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CORRELATIVE STUDIES OF DYNAMICS AND PATHOLOGY IN WHIP-LASH AND HEAD INJURIES[1]

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ABSTRACT. The authors have been doing experimental studies on the human dummy, the monkey and the monkey dummy to investigate correlation between dynamics and pathology. The dummy and the animal were fixed on the specially designed sled and the sled collided against the concrete barrier with various velocities. The linear and angular acceleration and other dynamic factors and the intracranial and the intraspinal pressure at the moment of impact were measured. These were correlated with pathological changes in the brain and the cervical cord of the monkey. Dynamic data obtained were in fairly good accordance with the equations deduced theoretically based on simplified models. The brain tolerance curves, here presented, in cases of the linear and the angular acceleration, may serve to evaluate the prognosis of cerebral functions for rehabilitation and to prevent possible injuries caused by head trauma and whip-lash mechanisms.

Changes of the intracranial and the intraspinal pressure at the time of whip-lash movement or of impact to the head will be estimated from theoretical equations based on simplified models.

The authors have been collaborating with Prof. Tsuyoshi Hayashi and his staff, Department of Aeronautics, Faculty of Engineering, University of Tokyo, to investigate correlation between dynamics and pathology in cases of head injuries and whip-lash injuries. This paper will deal with the results of this cooperative study.

1. LINEAR (TRANSLATIONAL) ACCELERATION OF THE HEAD AND THE INTRACRANIAL PRESSURE

It is assumed that a one-dimensional system composed of a vessel (skull) and an internal elastic fluid (brain) and a linear spring representing the elastic properties of the skull, the scalp with hair, and a helmet, if present, as well as the elasticity of the barrier will collide against a barrier, as shown in Fig. 1 (2). It is also assumed that the vessel has a uniform cross-section, is a rigid body, massless, and that the internal fluid (brain) has the elastic constant E, the internal cross-sectional area A, the length l, the density ϱ and that the above-mentioned linear spring has the elastic constant k. The elasticity of the barrier may be included in the k, then the barrier itself may be considered a rigid body. Thus the problem is reduced to solve the impact problem when the system will strike the rigid barrier with an initial velocity v_0.

Fundamental equations and the general solution are described in Fig. 1. The pressure in the fluid is expressed by $P(x, t)$. According to the relative stiffness ratio (kl/EA) of the cushion elasticity (k) to that of the fluid (EA/l), there may be the hard impact $(kl/EA \gg 1)$ and the soft impact $(kl/EA \ll 1)$, as shown in Fig. 2.

In case of the hard impact, repetition of shock waves travelling with the sound velocity c occurs and if the pressure $(\varrho c v_0)$ reaches the cavitation pressure p^* (probably -1 kg/cm^2) cavitation will be produced throughout the brain, causing severe brain damage. As this velocity $v_0^* = -p^*/c\varrho$ is pretty low, there is a possibility that such a hard impact may occur if an unprotected head be hit by a very rigid object.

In case of the soft impact, there will be pressure gradient as seen in Fig. 2. The maximum pressure distribution

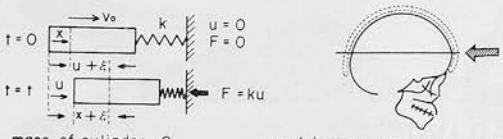
$$P(x, t) = -\frac{v_0}{2}\sqrt{\frac{\varrho k l}{A}}\left(1 - \frac{2x}{l}\right)$$

takes place at the time

$$t_1 = \frac{\pi}{2}\sqrt{\frac{\varrho A l}{k}}$$

[1] Read at the International Symposium on Rehabilitation in Head Injury, Göteborg 1971. Problem Commission of Physical Medicine and Rehabilitation of the World Federation of Neurology.

Simplified cylindrical model



mass of cylinder = 0
ρ density of fluid
A section area of cylinder
ℓ length of cylinder
K linear spring constant
Vo initial velocity of cylinder
u = u(t) displacement of cylinder
F repulsion from rigid wall
ξ relative displacement of section X to the left end of cylinder
E bulk modulus of fluid
$C = \sqrt{E/\rho}$ sound velocity in fluid
P(x,t) pressure in fluid at time t and at displacement x

The equation of motion of fluid particle

$$\frac{\partial}{\partial t^2}(u+\xi) = C^2 \frac{\partial^2 \xi}{\partial x^2} \qquad (0 \le x \le \ell)$$

boundary condition $\xi(0,t) = \xi(\ell,t) = 0$

$$F = ku = EA\left(\frac{\partial \xi}{\partial x}\right)_{x=0} - EA\left(\frac{\partial \xi}{\partial x}\right)_{x=\ell}$$

initial condition $\xi(x,0) = 0 \quad u(0) = 0$

$$\left[\frac{\partial}{\partial t}(u+\xi)\right]_{t=0} = V_0$$

General solution

$$P(x,t) = -4\rho c V_0 \sum_{n=1}^{\infty} \frac{\sin \omega_n}{[2\omega_n + \sin(2\omega_n)]} \cdot \sin \omega_n \left(1 - \frac{2x}{\ell}\right) \sin \frac{2c\omega_n t}{\ell}$$

$$\omega \cdot \tan \omega = \frac{k\ell}{4EA} \quad \text{(characteristic equation)}$$

Fig. 1

The maximum acceleration or deceleration a will occur at $t = t_1$.

$$a = \left|\frac{\partial^2 u}{\partial t^2}\right| = V_0 \sqrt{\frac{k}{\rho A l}}$$

Then

$$P(x,t) = -\tfrac{1}{2} a\rho l \left(1 - \frac{2x}{l}\right)$$

shows that the coup pressure is $\frac{1}{2} a\rho l$ and the contre-coup pressure is $-\frac{1}{2} a\rho l$.

The interesting thing is that the same results on the dynamical properties can be deduced under the assumption that a constant acceleration with the magnitude a is suddenly applied to the system for the time interval $2v_0/a$. Hayashi called this a constant acceleration theory (2).

At some value of a, the contre-coup pressure reaches the cavitation pressure p^* and the cavitation will occur in the contre-coup region.

$$p^* = -\tfrac{1}{2} a^* \rho l$$

We obtain the critical acceleration $a^* = -2p^*/\rho l$.

For the acceleration greater than a^*, the contre-coup pressure remains to be constant p^*, and the coup pressure is expressed as

$$\rho l(a - \tfrac{1}{2}a^*).$$

This is just like the curves drawn in Fig. 12.

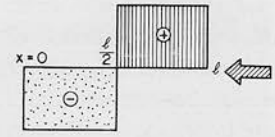
In practice, we encounter mostly the soft impact. Besides, the intracranial pressure distribution must be modified by the spinal canal, especially of the cervical spine, as illustrated in Fig. 3 (3). Two examples are presented in Fig. 4. These theoretical equations are in accordance with the experimental data described below.

As can be seen from the figure, the occipital blow with acceleration of 100 g is enough to produce cavitation pressure (-1 kg/cm^2) in the frontal pole, whereas the frontal blow with acceleration of 130 g is necessary to produce cavitation in the occipital pole. This is in accordance with the fact that the occipital blow is more apt to produce contre-coup injury than the frontal blow.

In case of the temporal impact, modification by the spinal canal is negligible, therefore, if we denote the lateral breadth of the skull b, the coup pressure will be $\frac{1}{2}\rho ab$ and the contre-coup pressure will be $-\frac{1}{2}\rho ab$, for a lateral acceleration a. Therefore, the frequency of occurrence of coup injury is equal to

Hard impact ($k\ell/EA \gg 1$)

$$P(0,t) = -\frac{4}{\pi}\rho c V_0 \sum_{1}^{\infty} \frac{1}{2n-1} \cdot \sin \frac{(2n-1)c\pi t}{\ell}$$



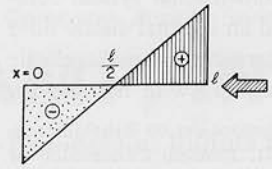
$$t_1 = \frac{\ell}{2c}$$

$$|P_{max}| = \rho c v_0$$

$$|a_{mean}| = 2\frac{cv_0}{\ell}$$

Soft impact ($k\ell/EA \ll 1$)

$$P(x,t) = -\rho c V_0 \omega_1 \left(1 - \frac{2x}{\ell}\right) \sin \frac{2c\omega_1 t}{\ell}$$



$$t_1 = \frac{\pi}{2}\sqrt{\frac{\rho A \ell}{k}}$$

$$|P_{max}| = \frac{1}{2}\rho \ell V_0 \sqrt{\frac{k}{\rho A \ell}}$$

$$|a_{mean}| = \frac{2}{\pi} V_0 \sqrt{\frac{k}{\rho A \ell}}$$

$$\left|\frac{\partial^2 u}{\partial t^2}\right|_{max} = V_0 \sqrt{\frac{k}{\rho A \ell}}$$

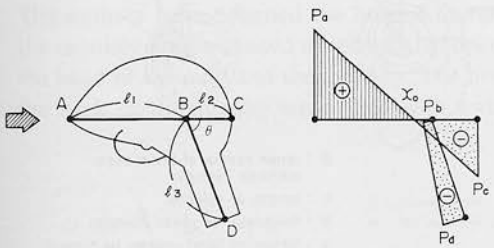
Fig. 2

that of contre-coup injury. In a series of 63 autopsies, the authors found that in cases of the frontal impact, coup lesions were found in 93% and contre-coup lesions were found in 21%, whereas in cases of the occipital impact, coup lesions were noticed in 17% and contre-coup lesions were found in 90%. In cases of the temporal impact, coup lesions and contre-coup lesions were of equal frequency, 60% respectively.

2. ANGULAR (ROTATIONAL) ACCELERATION OF HEAD AND NECK

Theoretical equations are presented in Fig. 5. As can be seen from the example of Fig. 5, in cases of whip-lash injuries, acceleration of superficial parts of the brain will be increased as compared with that in case of simple linear acceleration applied to the centre of gravity, according to the length of r_F. For example if r_F is 20 cm, acceleration of the impact point becomes 84% larger than that of the centre of gravity.

Intracranial pressure gradient by head impact (Gx)



$$P_A = \frac{1}{2}\alpha\rho\ell(1+\alpha)$$

$$P_B = -\frac{1}{2}\alpha\rho\ell\left(2\frac{\ell_1}{\ell} - (1+\alpha)\right)$$

$$P_C = -\frac{1}{2}\alpha\rho\ell(1-\alpha)$$

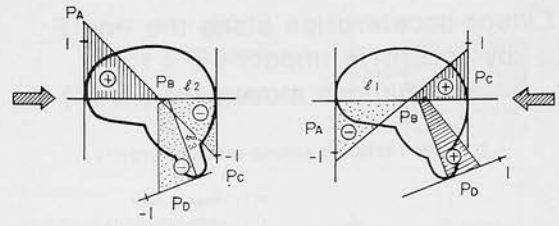
$$P_D = -\frac{1}{2}\alpha\rho\ell\left(\frac{2(\ell_1+\ell_3\cos\theta)}{\ell} - (1+\alpha)\right)$$

$$\alpha \equiv \frac{\frac{A_3\ell_3}{A_1\ell_1}\left(1+\frac{\ell_3}{\ell_1}\cos\theta - \frac{\ell_2}{\ell_1}\right)}{\left(1+\frac{\ell_2}{\ell_1}\right)\left(1+\frac{\ell_2}{\ell_1}+\frac{A_3\ell_3}{A_1\ell_1}\right)}$$

$$X_o = \frac{1}{2}\ell(1+\alpha)$$

α linear acceleration due to impact
ρ internal fluid density
ℓ $\ell_1+\ell_2$
P_A pressure at A P_B pressure at B P_C pressure at C
P_D pressure at D A_1, A_2, A_3 cross-sectioned area of tube AB BC and BD respectively

Fig. 3



$A_1 = A_2 = 50\,cm^2$ $A_3 = 25\,cm^2$
$\ell_1 = 12\,cm$ $\ell_2 = 6\,cm$ $\ell_3 = 12\,cm$
$\rho = 1.04\,g/cm^3$ $\theta = 70°$

frontal blow a = 130 g	occipital blow a = 100 g
P_A = 1.39 kg/cm²	P_A = −1.07 kg/cm²
P_B = −0.24 "	P_B = 0.19 "
P_C = −1.05 "	P_C = 0.81 "
P_D = −0.75 "	P_D = 0.61 "

Fig. 4

In case of angular acceleration, the brain shear deformation is always the problem. Hayashi (4) simplified the problem to the rotational movement of a concentric skull-brain cylinder elastically connected, as is shown in Fig. 6.

3. BRAIN TOLERANCE LEVELS

From Fig. 6, the duration of force to the head t_D is approximately obtained by the assumption such that the change of angular momentum of the skull will be equal to that when the maximum angular acceleration will act constantly for duration t_D:

$$t_D = \int_0^{2t} \ddot{\theta}dt/\ddot{\theta}_{max} = 2\pi\frac{I_s}{I_s+I_o}\sqrt{\frac{I_sI_o}{k(I_s+I_o)}}$$

If we consider that a brain lesion will be produced when the maximum value of brain shear strain γ_{max} reaches the critical value γ^*, the corresponding critical values of $\ddot{\theta}$ and Q are

$$\ddot{\theta}^* = \gamma^* k(I_s+I_o)\,h/2I_sI_oR$$

$$Q^* = I_s\ddot{\theta}^* = \gamma^* k(I_s+I_o)\,h/2I_oR$$

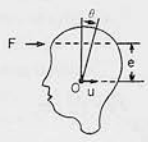
Therefore we obtain

$$\ddot{\theta}t_D^2 = 2\pi^2\frac{h}{R}\left(\frac{I_s}{I_s+I_o}\right)^2\gamma^*$$

Here γ^* is the critical value of the brain shear strain when a lesion is produced. $\ddot{\theta}t_D{}^2$ takes a constant value for a specific part of the brain in the same

Linear acceleration along the line F by eccentric impact (E I) or whiplash movement (W L)

1) Head rotation around center of gravity

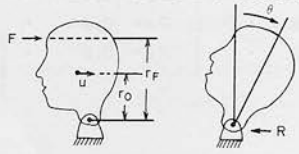


M : mass of head
O : center of gravity
u : horizontal displacement of O
Θ : angle of rotation around O
Io : moment of inertia around O
$i=\sqrt{Io/M}$: radius of gyration
R : reaction force at neck (E I) or force at neck (W L)

Linear acceleration along the line F

$$a=\left(1+\frac{e^2}{i^2}\right)\frac{F}{M}>\frac{F}{M}$$

2) Head rotation around the cervical support



Linear acceleration along the line F

$$a=\frac{r_F^2}{i^2+r_o^2}\cdot\frac{F}{M}>\frac{F}{M}$$

3) i.e.

i) e = 4cm, i = 6cm, $a=1.45\cdot\frac{F}{M}$

ii) rF = 20cm, ro = 13cm, i = 7cm, $a=1.84\frac{F}{M}$

Fig. 5

individual, determined by R, h, I_s, I_0 and γ^*. The corresponding shear stress is expressed as

$$\tau^*=\frac{I_sI_o\theta^*}{\pi R^2(I_s+I_o)}$$

Ommaya et al. (5) have carried out impact experiments in monkeys and observed the maximum angular acceleration $\ddot{\theta}$ and impact duration t_D as shown in Fig. 7. The results suggest the existence of a threshold dividing concussive (open circles) and non-concussive (black circles) areas. If we take a point lying on the threshold such as $\ddot{\theta}=7\times10^4$ rad/sec^2 and $t_D=6$ msec, and make $\ddot{\theta}t_D^2$ curve so as to involve this point, we obtain $\ddot{\theta}t_D^2=2.52$ and we can draw a curve as shown in Fig. 7.

This means that this theoretical equation is in accordance with experimental data and the obtained curve can be regarded as the brain tolerance curve of the angular acceleration.

The brain tolerance curve of the human $(\ddot{\theta}t_D^2)_H$ and that of the monkey $(\ddot{\theta}t_D^2)_M$ are considered to be in the following relationship.

$$\frac{(\ddot{\theta}t_D^2)_H}{(\ddot{\theta}t_D^2)_M}=\frac{[\gamma^*hI_s^2/R(I_s+I_o)^2]_H}{[\gamma^*hI_s^2/R(I_s+I_o)^2]_M}$$

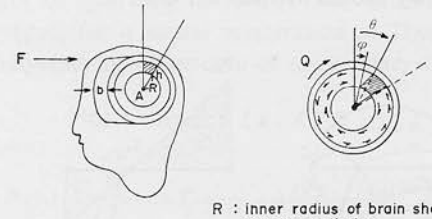
If γ^* is the same in the human and the monkey, this is expressed as

$$\frac{(\ddot{\theta}t_D^2)_H}{(\ddot{\theta}t_D^2)_M}=\frac{[hI_s^2/R(I_s+I_o)^2]_H}{[hI_s^2/R(I_s+I_o)^2]_M}$$

In a rough calculation it is found that $(\ddot{\theta}t_D^2)_H$ is about 1/3 of $(\ddot{\theta}t_D^2)_M$. Therefore the brain tolerance curve in case of angular acceleration in man is much lower than the curve in Fig. 7.

The brain tolerance curve in case of linear acceleration can be likewise derived from the equations described above. The derived equations are shown

Brain shear due to rotation of the cylinder



R : inner radius of brain shear member cylinder
b : length of cylinder
h : thickness of shear member
Q : torque of head caused by force F around the axis of rotation
Is : moment of inertia of skull cylinder
Io : moment of inertia of brain cylinder
γ : shear stress proportional to angular displacement
G : shear modulus of brain
$K: 2\pi GbR^3/h$

Equation of motion

skull cylinder $Is\,\ddot{\theta}=Q-K(\theta-\varphi)$

brain cylinder $Io\,\ddot{\varphi}=K(\theta-\varphi)$

initial condition $t=0,\ \theta=\dot{\theta}=\varphi=\dot{\varphi}=0$

Solution

at the time $t_1=\pi\sqrt{Is\,Io/K(Is+Io)}$

maximum shear strain

$\gamma_{max}=2Q\,Io\,R/Kh(Is+Io)$

maximum shear stress

$$\tau_{max}=G\gamma_{max}=\frac{Q}{\pi bR^2\left(1+\frac{Is}{Io}\right)}$$

maximum angular acceleration of skull $\ddot{\theta}_{max}=\frac{Q}{Is}$

Fig. 6

in Fig. 8. Namely, in case of the soft impact, the product of acceleration and impact duration is

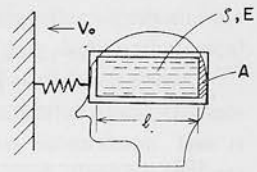
$$a\tau = \frac{4p^*}{\varrho l}\sqrt{\frac{m}{k}}$$

here, $m = \varrho Al$ is mass of the system and k is the spring constant, p^* is the cavitation pressure or other critical pressure which can produce irreversible lesions.

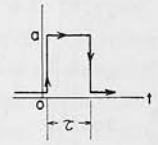
In case of the hard impact, if acceleration is expressed as ηg, the product of acceleration and impact duration is expressed as $\eta\tau = 2p^*/\varrho cg$, here c is the sound velocity in the brain.

If we admit these equations, the brain tolerance curve will be drawn as in Fig. 9 (soft impact) and in Fig. 10 (hard impact) in the form of hyperbola. Experimental data of Gurdjian et al. (1) (human tolerance curve, H.T.C.) may be regarded as the hard impact or a harder part of the soft impact in these equations, when the sound velocity in the brain is 400 m/sec which seems too large for the living human brain, so that Gurdjian's data may not be applicable to the living human brain.

a = acceleration
τ = impact duration
V_o = initial velocity



1) Soft impact



$$a\tau = 2V_o$$

$$V_o = a\sqrt{\rho A\ell/k}$$

$$\frac{1}{2}a\rho\ell = P^* = \text{cavitation pressure}$$

$$\therefore\ a\tau = \frac{4P^*}{\rho\ell}\sqrt{\frac{m}{k}} = \text{const.}$$

2) Hard impact

$$a\tau = 2(\rho CV_o)/(\rho C)$$

$$\rho CV_o = P^* \quad \text{cavitation pressure}$$

$$\therefore\ \eta\tau = \frac{2P^*}{\rho Cg} = \text{const.} \quad (a = \eta g)$$

Fig. 8

4. EXPERIMENTAL RESULTS

The authors have designed the human dummy and the monkey dummy based on physical properties of the head of the man and the monkey. The head and the neck of the dummy were filled with water and contained several pressure sensors. A three-dimensional acceleration sensor was fixed in the maxilla and another one-dimensional acceleration sensor was fixed on the vertex. Fig. 11 shows an example of the human dummy. The monkey was equipped with two one-dimensional acceleration sensors, one on the temporal bone, the other on the vertex, a pressure sensor being placed on the occipital dura.

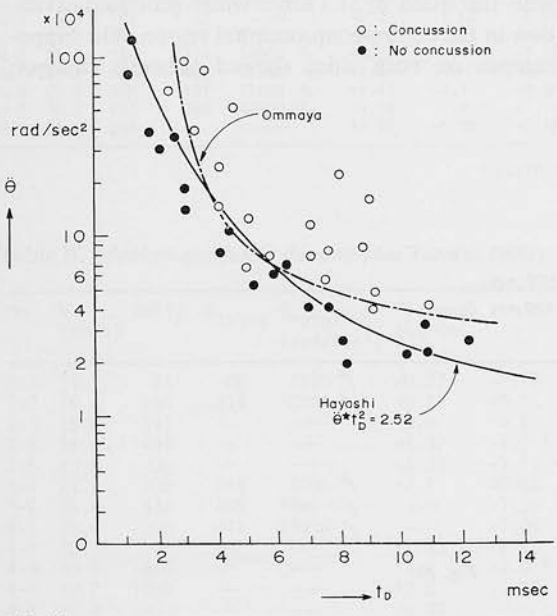


Fig. 7

The human dummy and the monkey dummy, and the monkey dummy and the monkey were fixed on the specially designed sled, the head, the neck and the upper body being freely movable. The sled collided against the concrete barrier with various velocities of 10–31.1 km/h, the forehead of the dummies and the animal being hit by the specially made plate.

Experimental results were listed in Tables I–III. Here, V means the velocity of the sled; $Gx(T)$ or Gx indicates the linear acceleration of the head; G_{trans} means the value at the centre of gravity of the head; G_{rotate} means the angular acceleration of the head (the arrows indicate the direction of the head as seen from the right-hand side); $P(occ)$ or $ICP(occipital)$, $P(front)$ and $P(cerv)$ indicate the intracranial pressure in the occipital and the frontal regions and the intraspinal pressure at the levels of the 3rd–4th cervical

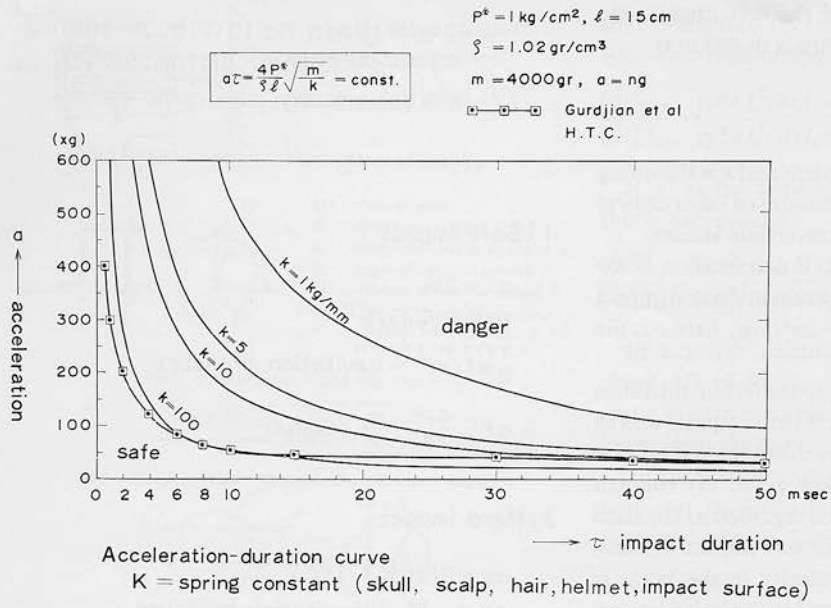


Fig. 9

vertebrae respectively. The intraspinal pressure at the cervical levels was similar to the intracranial pressure in the occipital region, but the values being smaller in most of the cases. In Table IIIB, clinical signs of monkeys are described. The arrows of resp/pulse mean decrease or increase of the rates of respiration or pulse. Monkey HA died of general bad conditions which were present before the experiment.

Fig. 12 summarizes the correlation between theoretical values and the experimental results of the linear acceleration and the intracranial pressure. As seen in Fig. 12, experimental data and the theoretical values were in fairly good accordance. The frontal impact, when it was sufficiently large, caused cavitation as is shown in the lower part of Fig. 12.

Pathological studies of the monkey showed coup and contre-coup injuries in the case Ta of Fig. 12 hit with the speed of 31.1 km/h which produced cavitation in the contre-coup occipital region. The hippocampus on both sides showed ischemic changes;

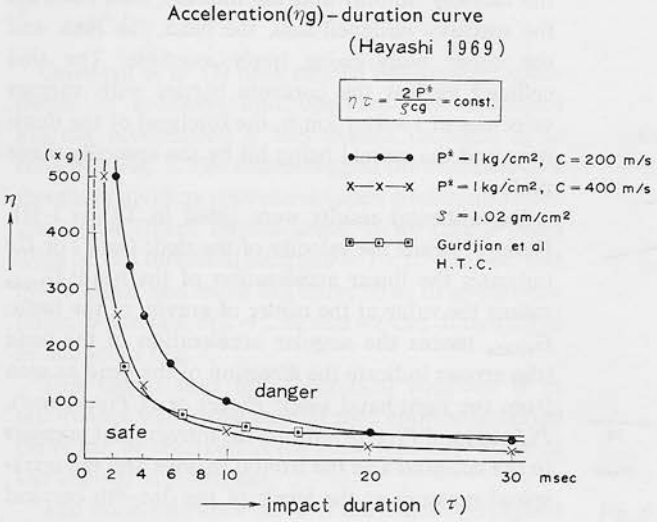


Fig. 10

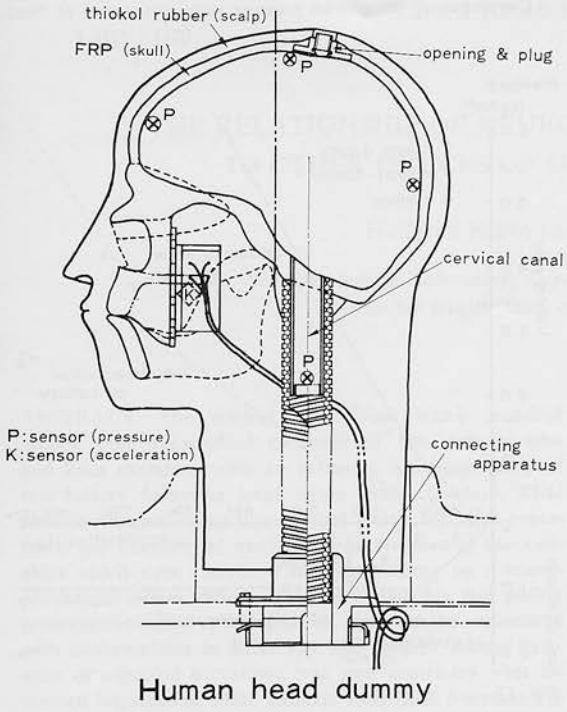


Fig. 11

hemorrhage was seen in the cerebellar vermis and the pons in this case. There were only slight pathological changes in other monkeys hit with lower speed.

The cervical spinal cord exhibited slight changes such as oedema, but no gross hemorrhage. This is probably because the intraspinal pressure changes were smaller than the occipital intracranial pressure. Besides, the angular acceleration of the cervical spine was smaller than that of the head because of damping effects of the flexible spine, therefore the acceleration may have produced only slight changes. The linear acceleration of the cervical spine may have also been smaller than that of the head according to Fig. 5.

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Table I. *Human crushable dummies (at Yatabe, 1970)*

No.	V (km/h)	Gx(T)	G_{trans}	G_{rotat} (rad/sec^2)	P(front) (kg/cm^2)	P(occ)	P(cerv)	Skull fracture
1-8	10.2	42	49	3110 ↖	+0.37	—	+0.48	
2-1	11.1	44	49	3710 ↖	+0.41	-0.45	-0.3	
2-2	16.6	89	87	—	+0.90	-0.80	-0.56	
4-1	20.4	316	274	27500 ↖	+6.75	-1.15 *	—	
4-2	25.3	240	174	18900 ↖	+3.79	-1.11 *	-1.14 → +1.92	
4-3	30.3	530	460	47250 ↖	+9.6	—	-0.73	++
4-4	20.2	158	131	11000 ↖	+1.43	-1.1	-0.38	
4-5	30.8	556	385	47500 ↖	+4.76	-1.0 *	-1.19 → +1.19	++
4-6	25.8	254	—	—	+3.50	-0.84	+0.78	

* Cavitation

Table II. *Monkey crushable dummies (at Yatabe, 1970)*

No.	V (km/h)	Gx(T)	G_{trans}	G_{rotat} (rad/sec^2)	P(front) (kg/cm^2)	P(occ)	P(cerv)	Fracture
2-1	11.1	61	68	3190 ↖	+0.27	-0.16	—	
2-2	16.6	191	214	10200 ↖	+0.71	-0.76	—	
2-3	15.6	141	—	—	+0.8	-0.62	-0.47	
2-4	16.0	430	—	—	+1.37	-1.0 *	+0.44 → -1.0	
2-5	23.6	730	—	—	+4.25	-1.17 *	+3.6	teeth
3-1	21.3	332	285	5700 ↖	+2.1	-0.64	-0.63	
3-2	25.9	530	499	9800 ↘	—	-1.39	+0.87 → -1.34 *	
4-1	20.4	335	412	23600 ↖	—	-1.04	+0.97	maxilla
4-2	26.3	—	—	—	+1.13	-2.23	-0.46 → +1.03	
4-3	30.3	>960	—	—	+2.6	-2.0 *	+3.02 → -0.79	maxilla & skull
4-4	20.2	1000	—	—	>+2.5	-1.18	+1.63	maxilla
4-6	25.8	—	—	—	+0.89	—	-0.21	

Table III A. *Monkeys*

No.	Name	V (km/h)	Gx(T)	G_{trans}	G_{rotat} (rad/sec^2)	P(occ) (kg/cm^2)
2-3	YA	15.6	148	342	34200 ↘	-0.1
3-1	JA	21.3	208	271	7490 ↘	-0.10
3-2	TS	25.9	210	480	37000 ↖	-0.38
3-3	TA	31.1	284	277	22600 ↖	-1.0
3-4	HA	25.3	169	24	57500 ↖	-0.53

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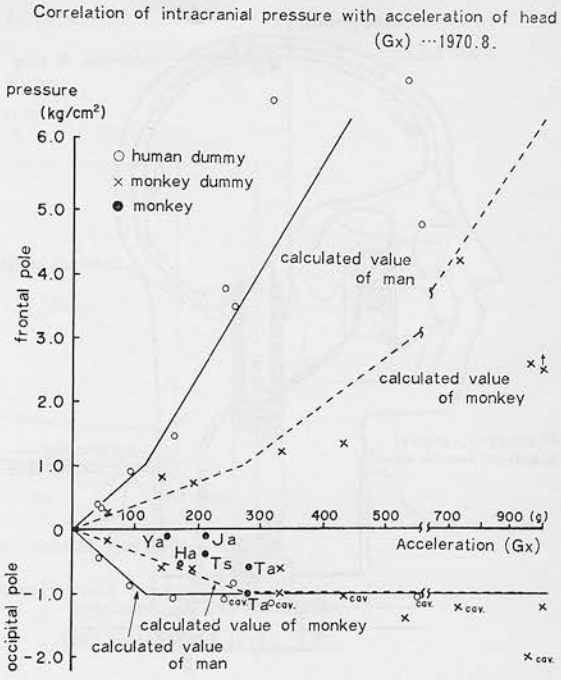

Fig. 12

Table III B. *Clinical signs of monkeys*

NO.	G_x	ICP(occipital) kg/cm^2	resp. / puls.	clinical course	CSF	brain contusion
2-3YA	342	-0.1	↘ / ↗	unconsciousness for 30′	clear	(-)
3-1JA	271	-0.1	↘ / ↘	minimal concussion prostrated	clear	(-)
3-2TS	480	-0.38	↘ / →	minimal concussion prostrated	bloody	(-)
3-3Ta	277	-1.0	↘ / ↗	fatal concussion 40 hrs.	bloody	(+)
3-4HA	24	-0.53	↘ / ↘	fatal concussion 4 hrs.	bloody	(-)