Experimental Head Injury in Monkeys —Concussion and Its Tolerance Level—

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Summary

The authors obtained the following results in this experiment in which monkeys were subjected to frontal and occipital blows with their heads in an unrestrained condition.

1) With respect to the impact to the head and the occurrence of concussion, translational acceleration is more important than rotational acceleration.

2) The concussion threshold of monkeys was an equal speed curve of 16 m/s for occipital blows, while in the case of frontal blows, a hyperbolic curve which is somewhat higher than that for occipital blows was obtained. This concussion tolerance curve was converted into the concussion tolerance curve for humans by means of dimensional analysis.

3) A close correlation was suggested between changes in the EEG amplitude and the severity of the concussion. The more severe the concussion, the greater the decrease in the EEG amplitude.

4) The concussions could be classified into three types pathologically. The first type showed capillary dilatation and vascular permeability. The second type showed hemorrhages at sites far from the cerebral cortex and the third type showed hemorrhages in the cerebral cortex. The second and third types were in parallel with the severity of the concussion.

Key words: Experimental head injury, concussion

Introduction

The authors performed studies on experimental head injuries by means of translational acceleration in 1976^{10,12)} and rotational acceleration in 1977,²¹⁾ and reported the mechanism of brain injuries and the limits of their incidence. In these two studies, concussions were generated in almost all cases and it was considered that the force of impact applied reached the concussion level.

In this work, the authors caused concussions in monkeys with their heads free to move by applying a milder impact of long duration and

investigated engineering, physiological and pathological items to determine the threshold of the concussions. In the experiment, cases with skull fracture were excluded because of the poor reliability of acceleration measurements and the major changes in physiological and pathological findings when the acceleration was maintained for even a short time after the fracture.¹⁾

Equipment

The experimental equipment consisted of an impactor system and a chair to restrain the monkeys. The impactor system was the same

recorded on EMG.

The blood pressure was measured continuously by means of a transducer (model MPV-0.5, Nihon Koden) connected to a catheter inserted into a femoral artery. These measured values were recorded on an electroencephalograph (EEG-4109) and a pen-writing oscillograph (RJG-3034, both made by Nihon Koden).

Starting within 10 seconds after the impact

Table 1 Resultant average acceleration (RAA) and velocity of monkey heads

Location of impact	n =	RAA (G)	Duration (msec)	Velocity (m/sec)
Frontal	31	140~1,100	1~8	6~34
Occipital	31	140~820	2~14	8~29

in most cases and thereafter, the corneal reflex, light reflex, ciliary reflex, blinking, eyeball movement and spontaneous movement of the extremities were checked periodically.

X-ray examinations of the skull and cervical spine were performed before and after the impact, skull measurements were performed and checks were made concerning skull fractures.

Autopsies were performed immediately after

Table 2 Resultant average angular acceleration (RAAA) and angular velocity (AV) of monkey heads

Location of impact	n =	RAAA (red/sec ²)	Duration (msec)	AV (rad/sec)
Frontal	26	$1 \sim 10 \times 10^4$	2~10	130~500
Occipital	29	$0.6 \sim 8 \times 10^4$	3~12	52~510

Table 3 Occipital impact without fracture

Impact No.	Acceleration		Apnea	Loss of corneal reflex	Bradycardia	Depression of EEG activity	Concussion grade
	average	duration					
11	180 (G)	4.6 (msec)	0	16"	—	—	0
12	350	3.3	0	18"	—	—	0
13	320	4.3	0	30"	—	—	I
14	370	4.9	0	34"	—	—	I
16	400	3.1	0	11"	—	—	0
17	650	3.2	8"	22"	—	—	I
18	260	9.9	50"	15"	—	—	I
19	510	3.3	26"	29"	—	—	II
20	430	4.7	30"	13"	—	—	I
21	370	4.9	36"	12"	+	—	II
22	290	3.9	32"	13"	—	+	I
23	550	4.8	70"	9"	+	++	II
44	230	6.9	0	5"	—	+	0
45	270	6.6	0	30"	—	++	I
46	310	8.1	0	8"	—	++	0
47	540	4.8	5"	15"	—	++	0
48	830	4.0	∞	110"	—	++	II
50	430	5.4	6"	28"	—	—	I
51	520	4.2	10"	15"	+	++	I
52	600	5.2	24"	33"	+	+	III
56	140	12.2	0	19"	—	+	0
57	140	12.3	10"	33"	—	—	I
58	220	13.1	0	18"	—	—	0
59	235	14.0	0	30"	—	+	I
60	255	12.6	20"	14"	+	—	II
62	510	2.2	0	10"	—	—	0
63	680	2.4	19"	10"	+	—	I
70	140	13.5	0	11"	—	—	0
71	140	5.8	0	6"	—	—	0
72	180	6.9	0	10"	—	—	0

death on the animals which died and after sacrificing by means of pentobarbital within 1–24 hours after the impact among the surviving monkeys (except for one animal after one week). The brains and cervical cords were fixed in 10% formalin and after about four weeks, slices were prepared at sites where macroscopic changes were found, as well as in the frontal, parietal and occipital lobes, the upper pons, medulla and cervical cord, and were stained by hematoxylin-Eosin and Klüver-Barrera. Twelve of the 18 monkeys were injected with 10 mg/kg of Evans blue before or just after the impact.

Results

I. Engineering measurements and concussion evaluations

The number of blows applied to 18 monkeys

was 74 in total and the measurement of the head acceleration (resultant averaged) was performed effectively in a total of 62 cases, 31 for frontal impact and 31 for occipital impact.

In the case of frontal impact, the resultant average acceleration and the duration were 140–1,100 G and 1–8 milliseconds respectively. In the case of occipital impact, these values were 140–820 G and 2–14 milliseconds.

The resultant average angular acceleration was 6,000–12,000 rad/sec² and the duration was 2–12 milliseconds for both frontal and occipital blows.

These blows did not result in skull fractures in 14 monkeys and of these, five died. Skull fractures were found in four monkeys and three of these died.

Concussions were evaluated by three physiological responses: respiration, bradycardia

Table 4 Frontal impact without fracture

Impact No.	Acceleration		Apnea	Loss of corneal reflex	Bradycardia	Depression of EEG activity	Concussion grade
	average	duration					
3	200 (G)	1.5 (msec)	5"	10"	—	—	0
4	440	3.9	5"	19"	—	—	0
5	350	3.0	5"	22"	—	—	1
6	380	3.6	8"	8"	—	—	0
7	480	2.5	5"	16"	—	—	0
8	670	1.5	15"	13"	—	—	0
9	490	3.1	3"	10"	—	—	0
10	600	2.3	15"	16"	—	—	0
25	460	5.2	0	12"	—	—	0
26	1040	1.5	30"	30"	+	++	III
27	1000	1.0	47"	110"	—	++	II
28	290	5.4	0	15"	—	—	0
29	175	6.4	2"	20"	—	+	I
31	570	3.7	0	10"	—	—	0
32	580	3.7	0	16"	—	—	0
33	710	4.1	5"	20"	—	—	I
36	330	4.7	8"	15"	—	—	0
37	370	5.4	8"	12"	—	—	0
38	360	6.8	10"	10"	—	—	0
39	320	6.7	4"	9"	—	—	0
41	450	5.5	0	20"	—	—	I
43	650	3.3	20"	23"	+	++	III
65	690	5.0	10"	10"	—	—	0
66	760	2.7	2"	10"	+	+	I
67	420	6.4	5"	11"	—	—	0
68	790	2.2	2"	10"	+	—	I
73	130	7.8	6"	12"	—	—	0
74	250	8.3	40"	13"	—	++	I

Fig. 3 Correlation between concussion severity and resultant average acceleration (RAA) vs duration.

Fig. 4 Correlation between concussion severity and resultant average acceleration (RAA) vs duration.

and the corneal reflex. There were three severity factors of concussion, i.e. apnea, disappearance of the corneal reflex for more than 20 seconds and a drop in the pulse rate within one minute after the blow to less than 75% of that before the blow. The severity of the concussion was divided into four levels: Grade 0 in which none

of these factors were found, Grade I in which one of these three factors were present, Grade II in which two factors were present and Grade III in which all were observed.

Excluding the blows which caused skull fractures, the incidence of Grade 0 to Grade III concussions in cases of frontal blows in which

Fig. 5 Concussion tolerance curve in man.

acceleration measurements were effective was 18, 6, 1 and 2 for each of the grades respectively. For the occipital blows, these figures were 11, 12, 6 and 2.

Concussions of Grades II and III tended to occur when the resultant averaged acceleration was higher or the duration was longer (Table 3). It is possible to assume that the equal rate curve at about 16 m/s is the boundary line between the Grade 0 non-concussion group and the Grades II and III severe concussion group in the case of occipital blows (Fig. 3).

In the case of frontal blows, the non-concussion and severe concussion groups can be divided by the hyperbolic curve which is at a

position slightly higher than that for the occipital blows. The authors consider that these are the concussion tolerance curves of blows to the head in monkeys.

These concussion tolerance curves were converted to human skull acceleration vs. duration by dimensional analysis. These were represented by hyperbolic curves of similar waves showing 320 G at 1 millisecond, 180 G at 3 milliseconds and 100 G at 6 milliseconds for both frontal and occipital blows. However, there was no correlation between the angular acceleration of the head and the duration and severity of the concussion.

There was a significant correlation between

Fig. 6 Correlation between EEG amplitude and resultant average acceleration vs duration.

Table 5 Pathological changes in monkeys without skull fracture

Outcome	Monkey No.	Location of impact	Concussion severity	Injection of E. blue	Hemorrhage and Evans blue stain												Cervical cord	SAH	SDH
					F. lobe		T. & P. lobe		O. lobe		Midbrain & Pons			Medulla					
					cortex	subcortex	cortex	subcortex	cortex	subcortex	Cerebellum								
Survived	A	F	0	(-)															
	B	F	I	(-)													(+)		
	C	F	I	(-)												H. (microscopic)			
	D	0	I	(+)		E.B.						E.B.							
	E	0	II	(-)															
	F	0	II	(-)															
	G	F	III	(+)		H E.B.	E.B.			H E.B.					E.B.	(+)			
	H	0	III	(+)		H						E.B.			H	(+)	(+)		
Fatal	I	F	II	(-)												H			
	J	F	III	(+)		E.B.						E.B.				H			
	K	0	III	(+)		H E.B.		E.B.		E.B.		E.B.	E.B.		E.B.	(##)			
	L	0	III	(+)		H				E.B.		E.B.	E.B.	H	H E.B.	(##)	(+)		

H: hemorrhage

E.B.: Evans blue stain

changes in EEG amplitude and the acceleration duration.

Changes in the amplitudes of the EEG within two minutes after the impact could be categorized into three classes: (1) the steady class with no changes in amplitudes after impact, (2) the mild class showing a mild decrease in the amplitude after impact, and (3) the severe class with great decrease in the amplitude. These were plotted on the acceleration vs. duration graph (Fig. 6). The boundary between the steady class and the severe class for both frontal and occipital blows could be drawn with a hyperbolic curve about the same as that of the concussion tolerance curve on frontal impact. There was no significant correlation between the EEG frequency and the concussion severity.

II. Pathological findings

Among the 14 monkeys with no skull fractures, autopsies were performed on 12. Eight of these 12 monkeys survived and the remaining four died due to respiratory disturbances after the impact. The degrees of concussion in these 12 monkeys included one in Grade 0, three in Grade I, three in Grade II and five in Grade III. Intracerebral and cervical cord hemorrhages were found in seven of the 12 animals.

The incidence of cerebral hemorrhages was three among 12 monkeys, and the location of the hemorrhage was in the gray matter which was considered to be a contrecoup or coup injury. The microscopical findings included diffuse hemorrhages, ring hemorrhages surrounding the vessels, capillary dilatation with congestion, and extravasation of white blood cells. There was a decrease in staining in part of the white matter near the hemorrhagic sites.

One of the monkeys had a hemorrhage in the cerebral white matter. Microscopically, this was a petechial hemorrhage and vascular dilatation. In another monkey, a hemorrhage was found in the medulla oblongata. This was a concentration of petechial hemorrhages in the vicinity of the dorsal motor nucleus and vestibular nucleus. There were also petechial hemorrhages along the vessels.

Hemorrhages of the cervical cord were found in five monkeys. All of these hemorrhages were centered in the gray matter, which ranged

from ring hemorrhages around the vessels to concentrations of petechial hemorrhages. One animal showed only capillary dilatation with congestion.

Subarachnoidal hemorrhages were found in five of the 12 monkeys and subdural hematomas in two of the 12 monkeys.

Evans blue was injected into the femoral artery in six of the 12 monkeys before or just after the impact, and in all these monkeys, Evans blue staining was found at one or more sites in the cerebral cortex, white matter, cerebellum, midbrain, pons, medulla and cervical cord. Preferential sites of staining were contused cerebral cortex, deep white matter, the corpus striatum, and the medial lemniscus of the midbrain and pons. Such hemorrhages and Evans blue staining were more frequently found in higher concussion grades.

Discussion

I. Concussions and impact forces

Since Holborn⁹⁾ suggested that shear strain

Fig. 7 Cortical contusion.

Fig. 8 Hemorrhage in upper cervical cord.

Section through the optic chiasm

Section through the upper pons

Section through the upper cervical cord

Fig. 9 Site of Evans blue stain.

caused brain injuries, it had been stressed that rotational acceleration contributes to brain injuries. Ommaya et al.¹⁷⁾ estimated the tolerance curve of concussions from acceleration duration in whiplash injury experiments in monkeys.

Denny-Brown²⁾ reported that translational acceleration was important in the occurrence of concussions. Gurdjian et al. determined the acceleration-time tolerance level in humans by means of frontal impact experiments using human cadavers based on the clinical experience that an injury causing a linear fracture is accompanied by an unconscious state.

In the present experiment, the authors found that the concussion severity is closely correlated with the tangential acceleration. In translational acceleration impact experiments performed in 1976, concussions not accompanied by cerebral injury were found

in cases of almost pure translational acceleration. According to Segawa et al.,²⁰⁾ translational acceleration is more important in the generation of concussions than rotational acceleration.

The concussion tolerance curves in man estimated by the authors closely agreed with the acceleration-time tolerance levels in humans of Gurdjian et al.⁷⁾ and the skull fracture tolerance curve of Masuzawa.¹¹⁾

II. Concussions and physiological changes

No final conclusions have been reached concerning whether the main factor involved in loss of consciousness due to injuries is a brain stem disorder, a disorder in the cerebral hemispheres or both. However, the evaluation of concussions in experimental head injuries in many cases is based on brain stem functions such as the disappearance of the corneal reflex, respiratory disturbances, pulse rate and changes in the blood pressure.

Meyer et al.¹³⁾ reported a decrease in EEG amplitude for 10–80 seconds during concussions; the decrease in amplitude and the appearance of delta waves continued for 10–160 seconds in cases of severe concussions. Foltz et al.⁵⁾ also found decreased electrical activity in the cerebral cortex in most cases of concussion in experiments on acceleration concussion using cats and monkeys. They also found parallel decreases in the subcortical and reticular formation electrical activity recorded simultaneously, and this electrical depression was maintained longer in the reticular formation than in the cortex or subcortex.

The authors also noted decreased electrical activity in many of the EEG waves from the parietal regions of monkeys. The threshold of this decrease was drawn as a hyperbolic curve on an acceleration vs. duration graph which closely matched the concussion tolerance curve at the time of frontal impact. In the EEG of the Grade III concussion severity group in particular, the amplitude decrease was classified as severe in two cases and mild in one. It is assumed that there was diffuse nerve cell dysfunction in the cerebral hemisphere at the time of the concussion.

Sano¹⁹⁾ suggested that alpha-suppression occurs during concussions and that this resulted in the decreased amplitude, but Foltz

et al.⁵⁾ did not find any correlation between concussions and frequency. In the authors' experiment, there was alpha-rhythm during the decreased activity and there was no correlation between the concussion and the EEG frequency.

III. Concussions and pathological findings

The authors considered concussion as a syndrome of the clinical level, in accordance with the definition of concussion by the Committee on Terminology in Neurotraumatology of the World Federation of Neurosurgical Societies¹⁾ and evaluated concussions in the experiment in accordance with the neurological responses. The pathological findings in monkeys showing concussion in the experiment could be divided into three types.

In the first type, there were no hemorrhages in the cerebral parenchyma or the cervical cord, but there was dilatation of the capillaries in the cerebral parenchyma and increased vascular permeability as seen by Evans blue staining. This type was seen in monkey D (Table 5).

These findings are more often seen in deeper structures such as the white matter, basal ganglia and medial lemniscus than in the cerebral cortex.

The second type showed hemorrhages along the blood vessels or perivascular hemorrhages at locations such as the deep white matter, the medulla oblongata and the cervical cord. Typical examples of this type are monkeys K and L.

Type 3 showed hemorrhages in the gray matter in the region of contrecoup or coup injuries as seen in monkey G.

The minimum changes seen pathologically at the time of a concussion are said to be chromatolysis and vacuolation.^{2,3)} Gurdjian⁸⁾ found chromatolytic changes in the lateral and medial reticular formation of the midbrain, pons, medulla oblongata and cervical cord of monkeys and dogs with concussions. These locations agreed well with the sites where the authors found Evans blue staining.

In mild concussions, why are these pathological changes seen at sites comparatively deep within the brain tissue? Evans et al.³⁾ reported that energy at the time of an injury causes paralysis of the capillaries and venules and as a

result, vascular permeability is exacerbated and disturbances in the cerebral blood flow such as brain edema occur.

Nakamura and Shimizu¹⁶⁾ reported that the vessels of the skull base are stretched by displacement and deformation of the brain as a whole due to impact, and that a gap occurs between the nerves and the vessels and this causes neurovascular friction which results in circulatory disturbances, hemorrhagia per diapedesis and hemorrhagia per rexin.

The lenticulostriate arteries and paramedian arteries distributed in the medial part of the basal ganglia, pons and midbrain are more susceptible to neurovascular friction than other vessels because of their courses. Therefore, even when rotational shear strain is not applied, i.e. when there is translational acceleration, circulatory disturbances occur in the brain stem due to neurovascular friction and this can be considered to lead to changes of the first type.

The second type was seen in two cases with hemorrhages in the white matter and the medulla, and both cases died.

In clinical cases of hemorrhages in the white matter, basal ganglia or brain stem, Evans et al.⁴⁾ noted that these hemorrhages were hemorrhagia per diapedesis and Mosberg et al.¹⁴⁾ noted that they were hemorrhagia per rexin.

Hemorrhages seen in the cervical cord are both perivascular ring hemorrhages which are considered to be hemorrhagia per diapedesis and petechial hemorrhages in groups which are considered to be hemorrhagia per rexin. In the basal ganglia, pons and midbrain, no hemorrhages have been found although there were occasions of increased vascular permeability.

The shear strain theory should be suitable to explain the parenchymal distribution of hemorrhages. The site of the most severe shear strain at the time of impact was the cervical cord and hemorrhages were most apt to occur there. This was followed by the cerebral white matter and the medulla oblongata. No hemorrhages occurred in the basal ganglia, midbrain or pons where the shear strain was the smallest. This suggests that the second type of change is very closely connected with shear strain.

The third type of change involves hemorrhages indicating contrecoup or coup injuries which occur in the cerebral cortex.

Sekino et al.²²⁾ found cerebral contusions in the CT scans of 7.6% of clinical patients who lost consciousness within 10 minutes after an injury. Thus, the possibility of pathological contusions accompanying a concussion has been shown experimentally and clinically.

When the concussion severity is compared according to these three types of pathological classification in Grade I, there are only changes of the first type in the pathological classification and/or changes of hemorrhagia per rexin found microscopically. Cases of the second and third types with macroscopic hemorrhages all had concussions of Grade II or III. Therefore, the pathological changes and concussion severity were in good agreement.

The localization of pathological changes in brain concussions can be explained by the theory of primary brain stem disturbances or by the theory of damage to the brain as a whole including the brain stem.

Gurdjian et al.⁶⁾ reported from experimental and clinical observations that cerebral concussion is the result of derangement in the function of the brain stem. Ommaya et al.¹⁸⁾ formed the hypothesis from the results of experimental head injuries that varying degrees of cerebral disconnections proceeding from the cortex inwards to the mesencephalic-diencephalic core result in the syndromes of cerebral concussion including confusion, amnesia and coma.

In the authors' experiment, pathological findings such as hemorrhages or circulatory disturbances often seen in the midbrain, pons, medulla oblongata and cervical cord indicate that the pathological changes responsible for concussions occur in the brain stem and cervical cord. However, a correlation was suggested between the decreased amplitude in EEG findings and the concussion severity, and Evans blue staining was also found in the cortex, white matter and basal ganglia. Therefore, disturbances in the cerebral hemispheres can not be ruled out as changes which cause a concussion. This is a problem for future research.

Conclusions

The following results were obtained when monkeys were subjected to frontal and occipital blows with their heads unrestrained.

1) With respect to the impact to the head and the occurrence of a concussion, translational acceleration is more important than rotational acceleration.

2) The concussion threshold of monkeys was an equal speed curve of 16 m/s for occipital blows, while in the case of frontal blows, a hyperbolic curve which is somewhat higher than that for occipital blows was obtained. This concussion tolerance curve was converted into the concussion tolerance curve for humans by means of dimensional analysis.

3) A close correlation was suggested between changes in the EEG amplitude and the severity of the concussion. The more severe the concussion, the greater the decrease in the EEG amplitude.

4) Concussions could be classified into three types pathologically. The first type showed capillary dilatation and vascular permeability. The second type showed hemorrhages at sites far from the cerebral cortex and the third type showed hemorrhages in the cerebral cortex. The second and third types were in parallel with the severity of the concussion.

5) The responsible regions for concussion were assumed to be in the brain stem and the upper cervical cord from pathological findings. However, the cerebral hemisphere was not neglected as a responsible region because of decreased amplitude in EEG.

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