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Strain Tolerance of the Vv. Cerebri sup. (Bridging Veins) Calculated from Head-on Collision Tests with Cadavers

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Summary. High-speed films recorded from head on collision tests with cadavers have been analysed with respect to the angular acceleration and angular velocity which were evaluated with Smoothed Cubic-Spline Functions. These results compared with the autopsy findings in these cases and data from studies on angular acceleration to the head of squirrel monkeys carried out by Unterharnscheidt and Higgins indicate the bridging vein disruption due to rotational movement of the head is obtained when the angular acceleration exceeds 4500 rad/sec^2 and/or the change in angular velocity exceeds 50 rad/sec. These tolerance levels are compared with data from collision experiments on living human beings.

Zusammenfassung. Mit Hilfe der Smoothed Cubic-Spline Functions wurden Winkelbeschleunigung und Winkelgeschwindigkeit des Kopfes auf Grund von High-speed-Filmen berechnet, die bei frontalen Kollisionen mit Leichen aufgenommen wurden. Die Resultate wurden mit den Sektionsbefunden in diesen Fällen und Daten verglichen, die bei Studien über die Winkelbeschleunigung des Kopfes von Affen (Samiri sciureus) durch Unterharnscheidt u. Higgins gewonnen wurden. Es zeigt sich, daß Rupturen der parasagittalen Brückenvenen dann auftreten, wenn die Winkelbeschleunigung des Kopfes 4500 rad/sec^2 überschreitet und/oder eine Veränderung der Winkelgeschwindigkeit von mehr als 50 rad/sec vorliegt. Diese Toleranzgrößen werden mit Daten verglichen, die bei Versuchen mit lebenden Menschen gewonnen worden sind.

Key words: Bridging veins, strain tolerance — Bridging vein disruption.

Introduction

When the human body is subjected to acceleration as a result of an impact from the front or the rear, a rotational movement of the head around a transverse axis will result in most cases. In occupants in frontal automobile collisions, such a movement is mostly caused by the impact of the head against the interior of the car, *i.e.* the dash-board, the wind-shield or the steering wheel. A similar movement occurs when the head is hit by a fist or when falling backwards or prostrate to the ground. The angular acceleration caused by such impacts to the head can primarily give rise to disruption of the parasagittal bridging veins (vv. cerebri sup.) or to gliding contusions (Lindenberg and Freytag) *i.e.* parasagittal lesions situated cortically or subcortically and surrounded by local subarachnoidal hemorrhages. These kinds of injuries are quite common and have been shown in no less than 25% of consecutive autopsy cases consisting of deaths caused by blunt trauma (Voigt and Löwenhielm). Improved knowledge of the dynamic mechanisms in-

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volved in producing these injuries is necessary in the construction of safer protective system used in, for example, frontally colliding cars. Since the intracranial injuries induced by such trauma are dominated by tearings and disruption of the parasagittal bridging veins, it is necessary to investigate the tolerance of the veins when the head is subjected to angular acceleration around a transverse axis. Unterharnscheidt and Higgins have tried to illustrate the tolerance of the brain in carefully controlled studies with squirrel monkeys. Their results, however, cannot simply be transferred to man. Voigt and Lange and Voigt *et al.* have produced bridging vein disruption in collision experiments with unembalmed cadavers. Their experiments were filmed at high-speed. In this paper an evaluation of these films is given with the aim of demonstrating the tolerance level of the parasagittal bridging veins when the head is subjected to rotational motion. Of special interest is the influence of angular acceleration and angular velocity.

Materials and Methods

The high-speed films were recorded with a filmspeed of 500 frames/sec. Of 23 films only 9 were further analysed. In these 9 films no side movement of the head was noticed. In the experiments a test sled was accelerated backwards by an impulse from an electrohydraulic piston. On the sled a conventional carseat, a dash-board which in the successive trials had various designs, and a wind-shield of celluloid were mounted. The cadavers used were unembalmed and had been kept at a temperature of 14° C so that rigor mortis no longer persisted when the experiments were conducted.

The analysed films were recorded with a stationary camera. The heads of the corpses had been wrapped up in a single layer of elastic bandage and furnished with markings which were partly sewn to the skin. From the different markings the angle-time-history of the head was measured directly on the films with a digital measuring microscope. In order to obtain the angular velocity and the angular acceleration the first and second derivative with respect to the time has to be calculated. However, conventional differentional methods for numerical derivation do not give satisfactory results, especially when working with experimental data. Therefore Smoothed Cubic-Spline Functions have been used in order to obtain high quality derivatives (Berghaus and Cannon; Reinish). A spline is a piece-wise-interpolation polynomial defined over a set of points (t_i). Individual cubic functions $h(t_i)$ are obtained for each interval $(t_i, t_i + 1)$ with the derivatives to the second order held continuous over the entire span (t_o, t_n).

The integral:

$$\int_{t_o}^{t_n} \frac{d^2 h(t)^2}{dt^2} dt$$

is minimized such that:

$$\sum_{i=0}^{n} \left(\frac{h(t_i) - y_i}{\delta y_i} \right)^2 \leqq S$$

where y_i are the data values and δy_i are the estimated errors or standard deviations for the various $y_i \cdot S$ is a redundant smoothing index which is used to adjust the degree of smoothing. These measures allow the function $h(t)$ to deviate from the data and assures a smoother first and second derivative than if the function is constrained to pass through all data.

With this method the films were analysed with respect to the angular acceleration and the angular velocity of the head. At the autopsies which were undertaken after the experiments the brain was dissected ad modum Flechsig thus permitting inspection of the bridging veins.

In 4 of the studied cases disruption of the bridging veins or laceration of the soft cerebral membranes was found.

Results

In Table 1 the autopsy findings and the causes of death for the cadavers used in the experiments are presented. The angular acceleration and the angular velocity evaluated with Smoothed Cubic-Spline Functions from the high-speed films are given in Fig. 1a—i.

The movement of the cadavers during an experiment was of two types. In cases 1—6 the pattern of movement was the following: When the front part of the dash-board on the accelerating sled hit the thorax of the cadaver the head began to rotate forward. This phase of acceleration correspond to the first big valley of the acceleration curve, Fig. 1a—f. The head rotated forward until the face, the forhead or the cheek hit the upper side of the dashboard, whereby the movement was hindered and a peak in the acceleration attained. Until this moment the angular velocity had been negative, but now became positive as the head began to rotate backwards. The body was thrown backwards and the movement was stopped by the back of the seat or the body submarined under the dash-board. The motion in cases 7—9 differs from that in cases 1—6 by the fact that the head after its contact with the upper side of the dash-board contacted the wind-shield. This explains the extra peaks and valleys in the angular acceleration curves presented in Fig. 1g—i. The contact with the wind-shield furthermore caused the head to remain in a anteflected position when moving backwards with the body.

In Fig. 4 the maximal angular acceleration as a function of the change in angular velocity is presented. Fig. 5 gives a similar relationship but these data are derived from studies on angular acceleration to the head of squirrel monkeys made by Unterharnscheidt and Higgins. Finally, in Fig. 6 the tolerance levels for bridging vein disruption are given. In this figure results from experiments with living human beings are indicated (Ewing and Thomas; Clarke *et al.*).

Table 1. Cause of death and gross autopsy findings of the head for the 9 studied cases

Fig.	Case	Cause of death	Gross autopsy findings of the head
1a	1	Hanging	Disruption of several bridging veins. Laceration of the soft cerebral membranes. Minor subarachnoidal hemorrhage.
1b	2	Hanging	Disruption of several bridging veins, with local surrounding subarachnoidal hemorrhage.
1c	3	Hanging	—
1d	4	Intoxication	Minor subarachnoidal hemorrhage.
1e	5	Intoxication	Laceration of the soft cerebral membranes along the mantel edge. Subdural hemorrhage.
1f	6	Hanging	Disruption of several bridging veins. Minor subarachnoidal hemorrhage. Laceration of the soft cerebral membranes along the midline.
1g	7	Myocardial infarction	—
1h	8	Tuberculosis, cor pulmonale	—
1i	9	Myocardial infarction	—

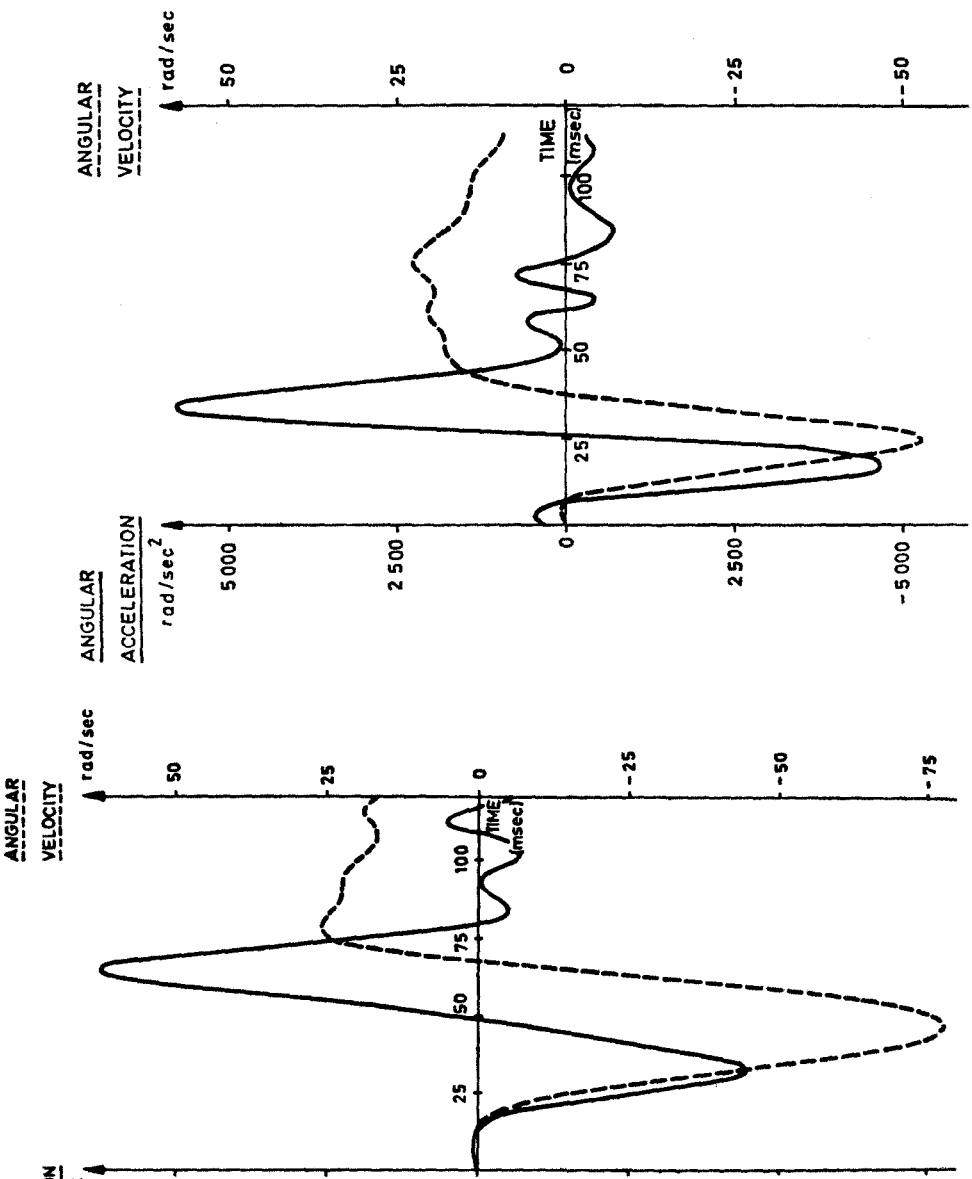
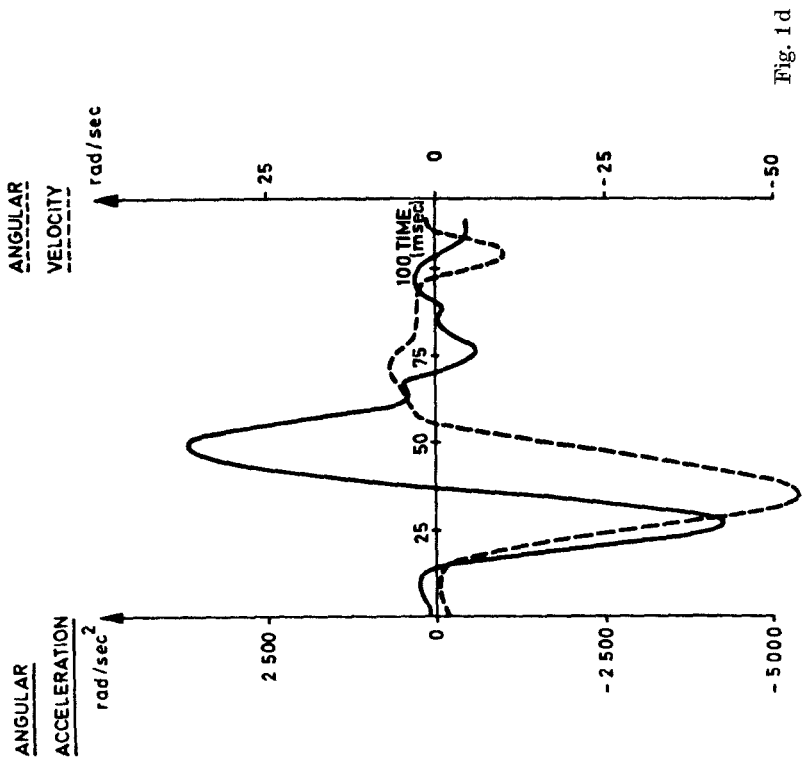


Fig. 1a—i. Angular acceleration (solid) and angular velocity (dashed) as a function of time, obtained by derivation of the angular position-time-history using Smoothed Cubic-Spline Functions

Fig. 1a

Fig. 1b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VELOCITY
rad/sec
25
0
-25
-50
100 TIME (msec)
75
50
25
0
ANGULAR
ACCELERATION
rad/sec²
2 500
-2 500
-5 000

Fig. 1 d

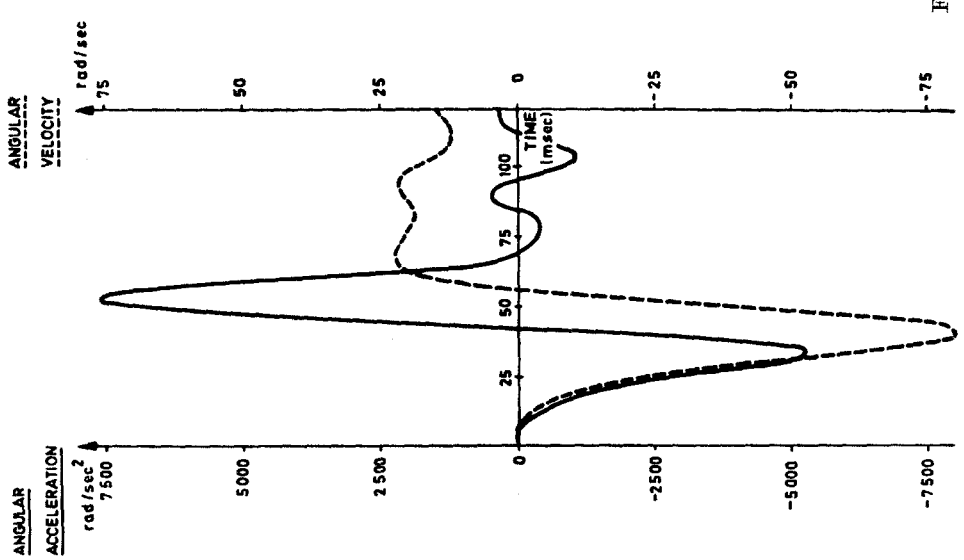

ANGULAR
VELOCITY
rad/sec
75
50
25
0
-25
-50
-75
100 TIME (msec)
75
50
25
0
ANGULAR
ACCELERATION
rad/sec²
7500
5000
2500
-2500
-5000
-7500

Fig. 1 c

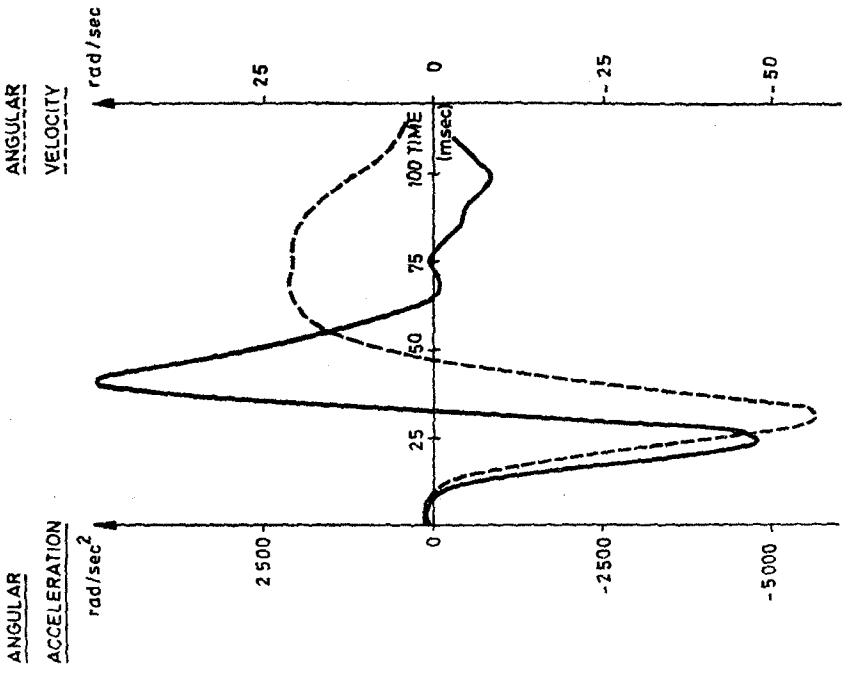
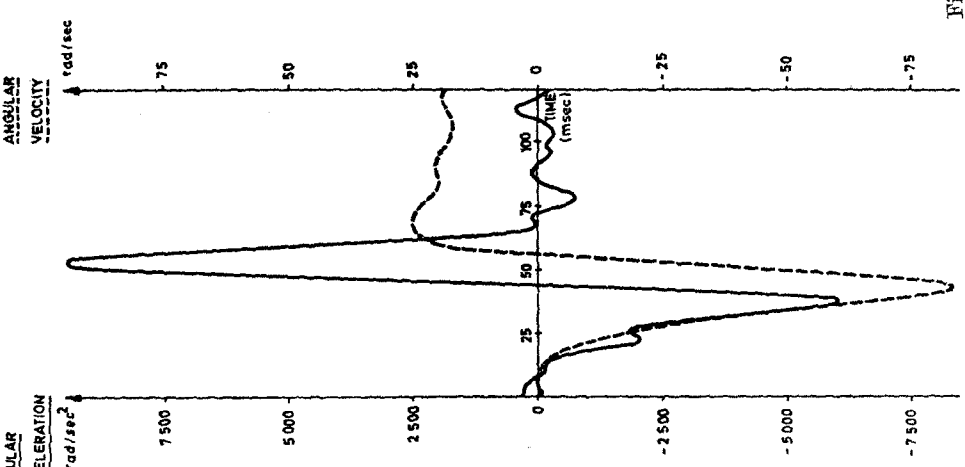

ANGULAR
ACCELERATION
rad/sec²
2 500
0
-2500
-5000
ANGULAR
VELOCITY
rad/sec
25
0
- 25
- 50
25
50
75
100 TIME
(msec)

Fig. 1 f


ANGULAR
ACCELERATION
rad/sec²
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5 000
2 500
0
-2500
-5000
-7500
ANGULAR
VELOCITY
rad/sec
75
50
25
0
- 25
- 50
- 75
25
50
75
100
TIME
(msec)

Fig. 1 e

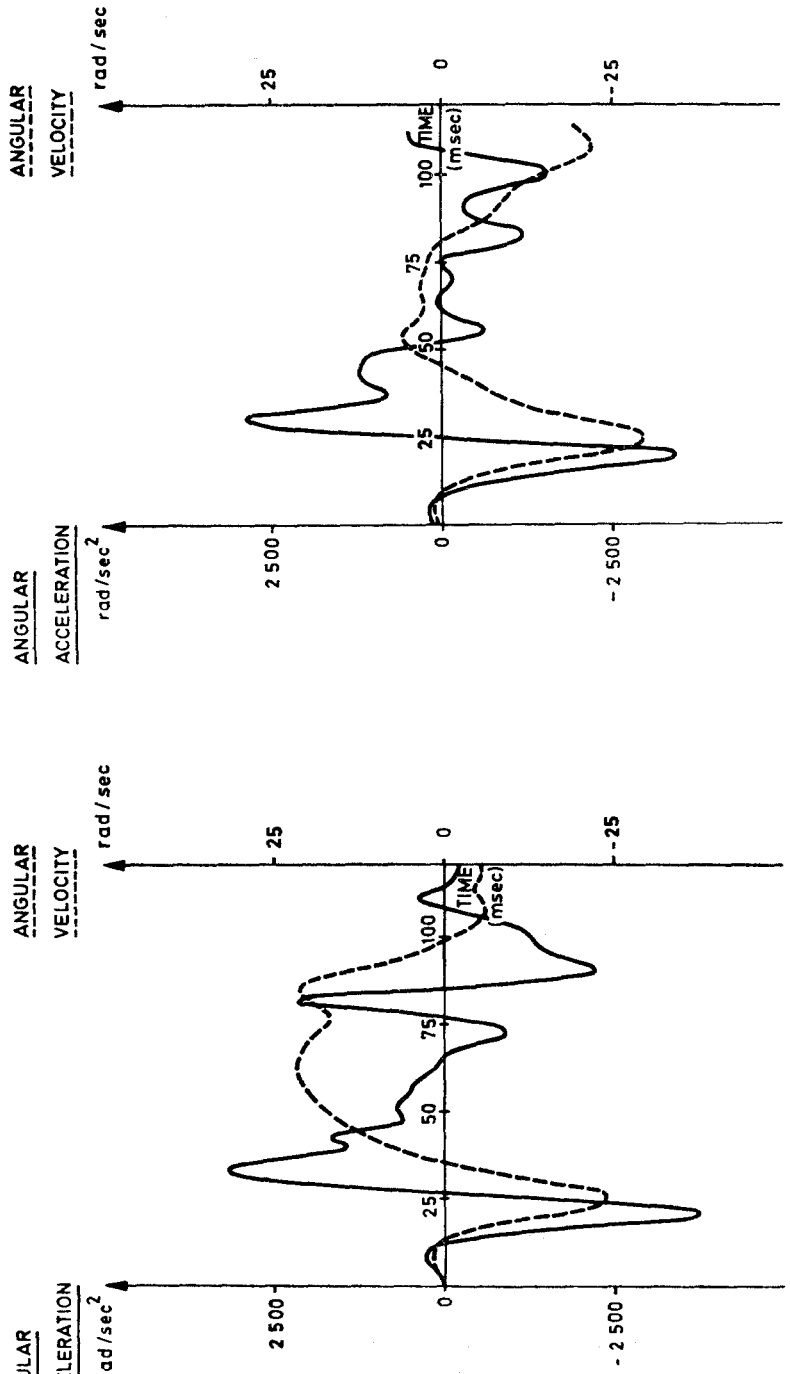
ANGULAR
VELOCITY
rad/sec
25
0
-25
TIME
(msec)
100
75
50
25
ANGULAR
ACCELERATION
rad/sec²
2 500
0
- 2 500

Fig. 1 h

ANGULAR
VELOCITY
rad/sec
25
0
-25
TIME
(msec)
100
75
50
25
ANGULAR
ACCELERATION
rad/sec²
2 500
0
- 2 500

Fig. 1 g

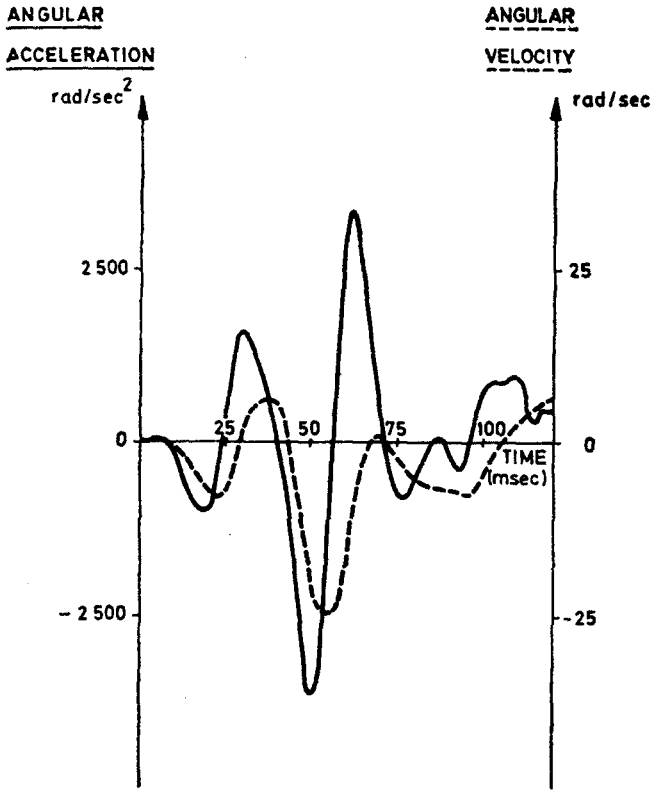

Fig. 1 i

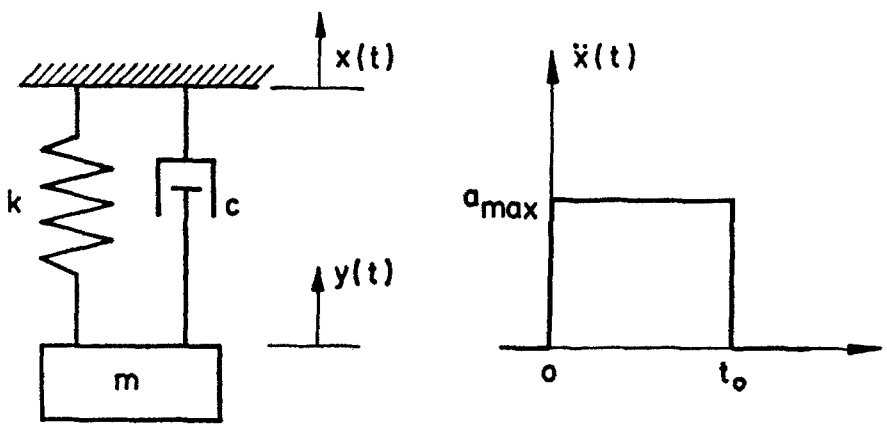

Fig. 2. Viscoelastic rheological model for the motion of the bridging veins (Kelvin-Voigt model), and the squareshaped acceleration pulse applied to the fundament (skull)

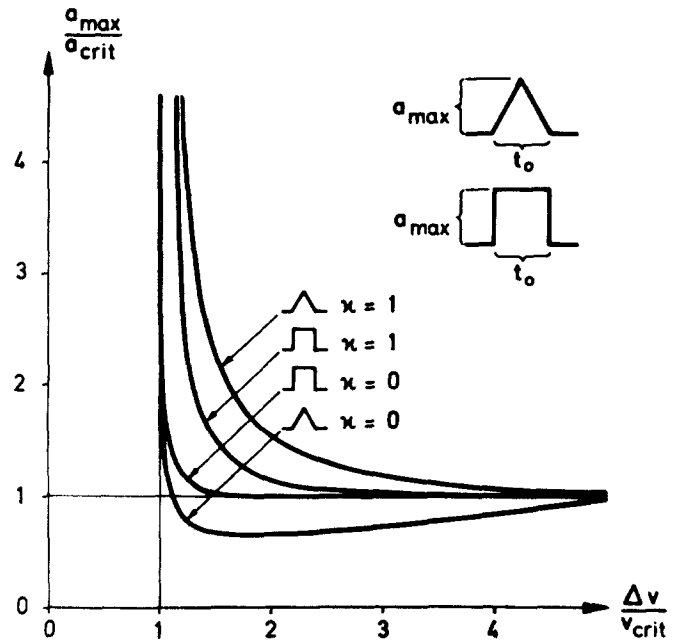

Fig. 3. Theoretical tolerance curves for parasagittal bridging veins with different damping properties when subjected to square shaped and triangular shaped acceleration pulses. The maximal elongation of the veins is expressed as a function of the maximal acceleration and the velocity change

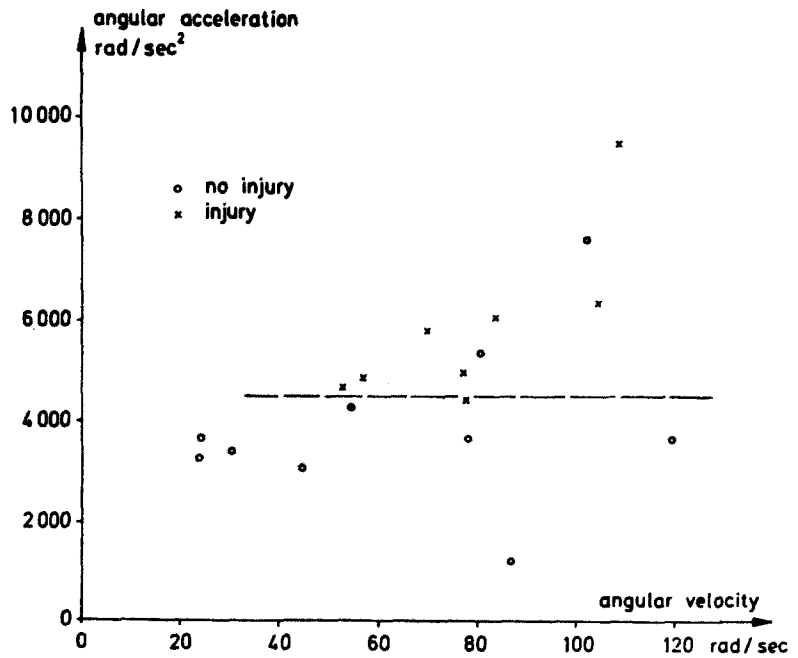

Fig. 4. Results from the evaluated high-speed films. The maximal angular acceleration is plotted versus the change in angular velocity

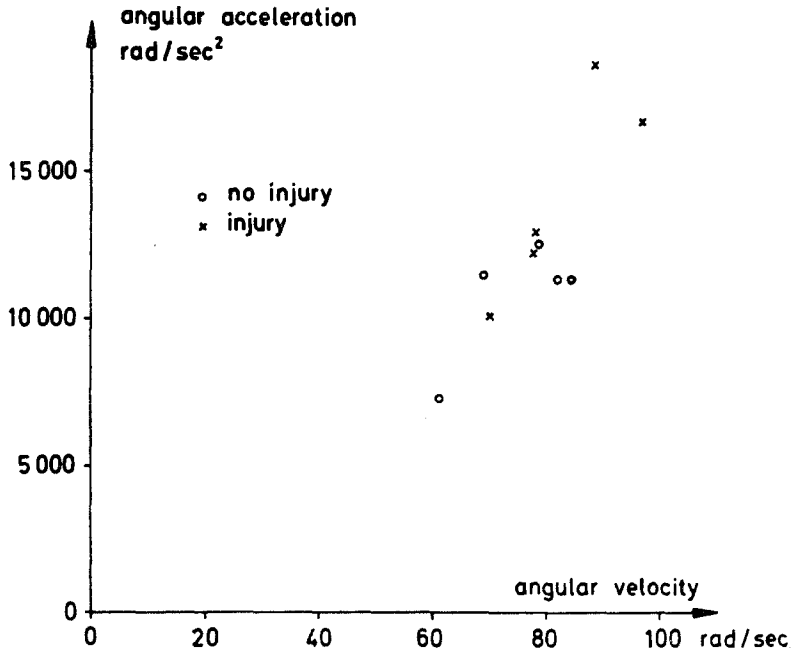

Fig. 5. Scaled data from studies on angular acceleration to the head of squirrel monkeys reported by Unterharnscheidt and Higgins. Note that the data points approximately fall along a branch of a parabola

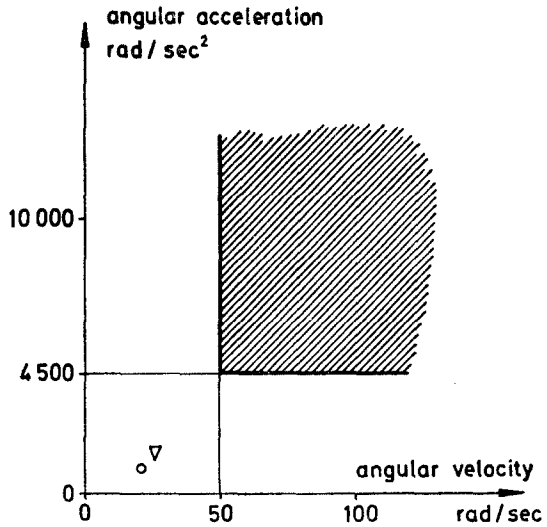

Fig. 6. Experimental tolerance levels for bridging vein disruption. ○ and ▽ indicate results from head-on collision experiments conducted by Ewing and Thomas and Clarke *et al.*, respectively. They used passively restrained human beings as test subjects. Hached area represent the injurious domain

Discussion

When the head is subjected to angular acceleration around a transverse axis a displacement of the brain surface relative to the dura will take place. This has experimentally been shown by Pudenz and Shelden. This displacement can induce bridging vein disruption in the subdural or the subarachnoidal space since the bridging veins together with connective tissue comprise the only connection between the upper parts of the brain surface and the dura. Due to angular acceleration deformation of the brain matter also takes place. Holbourn has shown with a two-dimensional photoelastic gelatinous model of the brain that deformation of considerable magnitude is to be expected close to the brain's surface. This result is confirmed by experiments and calculations carried out by Ljung on mechanical and mathematical models. The deformation of the brain substance can attain centimeter size and thus cause streching of the bridging veins not only in the subdural and subarachnoidal space but also in the parts of the veins running through the brain matter. This then explains the origin of the subcortical hemorrhages described by Lindenberg as gliding contusions or to the necrotic lesions of the white matter described by Voigt and Löwenhielm. The latter lesions are mostly situated subcortically in the posterior aspect of the superior frontal gyrus.

As an injury criterion for bridging vein disruption the ultimate elongation can be chosen irrespectively of the location of the disruption. The question is then what the maximal elongation depends on. Suppose the bridging veins can be described with a simple visco-elastic rheological model (Kelvin-Voigt) (Fig. 2).

The differential equation of motion can then be written:

$$m\ddot{y} = c(\dot{x} - \dot{y}) + k(x - y) .$$

Let $c/m = 2b$ and $k/m = \omega^2$. Laplace transformation of the equation yields:

$$Y(s) = \frac{2bs + \omega^2}{s^2 + 2bs + \omega^2} X(s) .$$

Studying the case when the acceleration-time history of impact is a square pulse Fig. 2, then:

$$x(t) = \begin{cases} 0 & : t < 0 \\ \dfrac{a_{\max} t^2}{2} & : 0 \leqq t < t_o \\ a_{\max} t_o t - \dfrac{a_{\max} t^2}{2} & : t \geqq t_o \end{cases} .$$

The Laplace transform of these conditions is:

$$X(s) = \frac{a_{\max}}{s^3} (1 - e^{-st_o}) .$$

Now study the elongation

$$Z(s) = X(s) - Y(s)$$

$$Z(s) = \frac{a_{\max}}{s(s^2 + 2bs + \omega^2)} (1 - e^{-st_o}) .$$

The inverse transform is then obtained as:

$$z(t) = \frac{a_{\max}}{\omega^2} \cdot \left\{1 - e^{-bt} \cdot \cos\left(\sqrt{\omega^2 - b^2} \cdot t\right) - \frac{b}{\sqrt{\omega^2 - b^2}} \cdot \sin\left(\sqrt{\omega^2 - b^2} \cdot t\right)\right\} \cdot \{H(t) - H(t - t_o)\}$$

where $H(t)$ is an unit step. Study the case when $t_o \gg \frac{1}{\omega}$, *i.e.* when the loading time is long compared with the characteristic period of the system.

Then:

$$z(t) = \frac{a_{\max}}{\omega^2} \left\{1 - e^{-bt} \cos\left(\sqrt{\omega^2 - b^2}\, t\right) + \frac{b}{\sqrt{\omega^2 - b^2}} \sin\left(\sqrt{\omega^2 - b^2}\, t\right)\right\}$$

and the maximal elongation is obtained as:

$$z(t)_{\max} = \frac{a_{\max}}{\omega^2} \left(1 + e^{-\frac{\varkappa}{\sqrt{1-\varkappa^2}} \cdot \operatorname{arctg}. \frac{\varkappa\sqrt{1-\varkappa^2}}{\varkappa^2 - 1/2}}\right) = \frac{a_{\max}}{\omega^2} \cdot f_1(\varkappa)$$

where $\varkappa = \frac{b}{\omega}$ is a dimensionless constant which describes the damping of the system. When $\varkappa = 0$ there is no damping while $\varkappa = 1$ indicates critical damping. Thus $z_{\max}$ is proportional to $a_{\max}$ when $t_o \gg \frac{1}{\omega}$. Now let $t_o \ll \frac{1}{\omega}$. Then

$$z(t) = \frac{a_{\max} \cdot t_o}{\omega^2} e^{-bt} \cdot \left\{\sqrt{\omega^2 - b^2} \sin\left(\sqrt{\omega^2 - b^2}\, t\right) + b \cdot \cos\left(\sqrt{\omega^2 - b^2}\, t\right)\right\}$$

and

$$z(t)_{\max} = \frac{a_{\max} \cdot t_o}{\omega} \cdot \sqrt{1 - \varkappa^2} \cdot e^{-\frac{\varkappa}{\sqrt{1-\varkappa^2}} \cdot \operatorname{arctg}. \frac{1/2 - \varkappa^2}{\varkappa\sqrt{1-\varkappa^2}}} = \frac{a_{\max} t_o}{\omega} \cdot f_2(\varkappa) .$$

In this case $z(t)_{\max}$ is proportional to $a_{\max} \cdot t_o = \Delta v$ *i.e.* the change in velocity. The maximal elongation of a bridging vein can consequently be written as:

$$z_{\max} = f(a_{\max}, \Delta v, \varkappa) .$$

In an equivalent manner similar properties of $z_{\max}$ can be shown for other pulse shapes of $\ddot{x}(t)$.

In Fig. 3 the appearance of $z_{\max} = f(a_{\max}, \Delta v, \varkappa)$ is presented for the cases when $\ddot{x}(t)$ are square and triangular pulses. These pulses can be regarded as extreme forms between which most physically occuring pulses fall. The different curves in Fig. 3 thus correspond to the limit for preserved solidity in different damping and loading cases. From Fig. 3 it can be seen that the different curves rapidly join the lines $a_{\max} = a_{\text{critical}}$ and $\Delta v = v_{\text{critical}}$. In spite of big variations in pulse shapes and damping properties the tolerance curves exibit small deviations. Of special interest is the narrow bands of variation comprised by the curves of equal damping. Thus an $a_{\max}$—Δv form is a good way of representation when giving tolerance thresholds, because of the insusceptibility to changes in the pulse shapes and because of the fact that the parameters $a_{\max}$ and Δv in most cases are easy to measure.

The property of the tolerance curves of rapidly joining the lines a_{critical} and v_{critical} makes it possible in practice to say that one of the conditions: $a_{\max} < a_{\text{critical}}$; $\Delta v < v_{\text{critical}}$ shall be satisfied in order not to compromise the solidity of the bridging veins.

Since a rotational movement of the head is the main cause for the stretching and disruption of the bridging veins the threshold for maintained solidity can be written as a function of the angular acceleration and the change in angular velocity of the head instead of the maximal acceleration and velocity change of the bridging veins themselves.

In Fig. 4 the results from the high-speed film analysis is presented. From Fig. 1 it is evident that at least two acceleration extremities are obtained at each trial. In those cases with no injury the values for both "peaks" can be used. However, when injury has occured it is not possible to determine at which phase of the movement the injuries were produced. In any case, both peaks have been presented in Fig. 4. This figure gives a threshold value for injury at the angular acceleration 4500 rad/sec^2. This value then approximately corresponds to the critical angular acceleration. Based on the data it is difficult, though, to estimate the critical change in angular velocity. Linear regression through all the data points yields a value of about 60 rad/sec. In order to examine this value it was compared with results reported by Unterharnscheidt and Higgins. They conducted carefully controlled studies of angular acceleration to the head of squirrel monkeys (Samiri sciureus). Their reported results were scaled according to the formulas (Ommaya *et al.*):

$$\dot{\vartheta}_{\text{man}} = \dot{\vartheta}_{\text{monkey}} \left(\frac{m_{\text{monkey}}}{m_{\text{man}}} \right)^{1/3}$$

$$\ddot{\vartheta}_{\text{man}} = \ddot{\vartheta}_{\text{monkey}} \left(\frac{m_{\text{monkey}}}{m_{\text{man}}} \right)^{2/3}$$

where $\dot{\vartheta}$ is the angular velocity, $\ddot{\vartheta}$ the angular acceleration, and m_{monkey} and m_{man} are the brain mass for monkey and man respectively. The scaled values are plotted in Fig. 5. Due to the fact that these experiments were carefully controlled (triangular acceleration pulses) the obtained results will fall along a branch of a parabola ($\ddot{\vartheta} = k\dot{\vartheta}^2$). These data give the threshold values $\ddot{\vartheta}_{\text{max}} = 11\,500$ rad/sec^2 and $\Delta\dot{\vartheta} = 75$ rad/sec. The scaled value of the angular velocity change is of the same magnitude as the value obtained from the cadaver tests. As the loading time in the experiments with the squirrel monkeys was about 3 msec and can be regarded as short compared with a characteristic period of the bridging vein (Löwenhielm), this value ought to be close to the critical change in angular velocity. When the values for monkey and man are compared some caution must be exercised. For example, the geometric forms of the skulls do not completely agree, the different experiments were conducted on living and dead tissues, a fact having consequences for the viscosity, which furthermore is very dependent on the temperature. In any case, the critical change in angular velocity should not differ too much from the values given above.

Based on the preceeding discussion it seems possible to put forth the following tolerance criteria for bridging vein disruption: If at least one of the conditions:

$$\ddot{\vartheta}_{\text{max}} < 4500 \text{ rad/sec}^2; \; \Delta\dot{\vartheta} < 50 \text{ rad/sec}$$

is fulfilled, the solidity of the bridging veins will not be compromised and the veins will remain intact.

In Fig. 6 the obtained tolerance levels are compared with results from impact experiments with living human beings reported by Ewing and Thomas and Clarke *et al.* In their experiments the test subjects were passively restrained. From Fig. 6 it can be seen that these experimental results fulfill both of the non-injury conditions.

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