EXPERIMENTAL BRAIN DAMAGE FROM FLUID PRESSURE DUE TO IMPACT ACCELERATION. STUDY OF EFFECTS OF "CONTRE-COUP TYPE" PRESSURE CHANGES INTRACRANIALLY IN RABBITS.

BY

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INTRODUCTION

At violent impact to the head various mechanical events are produced resulting in brain damage. Such events are: accelerations and deformations of the skull or movements at the cranio-spinal junction with pressure changes and displacement of the intracranial contents. For elaboration of proper protective devices the pathogenesis of brain lesions must be clarified.
Pressure changes elicited within the contents of an impacted "fluid filled" container such as the human skull have often been assumed as a cause of brain injury (Gurdjian et al. 1955 Hodgson 1968); deviations from the ordinary acceleration pressure pattern have been shown most interest (Lindgren 1966). In particular the subatmospheric pressure changes due to local skull deformation or part of the acceleration pressure pattern opposite the site of impact (contre-coup) have been considered as a major cause of intracranial injury (Unterharnscheidt & Sellier 1966). The "contre-coup" injuries have been assumed to occur at low subatmospheric pressure, causing tissue disruptions due to "cavitation" or some of its concomitant phenomena. (Unterharnscheidt & Sellier 1966, Hodgson 1970, Unterharnscheidt 1972). Other investigators have not believed in the occurrence of "cavitation" in tissues (Goldsmith 1966, 1972) but have advocated "the head rotation and skull distorsion hypothesis" stressed by Holbourn (1943) and Pudenz & Sheldon (1946) (cf. Ommaya et al. 1971).
With experiments in living animals simplified mechanical loading of the brain will be of value to elucidate the effects of acceleration "contre-coup" pressures particularly in relation to morphological changes (Stålhammar and Olsson 1975 a).
In animal experiments acceleration pressure changes are difficult both to elicit and to record partly because of the small size of the skull in most laboratory animals. A new experimental model has been designed to avoid such difficulties (Stålhammar 1975 a). When using such a model complicating mechanical factors must be avoided or analyzed; in the later case experiments have to be performed (Stålhammar and Olsson 1975 b) to study their significance . The aim of the present study was to:

1. design a model for production of intracranial impact acceleration fluid pressures with the "contre-coup" type pressures changes elicited intracranially in laboratory animals,
2. control and record extracranial mechanics in relation to intracranial mechanics in the developed model,

3. evaluate some physiological and morphological responses in relation to various extracranial and intracranial mechanical parameters produced in the model.

Basic idea of the model

A rigid fluid filled cylinder was connected with the skull cavity of a laboratory animal: the cylinder was impacted and the intracranial contents acted as a contre-coup end. Production of a pressure pattern similar to that encountered in human head at blunt impact would be possible.

The technique should be possible to apply to the rabbit because considerable information has previously been obtained in mechanical, pathophysiological and pathomorphological studies of experimental head injury on that animal in our laboratory (Lindgren & Rinder 1966, 1969, Rinder 1968, a, b. Rinder & Olsson 1968 a, b, Olsson et al. 1971).

MATERIAL AND METHODS

56 rabbits were used in the experiments. The animals were lightly anaesthesized when tested: three animals used for various intracranial pressure measurements were killed before the experiments.

Preparation for mechanical loading

Two modifications were used:

Type 1. Impact acceleration of the reinforced trephined skull for direct brain loading by fluid pressures (Fig.1). To a parietal hole a plexiglass tube ("T-tube" inner diameter 16 mm) was attached. The dura over the brain surface was removed.

Type 2. Impact acceleration of the reinforced intact skull. Basic preparation was similar in the two types with connection of the cylinder to the skull vault (Fig. 2).

Reinforcement of the skull - the entire skull vault was covered by an acrylate plate (excluding the parietal hole including 4-6 screws fixed to the bone. The intact skull was attached with a plexiglass plug ("skull adaptor" equipped with a light accelerometer) of the very same outer shape and size as the T-tube. The T-tube and the skull adaptor connected the skull vault with a plexiglass cylinder filled with physiological saline. The cylinder was 30 cm long and had an inner and outer diameter of 20 mm and 40 mm respectivly. The tube and skull adaptor respectively were directed from above in the sagittal plane, compare fig.1, 2.

An impact to the free end of the plexiglass cylinder was thus transmitted through the walls of the cylinder to the rabbit skull. The position of the animal at impact was similar in all experiments. It was lying on the table on the left side fixed only to the plexiglass cylinder by the T-tube or skull adaptor. The cylinder was held in a heavy vice fixed to an iron plate. The jaws of the vice were covered with plasticine.

Mechanical recordings

Acceleration (and velocity) was routinely recorded by a small accelerometer attached to the cylinder wall. In experiments for test of the uniformity of the mechanical response of the separate units of the system light accelerometers were fixed at the acrylate plate of the skull vault and at the skull base. Acceleration direction was kept unchanged ("traslational") by guiding the cylinder attached to the "T-tube" or "skull adaptor" fixed to the reinforced skull vault. Area of the transmitted impact and direction relative to the rabbit skull were uniform in all experiments .The total dislocation of the system was measured after each impact.

Pressure recordings in the contents were always performed in the T-tube and in impact end at direct loading of the brain with fluid pressures. In separate tests the intracranial pressure changes were recorded. In some experiments the pressure transducer was attached to the skull base itself positioned perpendicular to impact direction.

Physiological recordings.

Three physiological parameters, respiration, arteral blood pressure and pulse rate, commonly recorded for evaluation of functional disturbances in head injury experiments on anœsthesized animals were used (cf. Rinder 1969). Other signs were observed too: corneal and blink reflexes and the reaction to pain stimuli (tail, ear and paw pinch).

Morphological recordings.

Oedema, hœmorrhages and lacerations are commonly reported results of human and experimental head trauma (Unterharnscheidt 1972): the methods for studying these effects were directed to reveal vascular lesions. The same technique was used throughout the series. To study the presence of increased vascular permeability in the brain and in the spinal cord all animals were given a solution of Evans blue albumin (EBA) before the impact. The rabbits were allowed to survive for up to one hour after the impact. The animals were then perfused with formalin and a thorough morphological examination started. Skull bone fractures, bleedings in the cranial and spinal meninges were looked for. No fractures, however, were observed in the entire series. The surface and the coronary sections of the brain and spinal cord were also carefully checked for the occurrence of hœmorrhages, lacerations and areas of EBA-exsudation.

To reveal EBA-exsudation fluorescence-microscopical studies were done on frozen sections(Hamberger & Hamberger 1966, Steinwall & Klatzo 1967). In addition blocks were also embedded in paraffin and stained with hematoxyline-eosin and luxol fast blue-cresyl violet.

OBSERVATIONS AND COMMENTS

Mechanics

The acceleration " rise time" 0.1-0.4 ms in type I and 0.1-1.0 ms in type II, the peak and the duration of its positive phase could be varied on purpose by varying:

contact material - cork or paper 1-4 mm

impact velocity- directly depending on fall height (0.2-1.15 m) of pendulum

mass of impacting object - pendulum mass 2-7 kg

Typical recordings are reproduced in Fig.3-type I and in Fig. 4-type II. A short and pronounced positive acceleration phase was obtained. The duration was 1.0-1.4 ms in type I and 0.4-1.2 in type II. The peak of the cylinder acceleration was 160-520 g_n in type I and 400 - 2250 g_n in type II. By mounting the cylinder in plasticine no violent rebound effect was encountered. Thus a flat retardation phase was obtained. Velocity recordings were obtained from acceleration course. Maximum velocity change was 1.3-2.8 ms in type I and 2.2 - 4.8 ms in type II. The reliability of using cylinder acceleration as a reference when comparing signs of brain damage was supported by the following test: The skull vault accelerometer and the skull base accelerometer did show a similar (within $\pm$ 0.2ms) change of velocity irrespectiv of using a solid skull adaptor or the weaker T-tube for connection between the skull and the cylinder, at impact levels up to about 500 g_n (duration 1 ms).

The reliability of mechanical recording is discussed elsewhere. The total dis-

location of the cylinder was varied between 4-14 mm in type I and 11- 75 mm in type II.

Fluid pressures

Pressure changes are generated in human skulls at impact acceleration and is one intracranial parameter possible to record in a controlled moving system cf. Lindgren 1966.

Intracranial pressures are produced even in small animal skulls at impact acceleration. However, the magnitude of acceleration provided (about 2.000 g_n duration 0.7ms) to elicit biological effects of the type "concussive response" makes valid pressure measurements very difficult. By including the small skull cavity in a large fluid filled container at impact pole or counterpole a "concussive response" is elicited at lower levels of acceleration and reliable pressure mearurements may be performed (cf.Lindgren & Rinder 1966). In this model the rabbit brain was located at the counterpole - "contre-coup end". The general pressure pattern in a stiff fluid filled container is characterized by positive pressure at the impact site and subatmospheric pressures opposite: the latter is followed by a pressure rise at the end of the impact ("late positive pressure"). The pattern is modified by deformation accelerations probably involving local and remote flow of the contents (Lindgren 1966).

In this model possibility to uncontrolled deformation was reduced by reinforcement of the skull and choosing a stiff and short cylinder. However, partly quantified displacement of the vessel contens was obtained by introducing an air bubble of known volume (50, 100 150 mm^3) at the impact end. The pressure recorded adjacent to this indicates the course of volume change of this bubble. At an acceleration level of about 200 g_n the air bubble will be compressed to 1/6-1/7 of its initial volume. This corresponds to a displacement of the cylinder contents of 40-130mm^3. The intracranial initial negative pressure peak by means of the air bubble could be varied on purpose. Increased initial negative pressure peak was obtained at increased volume of the air introduced (Fig.5) Relation between pressure in the T-tube and intracranial is showed in Fig.6. General pressure pattern is locally modified when a closed container is opened (cf.Lindgren 1966 , Hayashi 1969 a b). Foramen magnum is the natural main opening in this model. In Fig. 7 is illustrated the pronounced initial negative peak obtained when foramen magnum is closed.

The studies showed

acceleration - velocity - dislocation of the rabbit head could be varied and predicted

skull deformation was minimized

reliable intracranial pressure measurements could be performed

varied intracranial pressure changes of "contre-coup type" including subatmospheric pressure transients near - 1 atm could be obtained

to some extent intracranial tissue displacement could be estimated.

Physiology

Impact acceleration of the reinforced rabbit skull did not elicit any "concussive response" (respiratory arrest longer than 3 sec. or blood pressure rise) with cylinder peak acceleration below about 1000 g_n (duration 1 ms), peak velocity below 3 m/s or total cylinder dislocation below 30 mm (Stålhammar , 1975 b). In tests with direct brain loading, with cylinder acceleration pressures lower levels of cylinder acceleration were used: peak about 200 g_n (duration about 1.2 ms) maximum velocity change about 1.5 m/s and total dislocation of 6 mm. Thus the pathophysiological effects produced might mainly be related to direct fluid pressure loading of the brain.

When cylinder acceleration was kept similar (180 - 220 g_n) the degree of concussive response was related to the peak of the initial subatmospheric pressure in the T-tube and the "late positive" pressure impulse Fig.8 (Stålhammar 1975 b)

Comments

By the results obtained the frequently debated question on relations between intracranial acceleration pressure changes of different character and flow in foramen magnum have been elucidated. The magnitudes of the contre-coup pressures, part of the acceleration pressure pattern, were increased by a graded injection of air volumes at impact end . The effect from the impact end positive pressure caused displacement of cylinder contents. The probable effect of displacement of brain in the parietal opening will be transmitted to the area of foramen magnum : it will be responsible for the physiological response recorded. Such relation between the " concussive response" and the flow in foramen magnum has previously been shown by Rinder (1969) for positive fluid volume pressure pulses. He found a correlation between the peak of the positive pressure pulses and the grade of "concussive response". There was also a correlation to the pressure impulse (Rinder personal communication).

Morphology

There is no unanimity concerning the important question of pathogenesis of the contre-coup lesions. Ommaya (1971) advocates "skull distorsion/rotation hypothesis" summing up results from experiments with translational and rotational traumata. Goldsmith (1966, 1972) and Ommaya (1970) repeatedly have pointed out the non-existence of evidence for cavitation occurring in living animal brain.

Unterharnscheidt (1963) in impact acceleration tests on rabbits and cats found "primary cortical cavitation traumata at the site of impact and antipole in all animals subjected to impact resulting in acceleration corresponding to 400 g_n".

Hashizume (1972) in a monkey produced a contusion in the parieto-occipital region which he considered contre-coup to a frontal impact, at head acceleration of of 284 g_n (duration 10 ms). He related this lesion to the occipital pressure changes showing a peak near - 1 atm.

Ommaya et al. (1971) could produce neither coup nor contre-coup lesions in frontal impact when the skull bone did not fracture. But in 32 monkeys receiving concussive occipital impacts 22 showed microscopic lesions at coup and contre-coup site even when no fracture was produced.

Unterharnscheidt & Higgins (1969) emphasized that the cortical contusions produced by non-deforming rotational acceleration show a pattern and quality of histopathological lesions quite different from those seen in pure translational traumata where the "cavitation mechanism"were the most important. They found that lesions produced by rotation were venorhectic in character and did not show the typical pin-point hæmorrhages seen in "cortical cavitation injuries" produced by translational acceleration.

Obviously these problems should be attacked in strictly standardized experiments on living animals if it should be possible to link any single mechanical event to the occurrence of tissue damage. The present model was designed to give such well-controlled experiments with recording of some extracranial and intracranial parameters. The occurrence of morphological lesions were studied on animals living about 60 min. after the impact.

The studies showed: Impact acceleration of the reinforced intact rabbit skull did not result in any significant morphological alterations intracranially irrespective of magnitude of acceleration and dislocation used. Thus, morphological changes recorded at lower (cf.above) acceleration magnitudes as in type I might be ascribed mainly to the effects of direct brain loading by fluid pressures. Morphological alterations were found in the lower brain stem and upper cervical cord (CI - CIII), but not in other parts of the brain excluding effects at the parietal hole (cf. Fig.9,10,11).

Comments

A statement that tissue displacement may cause tissue damage, in this case in the brain stem region, may seem justified. However, as seen above the genesis of contre-coup lesions seems less clear and we could not produce such alterations. The type of mechanisms and pressure changes discussed as their cause must therefore be briefly commented.

The contre-coup pressure phenomena studied by Gross (1958a,b) included high frequency acceleration disturbances in the shell explained by collapse of cavitation "bubbles". Lindgren (1966) found such high frequency disturbances of the pressure transducers near the lowest resonance frequency: however, he put more significance to the initial subatmospheric pressure plateau-level obtained at -0.8 - -0.9 atm as a possible indication of cavitation in the fluid. Hodgson (1968) also discussed similar phenomena. However, he did not report any pathologic findings in relation to this (cf. Hashizume 1972).

The mechanics (cf. Persson 1974) and material properties - gas contents, viscosity etc. influencing the appearance of cavitation will not be discussed here. However, in our animal experiments emphasis has been made on producing the " contre-coup pressure" complex, including initial subatmospheric and late positive pressure. It must also be stressed that in biological tissues volume changes and tissue displacements only can be minimized: this was evident from the experiments with the plexiglass skull, and with closed and open foramen magnum in the animal skull.

It seems clear that the routine experiments with the whole skull cavity included in the contre-coup end of the system should produce some morphological alterations if the pressure change complex per se had some injurious effect. The conclusion is that some other factors must be added to produce tissue damage: one factor is the deformation probably occurring and trans mitted from the impact site in the human skull.

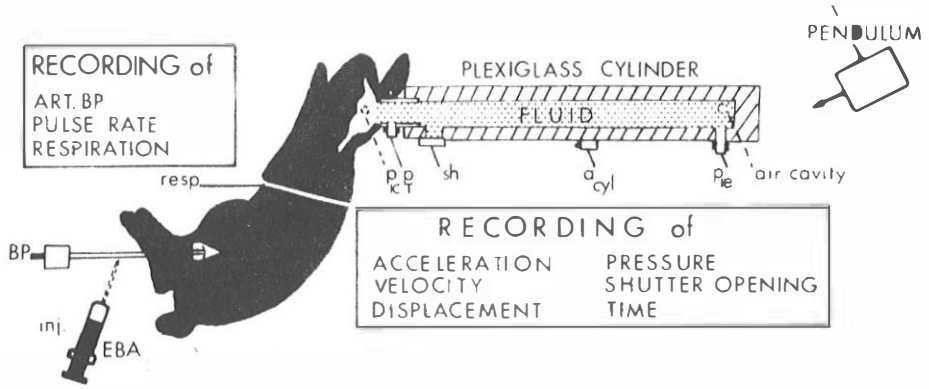

Fig 1. *Drawing of experimental arrangement. p_{ic}, p_T and p_{ie} – pressure transducers in the skull cavity, the T-tube and at impact end respectively. sh – shutter, a_{cyl} – accelerometer fixed at the cylinder wall.*

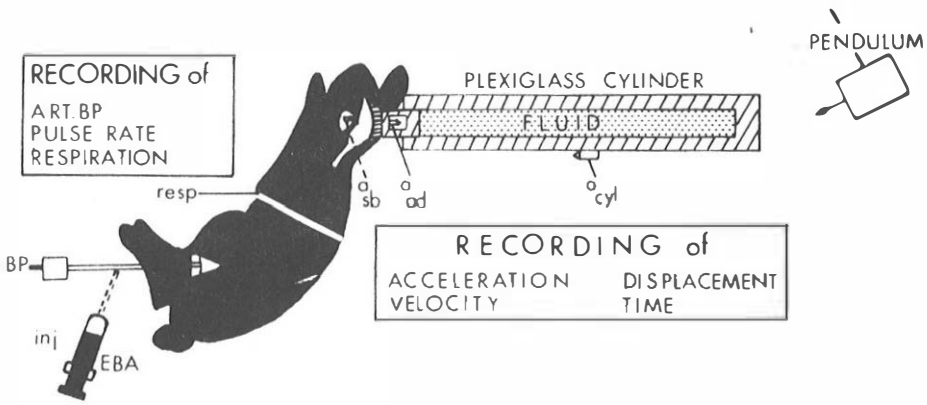

Fig 2. *Drawing of experimental arrangement. a_{sb} – skull base accelerometer, a_{ad} – skull adaptor accelerometer, a_{cyl} – accelerometer fixed at cylinder wall.*

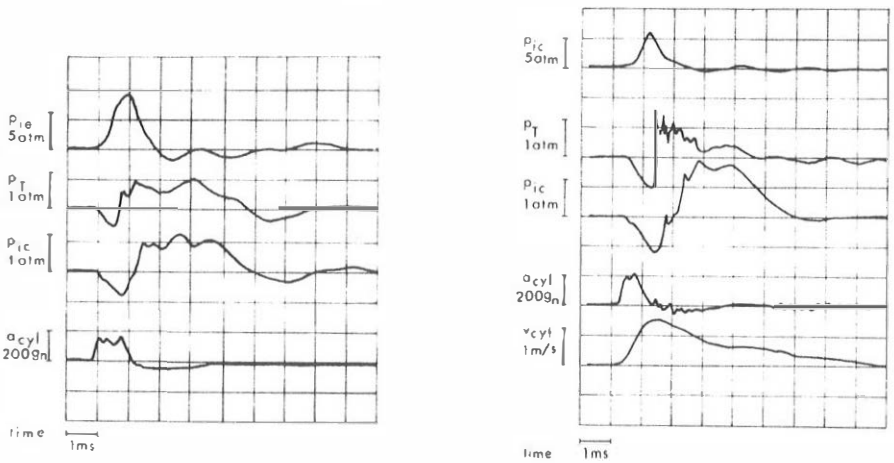

Fig 3. *Recordings of cylinder acceleration (a_{cyl}) and pressures at impact end (p_{ie}), in T-tube (p_T) and in the skull (p_{ic}) and a velocity recording (v_{cyl}). a) air bubble 50 mm^3. b) air bubble 150 mm^3.*

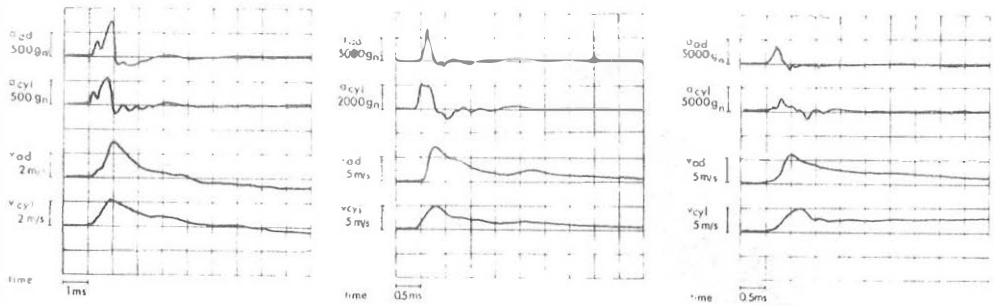

Fig 4. *Typical recordings of acceleration and velocity for cylinder and skull adapter accelerometer at various magnitudes of impact. a) No. 80, b) No. 96, c) No. 90.*

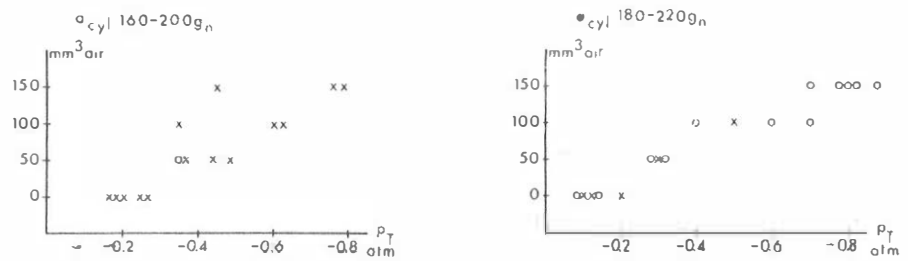

Fig 5. *Relation of initial peak negative pressures in the T-tube (p_T) to varying volume of air bubble at impact end at peak cylinder acceleration 160-210 g_n. a) obtained at simultaneous intracranial measurements (cf. fig. 7 and table 1). b) obtained in routine experiments with simultaneous recording of biological effects (cf. table 2). X – shutter closed. O – shutter opened.*

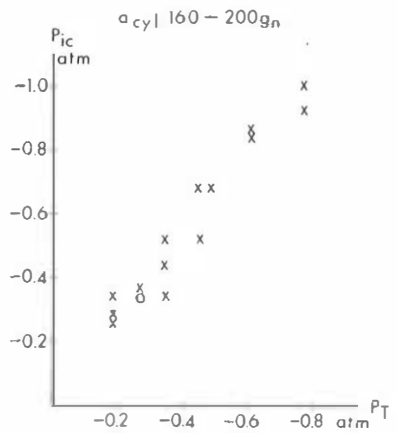

Fig 6. *Relation of initial peak negative pressures recorded intracranially (p_{ic}) and in the T-tube (p_T) at peak cylinder acceleration of 160-200 g_n. (cf. fig. 6 a*

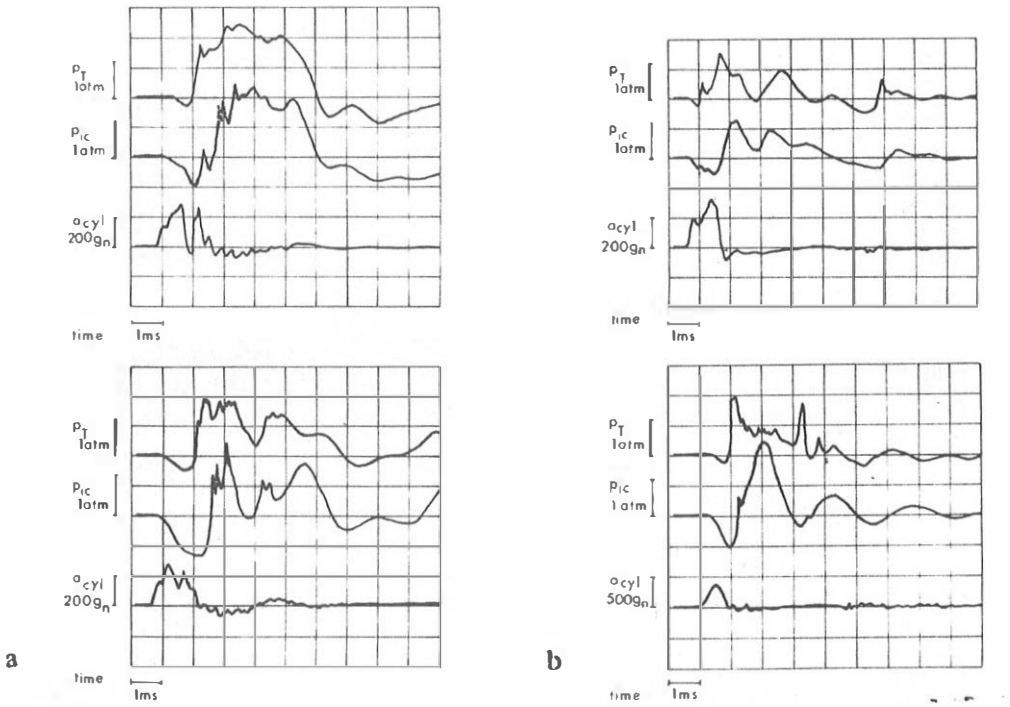

Fig 7. *Acceleration and pressure recordings showing the effects to the intracranial pressure course (p_{ic}) at varied closure of foramen magnum. a) plexiglass model of the rabbit skull. b) cadaver skull. Upper tracings show pressure recorded at »open» foramen magnum. Lower recordings show the increased initial negative peak when foramen magnum is tightly closed.*

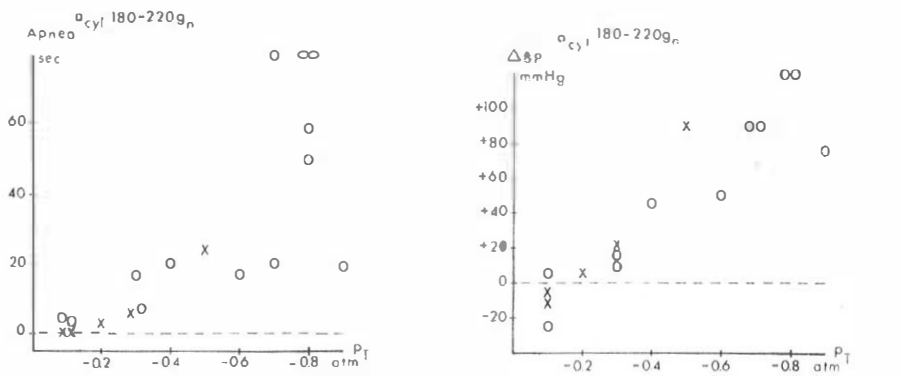

Fig 8. *Relations of length of apnea and maximum initial change in arterial blood pressure to initial negative pressure peak (p_T) when peak cylinder acceleration (a_{cyl}) was kept at 180-200 g_n. X-shutter closed. O-shutter opened.*

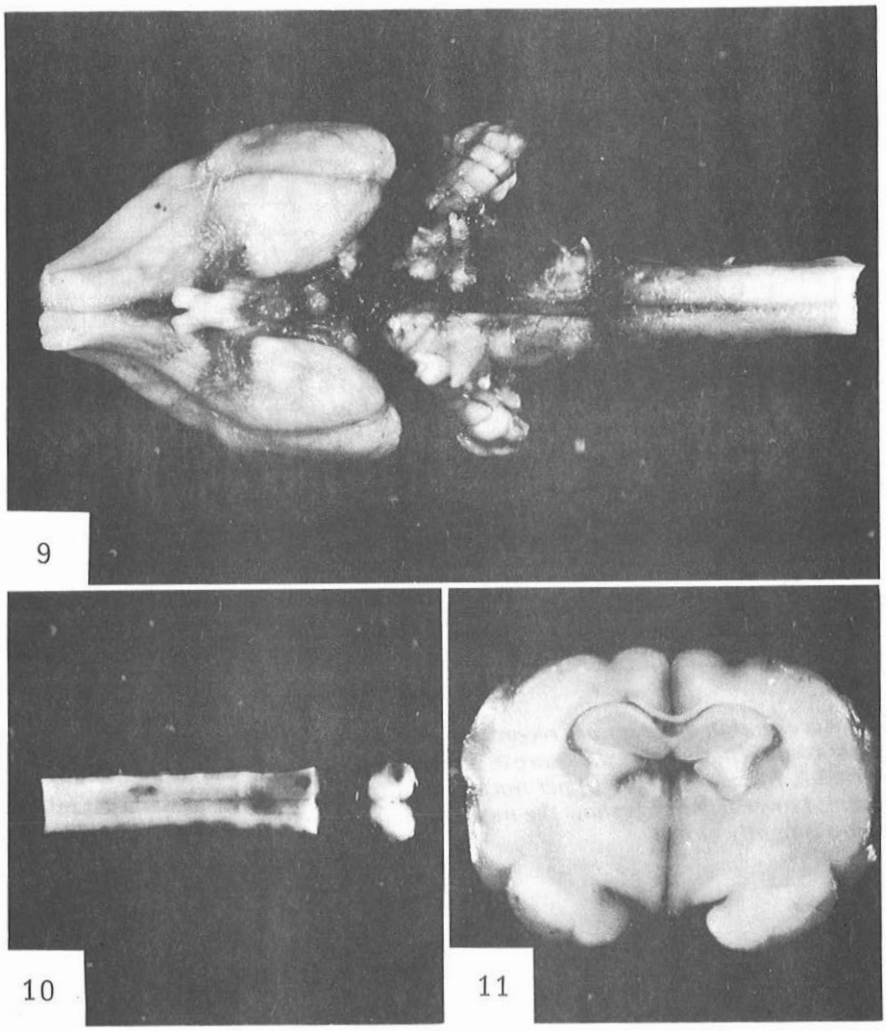

Fig 9. *Ventral view of a rabbit brain exposed to a sudden negative pressure of about −1 atm. (101). EBA given before the impact to visualize changes in vascular permeability. Note absence of EBA-exsudation in the temporal lobes.*

Fig 10. *Spinal cord from a rabbit (100) subjected to a negative pressure of −1 atm. Note exsudation of EBA in the upper part of the cervical cord (to the right).*

Fig 11. *Coronal section from a rabbit (102) exposed to a negative pressure of −1 atm. There are no signs of haemorrhages or of EBA-exsudation.*

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