12	9	19	2.9	1170	7.4							
154	No	6.6	136	114	53	5.1	4167	24.0	No	35	31	29	3.1	3159	23.1
175	No	9.6	158	125	62	5.6	3555	39.2	No	81	62	47	3.9	2535	19.3
182	No	8.1	256	208	85	5.9	5512	17.8	No	272	213	87	4.7	3206	27.2
No injury															
Average		7.0	154	121	60	5.0	4235	26.3		146	117	56	4.0	3983	26.1
SD		2.6	82	64	24	1.1	1716	13.1		121	101	22	1.2	1402	10.0
t statistic		2.45	3.04	3.16	3.10	2.91	2.69	1.26		6.05	6.14	6.03	7.56	5.47	2.46
P value		0.0104	0.0025	0.0018	0.00215	0.0034	0.0059	0.1089		9.0E−08	6.5E−08	9.9E−08	4.0E−10	7.1E−07	0.0088

[a] MTBI, mild traumatic brain injury; SI, severity index; HIC, head injury criterion; SD, standard deviation.

Figure 3.2 An overview of the NFL mTBI data. The table is reprinted from [13].

Several authors have used the NFL data to estimate the probability for a player to obtain a

mTBI for a given loading curve. The loading curve is often characterised by a single translational or rotational metric. In modern approaches the loading curve is first used as an input to a finite element code modelling a human brain and the output (strain, strain rate, shear stress, etc) is used as dependent variable. Two examples are given by King [14] and Zhang [15] whose main results we have included below.

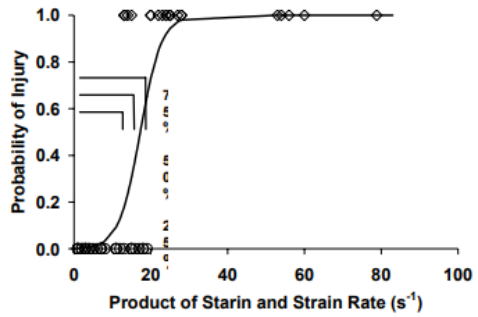


Fig. 9 - The probability of MTBI as predicted by the product of strain and strain rate in the midbrain region.

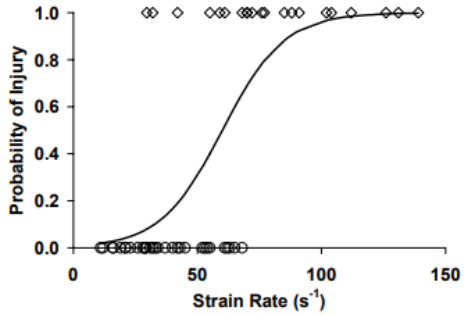


Fig. 10 - The probability of MTBI as predicted by strain rate in the midbrain region.

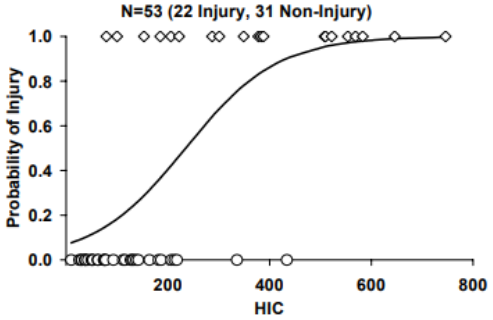


Fig. 11 - The probability of MTBI as predicted by HIC.

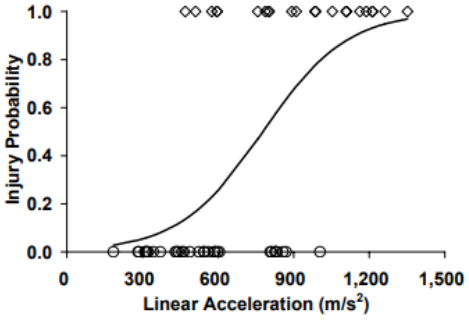


Fig. 12 - The probability of MTBI as predicted by head linear acceleration.

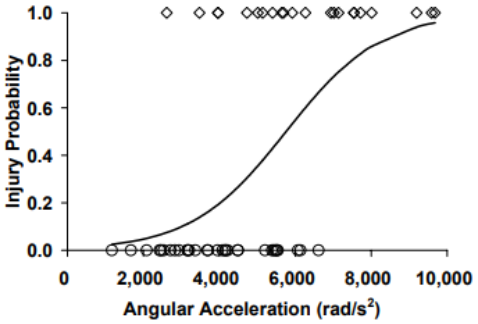


Fig. 13 - The probability of MTBI as predicted by head angular acceleration.

Table 2 - MTBI Tolerance Estimates for the Best Injury Predictors

Predictor	Probability of MTBI		
	25%	50%	75%
$_{max} \bullet d/dt_{max}$ (s^{-1})	14	19	24
d/dt_{max} (s^{-1})	46	60	80
HIC_{15}	136	235	333
Lin. Accel (m/s^2)	559	778	965
Ang. Accel (rad/s^2)	4384	5757	7130

Figure 3.3 The probability of injury as a function of various metrics. The Fig. is reprinted from King [14].

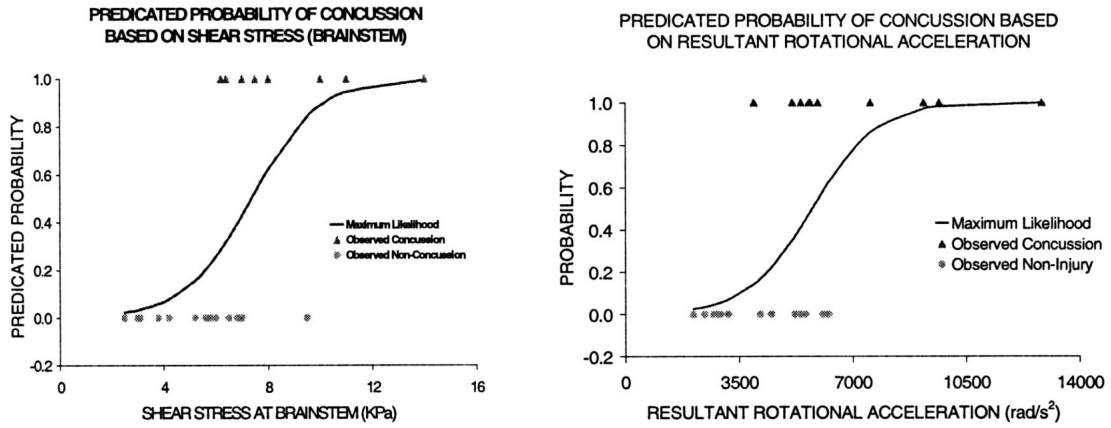


Fig. 8 Logist plots of the predicted injury probability based on shear stress at brainstem predicted by model and the input head rotational acceleration

Figure 3.4 The probability of concussion as a function of shear stress and rotational acceleration. The Fig. is reprinted from Zhang [15].

The NFL data are biased towards injurious impacts, which makes the statistical curves questionable. Concretely, the curves may be too conservative. To obtain more unbiased data J. R. Funk [16] collected data from college players at ”Virginia Polytechnic Institute and State University - Virginia Tech (VT)”. The players were wearing instrumented helmets (The Head Impact Telemetry System), and the measurements were performed during the 2003-2006 season. The resulting risk curves are shown in Fig. 3.5.

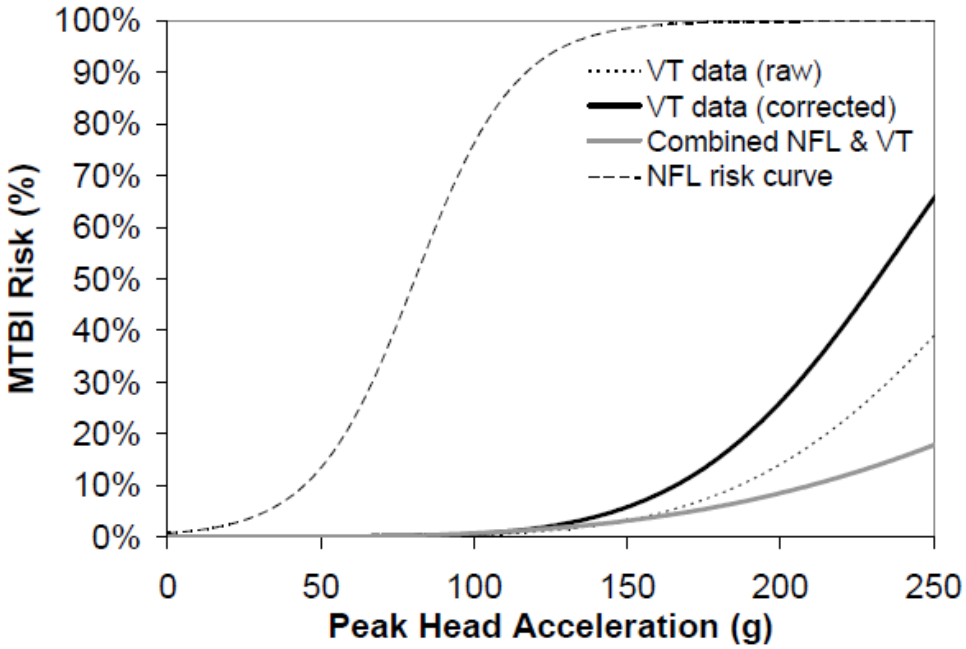


Figure 5 – Injury risk curves in terms of peak head acceleration.

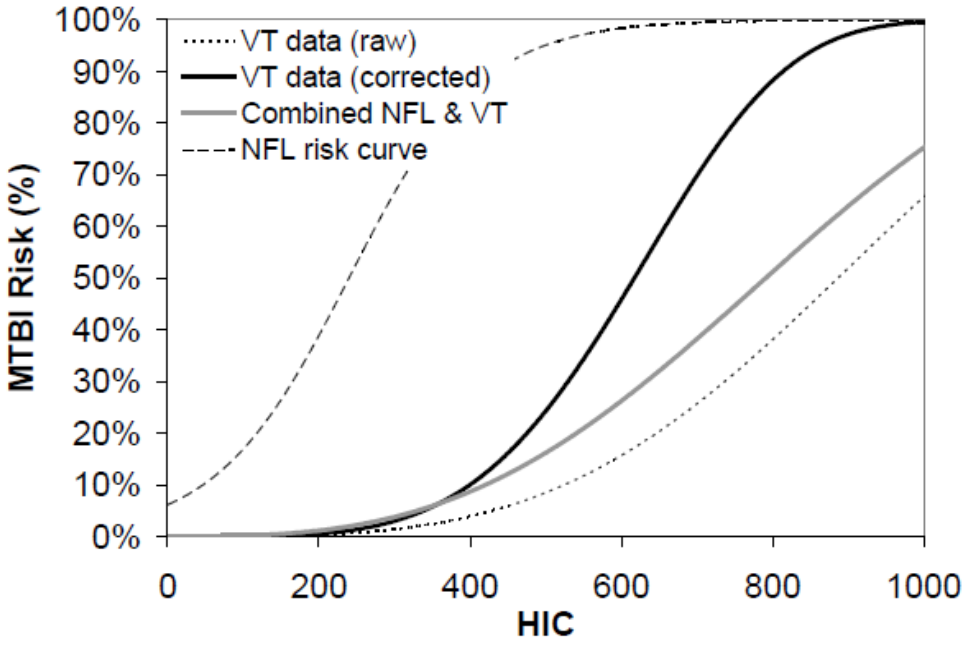


Figure 6 – Injury risk curves in terms of HIC.

Figure 3.5 The probability of sustaining mTBI as a function of head acceleration and HIC. The Fig. is reprinted from [16].

4 mTBI due to recoil

The literature directly assessing the likelihood of soldiers obtaining mTBI due to gun recoil is very limited. At the time of writing the only studies that the author has found are the ones forming a joint collaboration between the University of Waterloo and the Division of Defense Research and Development Canada (DRDC). The work consists of one journal publication [17] and two master theses published in 2024 [12, 18]. There is also a master thesis, published in 2024, from Marshall University (USA) which deals with the same issue from a biological perspective [19]. In this chapter we will in detail go through the work as described in the journal publication (together with the two accompanying master theses), and briefly summarize the master theses from Marshall University.

4.1 T. Seeburrun - Assessment of brain response in operators subject to recoil force from firing long-range rifles

In the work by T. Seeburrun et. al. three volunteers from the Canadian army were equipped with a mouthguard and the motion of the mouthguard was measured when firing three different 0.50 caliber suppressed long-range sniper rifles. The recorded head motion was used as input in a finite element model, and the damage to the brain was calculated and compared with literature thresholds. A graphical summary of the journal publication is shown in Fig. 4.1

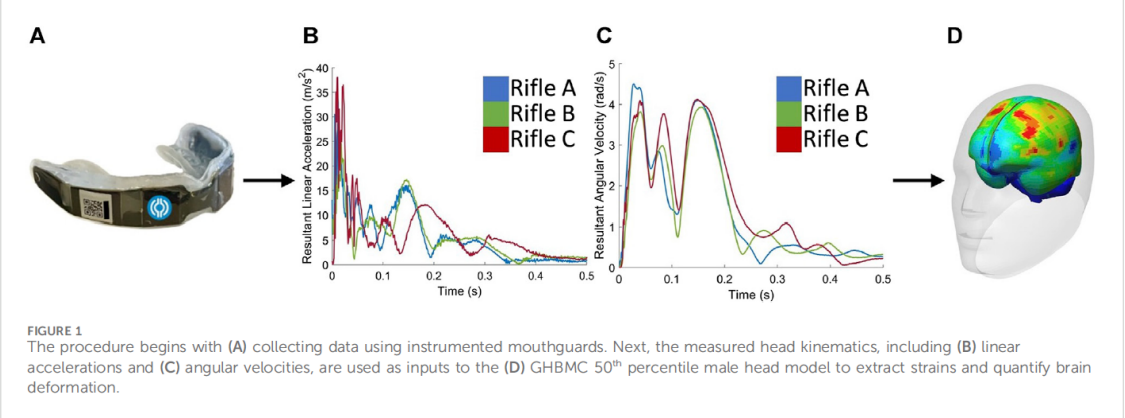


FIGURE 1
The procedure begins with **(A)** collecting data using instrumented mouthguards. Next, the measured head kinematics, including **(B)** linear accelerations and **(C)** angular velocities, are used as inputs to the **(D)** GHBMC 50th percentile male head model to extract strains and quantify brain deformation.

Figure 4.1 An overview of the method used to determine the risk of recoil-induced mTBI. The Fig. is reprinted from [17].

4.1.1 Head motion curves

The recorded head motion due to recoil is shown in Figs. 4.2 and 4.3. When adding together the x, y, and z components, the peak translational acceleration and rotational velocity were approximately $2.82\,g$ and $7.45\,\text{rad/s}$ respectively. We can easily compare the head motion due to recoil, with the mTBI-inducing head motion in sports shown in Fig. 3.1 and Fig. 3.2. In the sports data, for the cases were mTBI was detected the peak translational acceleration and peak angular velocity were $48 - 135\,g$ and $12.8 - 80.9\,\text{rad/s}$ respectively. Note that the translational acceleration due to recoil is a factor $10 - 100\,g$ lower than the translational acceleration due to a sports tackling, indicating that translational acceleration due to recoil is not the dominant motion behind potential

mTBI. The peak angular velocity due to recoil is also lower than the corresponding value due to a sports tackling, but only by a factor 1.7, if we consider the lowest peak angular velocity from a tackling that caused mTBI. Also note that the recorded rotational motion due to recoil is long (approx. 300 ms) compared to the recorded rotational motion due to tackling (25 ms). The duration of the head motion is obviously also important, and will manifest itself in the metrics which we will discuss later. The aforementioned observations indicate that if mTBI can occur due to recoil, then it is likely a consequence of the heads rotational motion and not the translational motion. In any case, the risk of recoil induced mTBI seems to be much lower than in sports.

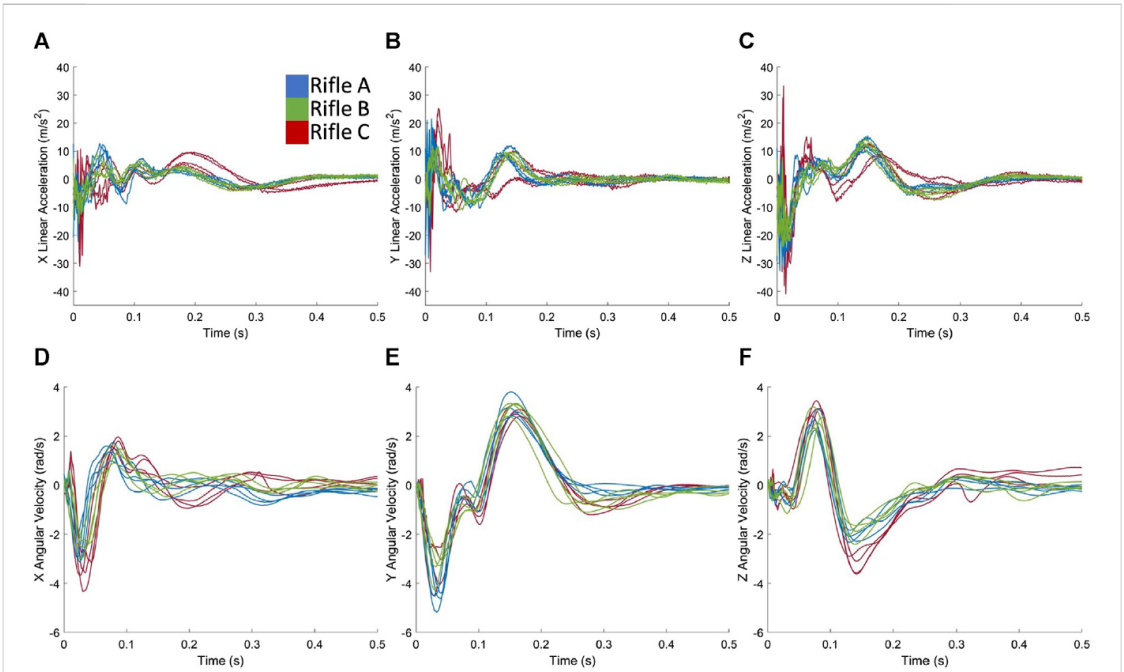


FIGURE 4
Exemplar case showing head kinematics **(A)** linear acceleration in X, **(B)** linear acceleration in Y, **(C)** linear acceleration in Z, **(D)** angular velocity in X, **(E)** angular velocity in Y, **(F)** angular velocity in Z for all shots taken by Volunteer 1 using rifles A, B and C.

Figure 4.2 An example of some of the recorded head motion. The Fig. is reprinted from [17].

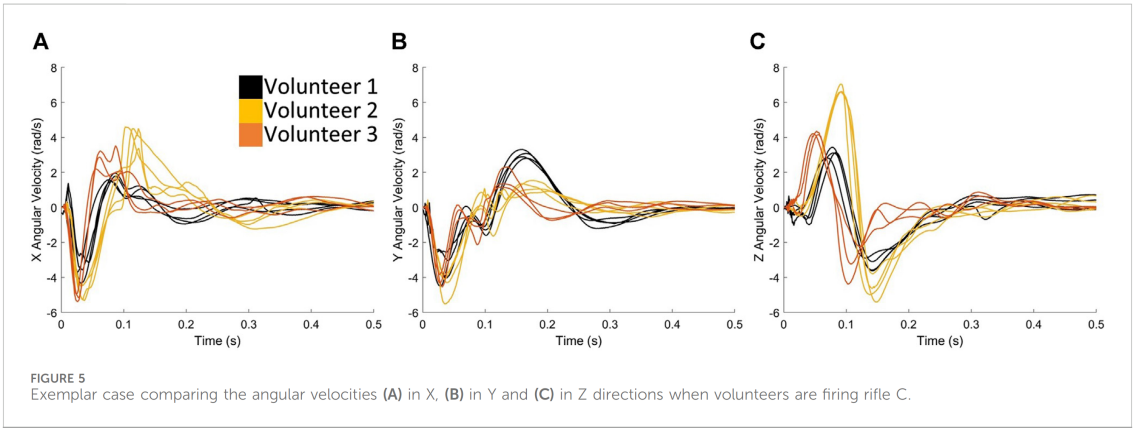


FIGURE 5
Exemplar case comparing the angular velocities **(A)** in X, **(B)** in Y and **(C)** in Z directions when volunteers are firing rifle C.

Figure 4.3 An example of some of the recorded head motion. The Fig. is reprinted from [17].

4.1.2 Kinematic based measures

In this study two measures based on kinematics were used: The head injury criterion (HIC_{15}) and the brain injury criterion (BrIC). Both of these metrics were discussed in Ch. 2. A summary of the HIC_{15} and BrIC values obtained in this paper is shown in Tab. 4.1. All of the HIC_{15} values are several orders of magnitude below the ones that have been reported to give mTBI in sports, see e.g. the HIC risk curves in Figs. 2.2 and 3.3. However, the BrIC values are more interesting, when we consider the risk curves shown in Fig. 2.3. The BrIC values that correspond to a 50% probability of AIS 1 and AIS 4 injuries, using the maximum principle strain based injury risk curve, were approximately 0.15 and 1.1 respectively. The values marked in bold in Tab. 4.1 correspond to BrIC values that may give an AIS 1 injury. In other words, if the risk curve is correct, then these data indicate that mild brain injuries may occur due to recoil.

Table 4.1 An overview of the HIC_{15} *and BrIC values measured in [17]. The values in bold are overlapping with the lowest reported mTBI thresholds.*

	Volunteer 1			Volunteer 2			Volunteer 3		
	Rifle A	Rifle B	Rifle C	Rifle A	Rifle B	Rifle C	Rifle A	Rifle B	Rifle C
Mean HIC_{15}	.1335	.0861	.1855	.2588	.1345	.2101	.1765	.0955	.1288
Mean BrIC	.1162	.1008	.1193	**.1858**	**.1474**	**.1963**	**.1616**	**.1523**	**.1509**

Again, the metrics presented here indicates that the translational accelerations observed in recoil are not large enough to produce brain injuries. Yet, the observed rotational motion are right on the edge of being sufficiently strong to cause mild brain injuries. From a physical point of view, rotations are considered more dangerous than translations because the brain has a very high bulk modulus compared to its low shear modulus. In simple terms, the brain tends to deform more under rotations than translations.

We should note that HIC_{15} may not be a suitable metric for assessing the likelihood of mTBI in the firearm recoil scenario. Firstly, the head motion seems to last 100-200 ms, which makes HIC_{15} very sensitive to high-frequency noise as it only considers 15 ms. Secondly, the HIC_{15} only accounts for linear acceleration, and ignores rotation. Also note that while BrIC is a standard criteria to use for rotational motion, it only takes into account the peak rotational velocity, not the

duration of the loading, which is an obvious oversight.

4.1.3 Finite element head model

In the paper, the head motion curves were used as input in a finite element code. The loading was imposed on a validated head model, which was developed by support from The Global Human Body Models Consortium (GHBMC). The head model was validated using cadaver data, but obviously does not take anatomical and physiological variations of individuals into account. The GHBMC model represents a 50th percentile male. The head model also includes 34 mm of the spinal cord, as it was extracted from a whole-body model. The skull of the head model was treated as rigid. The simulations were done in LS-DYNA.

The GHBMC model is shown in Fig. 4.4 and includes the following eight regions of the brain:

- **Cerebellum:** Does not initiate movement, but contributes to coordination, precision, and accurate timing. Uses input to produce fine-tuned motor activity.
- **Cerebrum gray matter:** Information processing. Sensation, perception, voluntary movement, learning, speech, and cognition.
- **Cerebrum white matter:** Provide communication between different grey matter areas, and between grey matter and rest of body.
- **Corpus callosum:** Convey information from one side of the brain to the other (e.g. from left to right frontal lobes).
- **Thalamus:** All information picked up from the senses (except smell) must be processed by the thalamus, before it is sent to the cerebral cortex for interpretation.
- **Brainstem midbrain:** Important functions in motor movements, particularly eye movement, and in auditory and visual processing.
- **Brainstem:** Regulate vital involuntary body functions such as breating and heart rate. Also helps with balance, coordination, and reflexes.
- **Basal ganglia:** Primarily responsible for motor control. Acts as a gate-keeping mechanism for the initiation of movement. Chooses which actions to allow and which to inhibit.

In the list above we have also included a small description of each brain regions function. We emphasize that the author is not a biologist, and as such only a simplified description is given.

In the literature, different head models have been used to estimate injury thresholds. Consequently, as is always the case, the output of the finite element code may vary when using different head models. It should be said that the head model employed herein seems reasonably validated, i.e. it does not contain obvious inaccuracies.

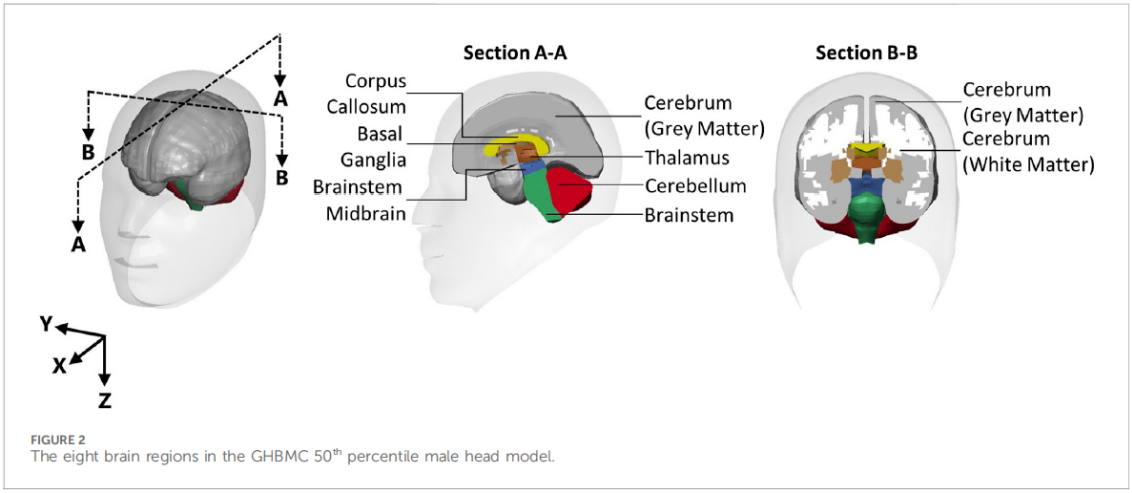
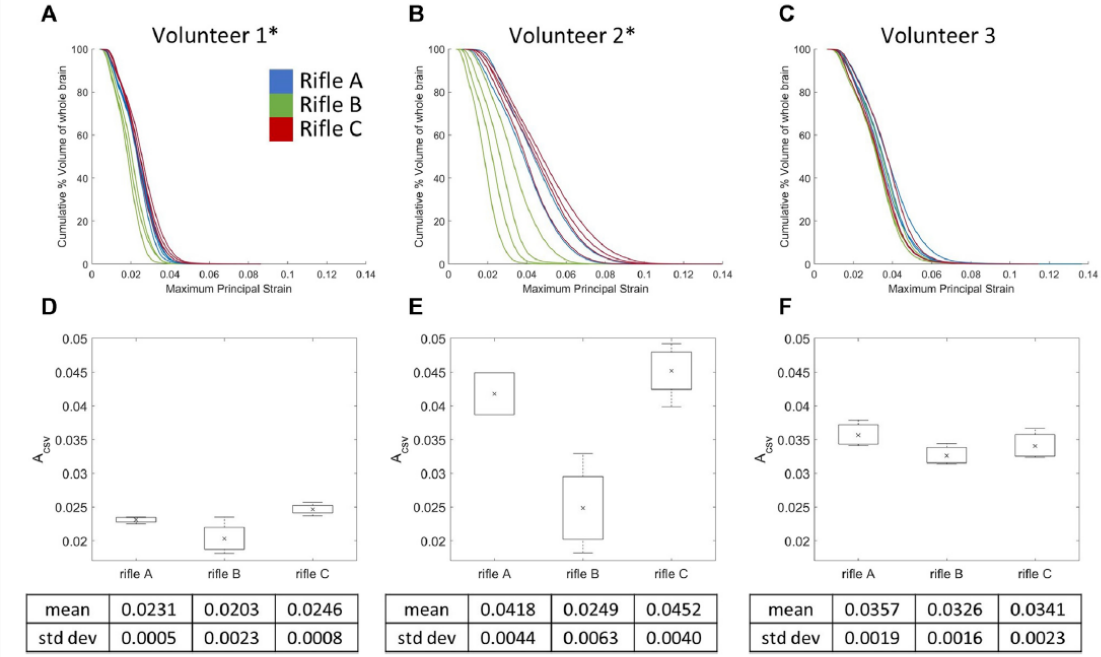


Figure 4.4 An overview of the GHBMC head model used for the accompanying finite-element simulations. The Fig. is reprinted from [17].

4.1.4 Strain distribution on entire brain

In this work two strain based metrics were used: 95th percentile of maximum principal strain (MPS_{95}) and the cumulative strain volume (CSV). In Fig. 4.5 and 4.6 the CSV curve is plotted as a function of maximum principle strain (MPS). In simplified terms, the curve represents the percentage of the brain which experiences strains larger than the specific MPS value. An estimate for the range of maximum principle strains that are considered injurious is 0.06 – 0.448, according to Rycman [20]. Most of the measurements in this work are smaller than this range, but the tails of volunteer 2 in Fig. 4.5 and rifle C in Fig. 4.6 reveals that a small percentage of the brain experiences strains that lie in the lower end of the injurious MPS interval. If the head model and data is correct this indicates that it may be possible to experience small dangerous strains depending on the shooter's biology, technique, and weapon.



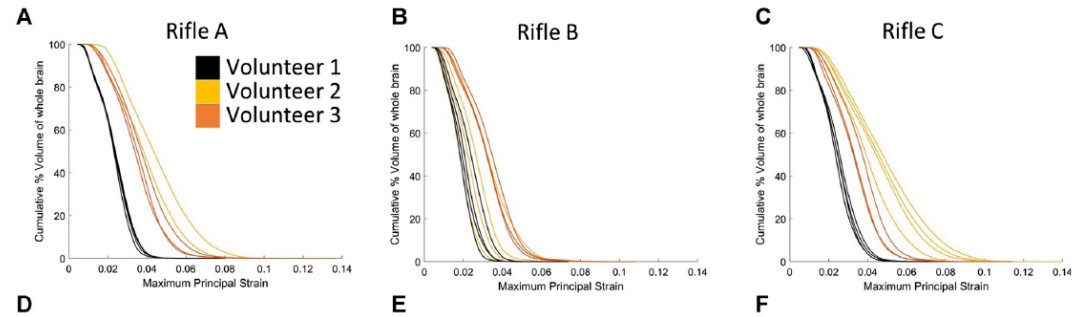
mean	0.0231	0.0203	0.0246
std dev	0.0005	0.0023	0.0008

mean	0.0418	0.0249	0.0452
std dev	0.0044	0.0063	0.0040

mean	0.0357	0.0326	0.0341
std dev	0.0019	0.0016	0.0023

FIGURE 9
Cumulative strain-volume curves for each shot across the three rifles for **(A)** Volunteer 1, **(B)** Volunteer 2 and **(C)** Volunteer 3. Mean A_{CSV} variation for all three rifles of **(D)** Volunteer 1, **(E)** Volunteer 2 and **(F)** Volunteer 3. * = statistically significant different detected between rifles. Number of trials for Volunteer 1 (rifles A:4, B:4, C:4), Volunteer 2 (rifles A:2, B:4, C:4) and Volunteer 3 (rifles A:3, B:3, C:3).

Figure 4.5 The CSV vs MPS curves obtained from the accompanying finite element simulations. The Fig. is reprinted from [17].



mean	0.0231	0.0418	0.0357
std dev	0.0005	0.0044	0.0019

mean	0.0203	0.0249	0.0326
std dev	0.0023	0.0063	0.0016

mean	0.0246	0.0452	0.0341
std dev	0.0008	0.0040	0.0023

FIGURE 10
Cumulative strain plots for each shot taken by all three volunteers using rifle A **(A)**, rifle B **(B)**, and rifle C **(C)**. Mean A_{CSV} variation for all three volunteers using **(D)** rifle A, **(E)** rifle B and **(F)** rifle C. Number of trials for Volunteer 1 (rifles A:4, B:4, C:4), Volunteer 2 (rifles A:2, B:4, C:4) and Volunteer 3 (rifles A:3, B:3, C:3).

Figure 4.6 The CSV vs MPS curves obtained from the accompanying finite element simulations. The Fig. is reprinted from [17].

4.1.5 Strain in individual brain regions

In Fig. 4.7 the area under the cumulative strain-volume curves are plotted for different regions in the brain. The magnitude of the strains depend on both the shooter's biology, technique, and weapon. In most cases, the corpus callosum experiences the highest strain. Hernandez [2] argues that strains in the corpus callosum are very sensitive to coronal rotations. The corpus callosum's primary function is to transfer information between the left and right brain, when strained this might result in symptoms similar to mTBI. In this work, we can group the regions into the following classes:

- **Highest strain:** Brainstem midbrain and Corpus callosum.
- **Medium strain:** Basal ganglia, Cerebrum gray, Thalamus, and Cerebrum white.
- **Lowest strain:** Brainstem and Cerebellum.

If the field is to be explored further, it is very important to determine which types of motion (and shooting positions) strains different regions of the brain.

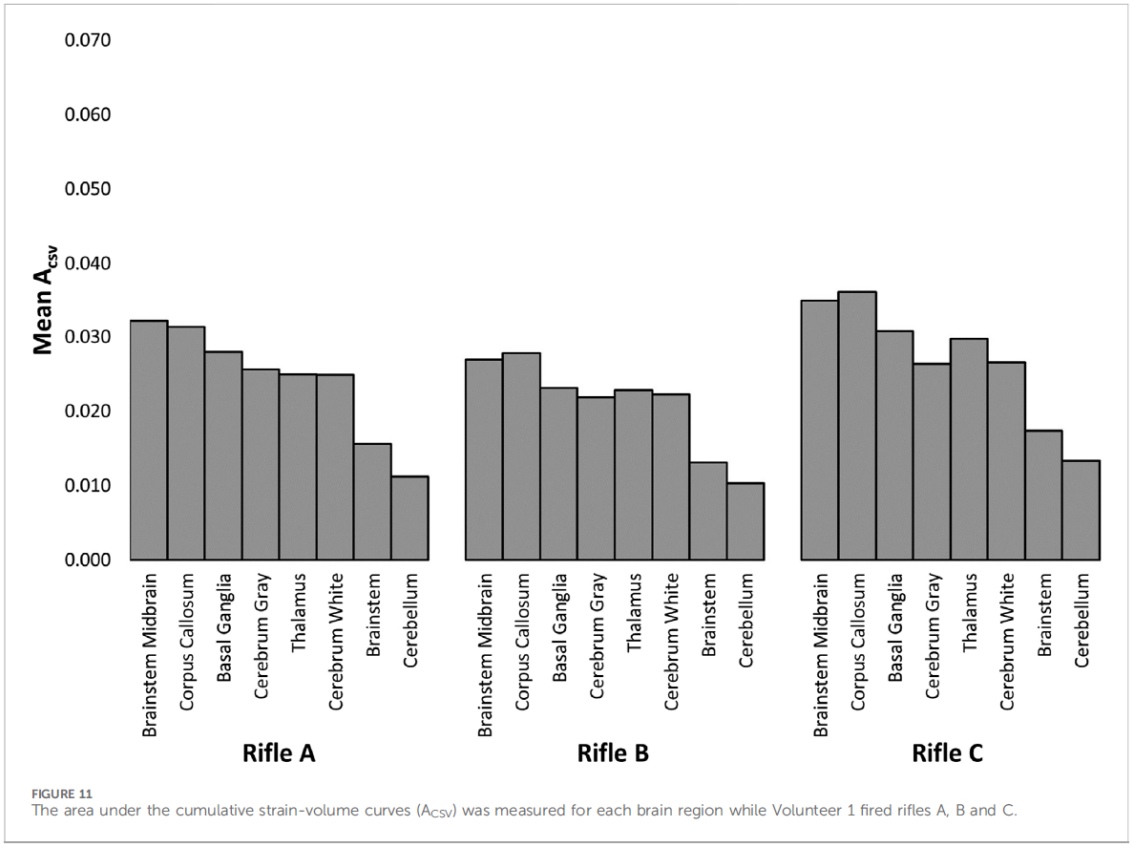


Figure 4.7 Fig. from.

4.1.6 Conclusion regarding mTBI

Both protective equipment and firing technique influence the magnitude of the strain induced on the brain during firing of long-range sniper rifles. Brain regions, such as the corpus callosum and brainstem midbrain exhibit the highest strain levels compared to e.g. the cerebellum which exhibits lower strains. Therefore certain regions may act as reliable indicators of mTBI.

If mTBI occurs due to recoil, then the dominant contribution is most likely the rotational motion and not the translational motion. In particular, coronal rotations are believed to be the most dangerous. The rotational kinematic and finite-element measures were typically slightly smaller or right on the edge of the mTBI threshold. Realistically we interpret this as that there is a small probability of inducing mTBI due to repetitive shooting over a long period. In addition, if the soldier already has undiagnosed mTBI (due to e.g. blast) it seems likely that a shooting session may induce symptoms. In fact, some members of the Canadian armed forces (CAF) have reported experiencing symptoms of mTBI, during repeated firing of long-range rifles.

4.2 Brief overview of the conclusion of L. D. Jones' thesis

The thesis' hypothesis is that exposure to rapid, repetetive recoil may cause physiological disturbances in the brain, which may result in mTBI. In this study volunteers were invited to perform trap and skeet shooting. Previous investigations indicate that peripheral serum glial fibrillary acidic protein (GFAP), ubiquitin C-terminal hydrolase-L1 (UCH-L1), and S100B show the proteins' ability to indicate and measure severity of mTBI. Therefore blood samples were collected 30 minutes pre-shooting, 30 minutes after shooting, and 24 hours after shooting. If the recoil force interferes with the neurometabolic function, then the shooter may be suceptible to mTBI. In total there were 11 volunteers, and 6 of them with detectable protein levels ($n = 6$). In blunt terms, there was a statistically insignificant increase in GFAP and UCH-L1 after shooting. Before rigorous conclusions can be drawn, a larger study should be performed where several other proteins are also measured. Nevertheless, this finding indicates that if mTBI occurs due to recoil, it is likely a small effect as discussed previously.

5 Summary and conclusion

In this literature study we have evaluated if repeated exposure to recoil can be a mechanism that induces mTBI in soldiers. In all of the openly published works that we have evaluated recoil-induced mTBI has not been directly observed. This may be either because the recoil force is not large enough to induce mTBI in the volunteers, or because the number of shots performed in each work was too small. Nevertheless, the recorded head motion does indicate that if recoil-induced mTBI exists then it is likely a consequence dominated by rotational motion and not translational motion. The physical explanation of this is that the brain's shear modulus is much lower than its bulk modulus, i.e. the brain tends to deform more under rotations than translations. We emphasize that some of the recorded rotational motion is actually overlapping with the lowest recorded thresholds that may cause mTBI. In addition, some members of the Canadian armed forces (CAF) have reported experiencing symptoms of mTBI, during repeated firing of long-range rifles. At the time of writing, it is unclear if these mTBI symptoms are due to shock loading, already were suffering from undiagonosed mTBI and the symptoms were triggered by shooting, or if the mTBI was induced purely by repeated firing of long-range rifles. The results presented here also indicate that the soldier's training and physiology play a role in reducing the risk of injury. If the field is to be explored further we recommend the following:

- A study with a large number of shooters performing one or several long shooting sessions, with suppressed high-recoil weapons, such as snipers. The soldiers head motion should be recorded simultaneously. There should also be a physician on-site to diagnose potential mTBI. This will give the foundation to perform a statistical analysis to determine the risk of mTBI due to recoil.
- In the works discussed here the recoil from a single shot has been used as input in the finite-element code. This does not take into account the cumulative effect of strain that may build up in the brain during a longer shooting session. It would therefore be interesting to impose periodic recoil loading, on the finite element head model, which correspond to repeated firings.

References

[1] CDC: Centers for Disease Control and Prevention (USA). Symptoms of mild tbi and concussion. https://www.cdc.gov/traumatic-brain-injury/signs-symptoms/index.html. Accessed: 15.08.2024.

[2] Fidel Hernandez, Lyndia C Wu, Michael C Yip, Kaveh Laksari, Andrew R Hoffman, Jaime R Lopez, Gerald A Grant, Svein Kleiven, and David B Camarillo. Six degree-of-freedom measurements of human mild traumatic brain injury. *Ann. Biomed. Eng.*, 43(8):1918–1934, August 2015.

[3] E. S. Gurdjian, V. L. Roberts, and L. M. THOMAS. Tolerance curves of acceleration and intracranial pressure and pressure and protective index in experimental head injury. *Journal of Trauma and Acute Care Surgery*, 6(5), 1966.

[4] T. B. Hoshizaki, A. Post, M. Kendall, J. Cournoyer, P. Rousseau, M. D. Gilchrist, S. Brien, M. Cusimano, and S. Marshall. The development of a threshold curve for the understanding of concussion in sport. *Trauma*, 19(3):196–206, 2017.

[5] Charles W Gadd. Use of a weighted-impulse criterion for estimating injury hazard. Technical report, SAE technical paper, 1966.

[6] Priya Prasad and Harold Mertz. The position of the united states delegation to the iso working group 6 on the use of hic in automotive environment. Technical report, SAE technical paper, 06 1985.

[7] M. Mackay. The increasing importance of the biomechanics of impact trauma. *Sadhana*, 32(4):397–408, Aug 2007.

[8] A. K. Ommaya. *Biomechanics of Head Injury: Experimental Aspects*. Advances in Neurotraumatology, 1985.

[9] E. G. Takhounts, M. J. Craig, K. Moorhouse, J. McFadden, and V. Hasija. Development of brain injury criteria (BrIC). *Stapp Car Crash J.*, 57:243–266, November 2013.

[10] Association for the advancement of automotive medicine. The abbreviated injury scale. https://www.aaam.org/abbreviated-injury-scale-ais/. Accessed: 20.08.2024.

[11] The Cochran Firm. The abbreviated injury scale. https://www.cochranfirm.com/new-orleans/injury-scale/. Accessed: 20.08.2024.

[12] T. Seeburrun. *Investigation of Brain Response in Canadian Armed Forces Volunteers Subjected to Recoil Force from Firing Long-Range Rifles Using Instrumented Mouthguards and Finite Element Head Mode*. Master thesis, University of Waterloo, Waterloo, Ontario, Canada, 2024.

[13] E. J. Pellman, D. C. Viano, A. M. Tucker, I. R. Casson, and J. F. Waeckerle. Concussion in professional football: reconstruction of game impacts and injuries. *Neurosurgery*, 53(4):799–814, 2003.

[14] Albert King, King Yang, Liying Zhang, and Warren Hardy. Is head injury caused by linear or angular acceleration? *IRCOBI Conference – Lisbon (Portugal)*, 01 2003.

[15] L. Zhang, K. H. Yang, and A. I. King. A proposed injury threshold for mild traumatic brain injury. *Journal of biomechanical engineering*, 126(2):226–236, 2004.

[16] J. R. Funk, S. M. Duma, S. J. Manoogian, and S. Rowson. Biomechanical risk estimates for mild traumatic brain injury. *51st annual proceedings association for the advancement of automotive medicine*, 51(PMC3217524):343–61, 2007.

[17] Tanvi Seeburrun, Michael C Bustamante, Devon C Hartlen, Austin Azar, Simon Ouellet, and Duane S Cronin. Assessment of brain response in operators subject to recoil force from firing long-range rifles. *Front. Bioeng. Biotechnol.*, 12:1352387, February 2024.

[18] J. A. Maldonado-Echeverria. *Brain Response to Overpressure and Recoil Loads from Discharge of Long-Range Precision Rifle*. Master thesis, University of Waterloo, Waterloo, Ontario, Canada, 2024.

[19] L. D. Jones. *Examining the impact of firearm recoil during a round of trap shooting on neurometabolic changes associated with traumatic brain injury*. Master thesis, Marshall University, Huntington, West Virginia, USA, 2024.

[20] A. Rycman, S. McLachlin, and D. Cronin. Spinal cord boundary conditions affect brain tissue strains in impact simulations. *Annals of Biomedical Engineering 51 (4)*, 783-793, 2023.

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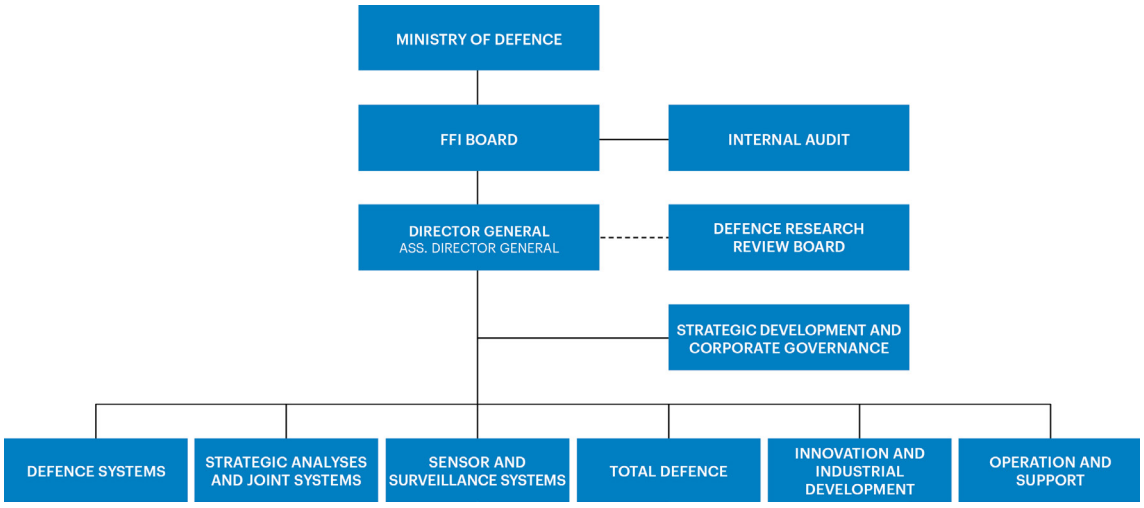

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