ON THE MECHANISM OF CORTICAL BRIDGING VEIN RUPTURE.

Peter Löwenhielm
Department of Forensic Medicine
University of Lund

ABSTRACT

A common head injury in car crash victims is rupture of the parasagittal bridging veins. These ruptures are not only seen at the surface of the brain, but also in the depth of the convolutions in the gray and the white matter. The cause of these deeper ruptures has been obscure and has been described as gliding contusions. In order to explain this type of injury the dynamic properties of the bridging veins were investigated. The results indicate that the elongation capacity of the vessels is strongly dependent on the elongation rate. Thus, at static loading the elongation may be more than 100 % while at elongation rates at about 500 sec^{-1} the elongation capacity is reduced to 20 %. The deformation of the brain close to the brain surface is considerable, when the head is subjected to angular acceleration and will elongate the bridging veins more than 20 %, thus causing rupture.

INTRODUCTION

Voigt & Saldeen[1] were first to show that traumatic ruptures of bridging veins often are coincident with brainstem lesions. Such injuries lead to immediate or eventual death. Pinshaped bleedings in the depth of the parasagittal brain convolutions were described as gliding contusions by Lindenberg[2]. He differentiated them from "real" cortical contusions, which are characterized by a local subarachnoid haemorrhage together with many pinpoint bleedings at the top of the convolutions. The gliding contusions are produced by a stretching of the connection between the dura and the surface of the brain (bridging veins, Pacchionian granulations). In the autopsy material from 1971 and 1972 at the Department of Forensic Medicine, Lund, 152 out of 563 cases (~ 27 %) showed rupture of the bridging veins. In 40 % of these cases there was no fracture of the skull. Reconstruction of the sequence of events in the accident was made in many cases by means of a simple dummy. Thus it was possible to correlate the victim's injuries found at autopsy with the damage on the car and develope some conception of the genesis of the injuries. It was found that ruptures of the bridging veins occurs when the head is subjected to angular acceleration of the magnitude 1000 - 10000 rad/sec^2. This happens for example when the driver's chin hits the steering-wheel or the front passenger's forehead hits the wind-shield. Angular acceleration during whip-lash (retroflexion of the head as a consequence of a rear-end collision) does not seem to be great enough to produce rupture.

PROBLEM FORMULATION

The mechanism of the gliding contusions has not been explained. As connective tissue is formed around the Pacchionian granulations with age the brain surface along the manteledge in the adult is progressivly prevented from moving relative to the skull. This fibrous anchor could cause greater movement in the deep lying parenchyma of the brain with acceleration (gliding contusions are most frequently seen in adults). Ljung[3] has shown that the brain matter is destorted when given an angular acceleration.

It is well known from static tests[4], that human veins can elongate 100 % or more. If this value were applicable for dynamic loading a considerable brain dostortion would have to take place before rupture of bridging veins could occur. Therefore, an investigation of the dynamic properties of the bridging veins was necessary.

METHODS AND MATERIALS.

An experimental setup was constructed, see fig. 1 and fig. 2. Under tensile loading, such dynamic properties as elongation capacity and ultimate stress were studied at constant elongation rates.

The test device (fig. 1) consists of a cylinder (1) which can be accelerated with compressed air in a tube. Its kinetic energy is transfered to another cylinder (2), which then is given an instant velocity. The speed is measured by means of two leads (3) mounted at right angel to the direction of movement and at a fixed distance. The leads are used as switenes in an electrical circuit. The cylinder (2) breaks off the leads and the time interval between the breakes is measured. The bridging veins to be tested are mounted between the two clips (4), and the tensile force is recorded by means of a piezoelectric force transducer (5). Of course the cylinder (2) loses speed because of friction forces in the bearings (6) and tension in the vein, but when rupture occurs the reduction is less than 4 % and is therefore neglected. An outline of the complete setup is shown in fig. 2.

CALCULATIONS.

a) Elongation rate ($\dot{\varepsilon}$).

The time (t) between the brakes of the leads used as position switches is recorded on an oscilloscope. If the leads are fixed at a distance (s) the mean spead of the cylinder (2) is

$$\bar{v} = \frac{s}{t}$$

The elongation rate ($\dot{\varepsilon}$) is then defined as $\dot{\varepsilon} = \frac{\bar{v}}{l_o}$

where l_o is the length of the bridging vein between the two clips.

b) Stress (σ)

The force (F) is shown as a deflection on the oscilloscope. The stress is defined as

$$\sigma = \frac{F}{A}$$

where A is the cross sectional area. A is calculated from the frozen right angle sections of bridging veins, which are photographed with a microscope camera.

c) Elongation (ε)

ε is defined as

$$\varepsilon = \frac{\bar{v}\, t_s}{l_o}$$

where t_s is the time to rupture.

DISCUSSION OF THE RESULTS.

The results are presented in fig. 3 and fig. 4. The elongation capacity is markedly reduced when the elongation rate is increased. For example, when $\dot{\varepsilon}$ has a value of 500 sec^{-1} or more, the elongation is reduced to about 20 %. Ljung has experimentally shown the displacement pattern of brain matter when subjected to angular acceleration, fig. 5. The maximal shear stresses occur near the brain surface, and can be 45 degrees at a peripheral speed of 4 m/s. The bridging veins can be supposed to follow the brain matter passively[5] and will thus elongate as the brain is deformed. Fig. 6 indicates that a bridging vein elongated 20 % has only deviated about 34 degrees. A 45 degree deviation would correspond to 41 % elongation. Fig. 4 shows the stress versus time to rupture. The result is in agreement with the behaviour of other materials at dynamic loading.

CONCLUSIONS.

Ruptures of bridging veins within the brain matter near the mantel edge, i.e. gliding contusions, can occur in closed brain injuries as a consequence of angular acceleration. When subjected to angular acceleration the brain will be distorted due to shearing forces. This deformation is pronounced near the brain surface, causing the bridging veins to elongate to values exceeding their dynamic tolerance. The needed angular acceleration can easily be achieved during automobile collisions.

REFERENCES.

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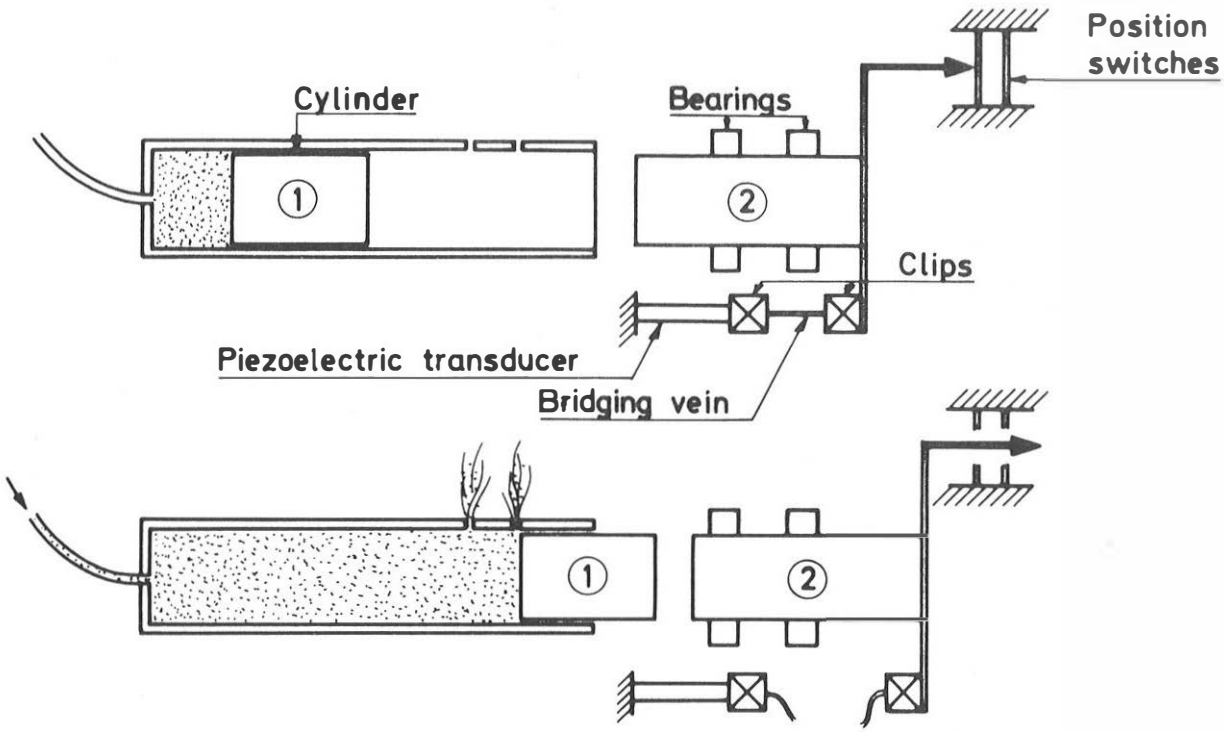
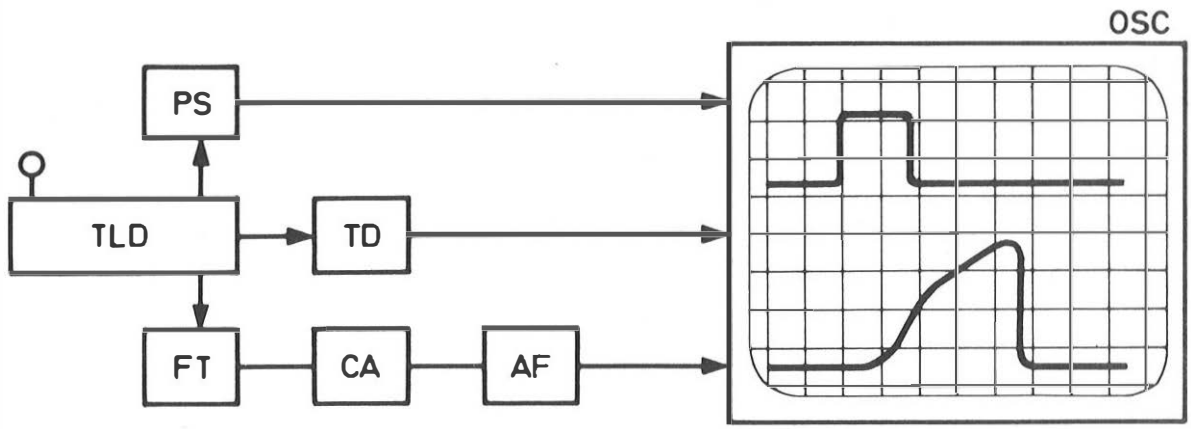


Fig. 1



PS : Position switches
TLD : Tensile loading device
TD : Trigger device
FT : Force transducer
CA : Charge amplifier
AF : Active filter
OSC : Oscilloscope

Fig. 2

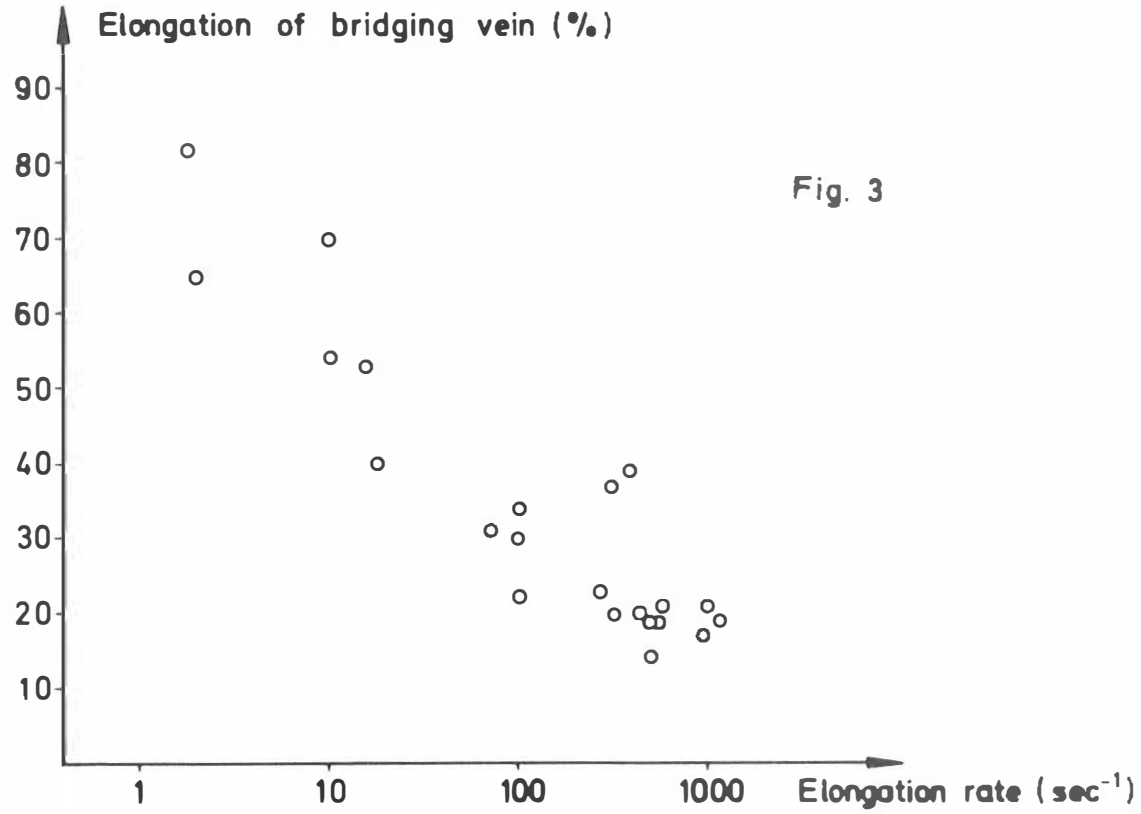

Elongation of bridging vein (%)
90
80
70
60
50
40
30
20
10
1
10
100
1000
Elongation rate (sec^{-1})

Fig. 3

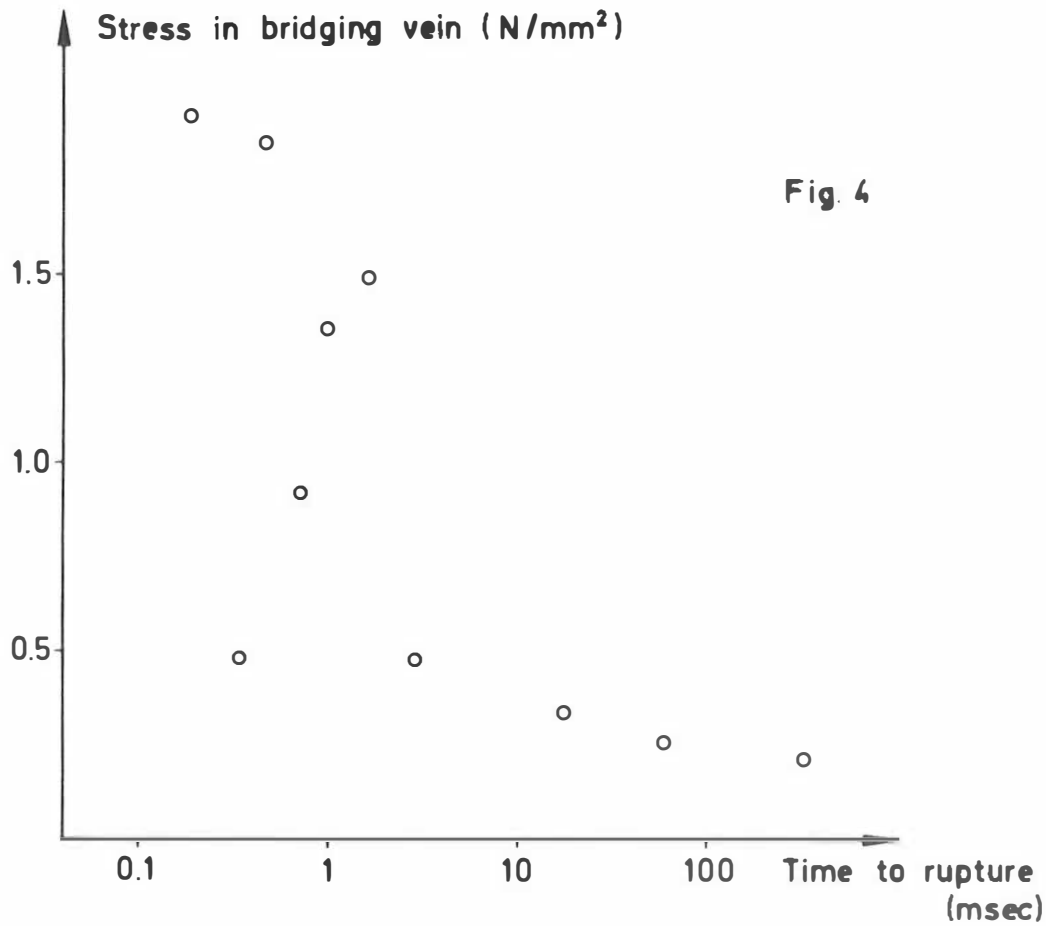

Stress in bridging vein (N/mm^2)
1.5
1.0
0.5
0.1
1
10
100
Time to rupture (msec)

Fig. 4

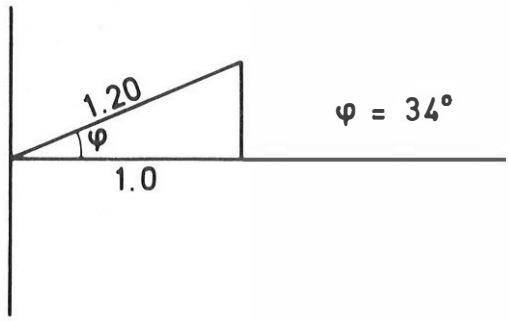


Fig. 5

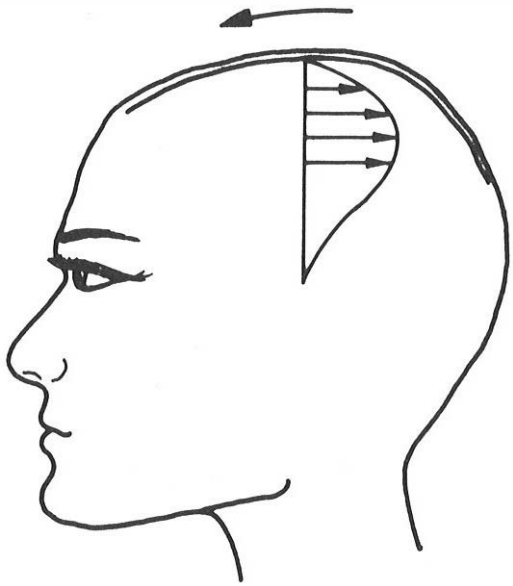


Fig. 6