CORRELATION OF THE INTRAOCULAR PRESSURE WITH INCREASED INTRACRANIAL PRESSURE IN RABBITS

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Abstract - Although measurement of intracranial pressure by noninvasive methods has been suggested, but mainly invasive methods are used for this purpose. Increase in episcleral venous pressure can be expected to result in a linear increase in intraocular pressure. Congested ocular veins with capillary leakage and hemorrhage are seen when the ICP is increased, thus theoretically measurement of Intraocular pressure can be a procedure for estimation of the ICP. This study was performed to find whether there is any relationship between intraocular pressure and ICP, so we used 12 albino rabbits in two divided groups. Our study was not designed to elucidate the mechanism of change but merely to record any changes observed. All measures except an increase in ICP were applied on the test group as well as on the control group. After general anesthesia with the combination of ketamin, rampune, and pentobarbital, a burr hole was made in the lambda region of the skull and a cannula was placed in the subdural space. The ICP in the test group increased up to 15 mmHg and was constant throughout the experiment. Intraocular pressure was measured by Schiotz tonometers after general anesthesia, after cannulation of the skull, and immediately after increasing the ICP which was repeated in 15 minutes interval for 4 hours. There was no statistical difference between the two groups (P: 0.997). Results show that neither cannulation nor general anesthesia for 4 hours produce alteration in IOP in the control group nor increasing of the ICP to level of 15 mmHg produces any alteration in IOP on the test group.

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INTRODUCTION

Intracranial pressure (ICP) monitoring has become a useful tool in the management of patients with brain injury, cerebral edema, and progressive intracerebral hemorrhage. Intracranial pressure monitoring can detect elevation in pressure before secondary brain injury occurs. In addition, monitoring helps as a guide to medical therapy and contributes to the estimation of prognosis (1). ICP was found to be one of the best predictors of outcome in head injury (2). Available techniques of monitoring, with few exceptions

(3,4,5,6,7), are invasive and associated with complications such as infection, hemorrhage and herniation. The cost and risks of invasive methods are justified in patients likely to develop intracranial hypertension as determined by history, neurological examination and radiological studies. The criteria for the duration of ICP monitoring are not firmly established (9).

In large increments of the pressure in the extraocular veins part of the pressure will be transmitted into the intraocular veins, causing venous congestion. Congested ocular veins with capillary leakage and hemorrhage are seen when the ICP or the pressure within the sheath of optic nerve is increased (10). Increases in episcleral venous pressure can be expected to result in a linear increase in Intraocular pressure (11). Elevation of episcleral venous pressure will result in a decreased outflow of aqueous through the trabecular meshwork, until the IOP has risen to a new value, reestabilishing the flow at a new steady state (12).

Based on this constellation of data, we studied the correlation between IOP and increased ICP in rabbits as a noninvasive method for estimation of ICP changes.

MATERIALS AND METHODS

In this study 12 female albino rabbits with average weight of 2.7 kg were divided in two identical groups. All measures except increas in ICP were applied to the control group as well. Each experiment was started at 8 am, (in avoidance of diurnal variation of IOP as a confounding factor (13), rabbits were anesthetized with intraperitoneal injections of Ketamin - Rampune combination in dose of 75 mg/kg and 4 mg/kg respectively which was followed, after 15 minutes by pentobarbital in a dose of 40 mg/kg by the same route, then the animals were tracheostomatized and intubated. Arterial and venous cannulation in the inguinal region was performed for blood pressure monitoring and pentobarbital in a maintenance dose 4 mg/kg per hour, was injected. Then after making a fine scalp incision, a burr-hole of 2mm in diameter was created in the skull at the midline of the lambda region.

A suitable diameter heparinised saline filled cannula was left in the subdural space, sealed around by bone wax and glue to prevent leakage. This cannula was connected to one arm of a Y shaped plastic tube which was connected to the transducer of an electrophysiograph (Beckman, USA) for recording of ICP changes. The other arm of the Y shaped tube was connected to a bottle of saline solution in the test group, the ICP was increased to 15 mmHg which was regulated throughout the experiment to about 15 mmHg by changing the height of the bottle.

Selection of the level of pressure was based on the pilot studies of this investigation which demonstrated the normal ICP of rabbits to be about 1 mmHg, Sufficient pressure was produced for overcoming the pressure drop of ocular veins as they pass through the sclera.

In each experiment the animal was placed horizontally in the prone position with the head and neck rotated to the left side. During the procedure, IOP was measured by Shiotz tonometer immediately after anesthesia, after subdural cannulation, and immediately after increasing the ICP which was repeated in 15 minute intervals for 4 hours. The animals were finally killed by injection of potassium chloride while they were anesthetized.

RESULTS

12 experiments were performed. All animals were in good condition with a mean arterial pressure of 100 mmHg. The normal IOP ranged from 16-18 mmHg. The IOP did not change in any step of the experiment, neither in the control group non in the group with artificially increased ICP. The IOP in the test and control group were not statistically different. (Pvalue: 03.997) the results summarized in table 1.

Table 1. Correlation between IOP and ICP in rabbits

Group	Control		Test	
Step	ICP(mmHg)	IOP(mmHg)	ICP(mmHg)	IOP(mmHg)
After anesthesia	1	17.7 ± 0.75	1	17.33 ± 1.03
After cannulation	1	17.7 ± 0.75	1	17.33 ± 1.03
Immediately after ICP elevation	1	17.7 ± 0.75	1	17.33 ± 1.03
One hour after ICP elevation	1	17.7 ± 0.75	15	17.33 ± 1.03
Four hours after ICP elevation	1	17.7 ± 0.75	15	17.33 ± 1.03

DISCUSSION

The IOP is maintained within a fairly narrow range by a complex and dynamic equilibrium in which a nearly constant rate of aqueous humor production is matched by a nearly constant rate of aqueous humor escape from the eye through drainage pathway. Aqueous flow from the citiary processes into the posterior chamber is not sensitive to IOP (14). The principle outflow pathway for aqueous is the pressure dependent flow through the trabecular meshwork. Aqueous exits the eye through the trabecular meshwork and through that passes into the Schlemm's canal (13).

Once within the canal, aqueous flow through connector channels into the episcleral veins is determined principally by the level of episcleral venous pressure. These veins connect with vortex veins and these in turn with superior and inferior ophthalmic veins and the latter connect with cavernous sinus. A good estimate of the pressure in the veins leaving the eye can be made by measuring the IOP, since these two values are almost equal at normal and high IOP levels (15,16). Outside the eye the pressure in the ocular veins is lower than the IOP. This means that there may be a pressure drop in the ocular veins as they pass through

the sclera, resulting in partial collapse of the veins (11). Known factors that affect the IOP are hyperthermia, pulse rate, obesity, hemoglobin concentration, sex, age, refractive errors, hyperthyroidism, pregnancy, diabetes mellitus, hypoglycemia, and drugs such as hypnotics and glucocorticoids (13,17,18). In clinical studies of the correlation between IOP and increased ICP, other factors such as location and severity of trauma, location of the space occupying lesion, the nature of underlying disorders leading to elevation of ICP, and associated diseases per se or in conjunction with the factors mentioned above, may affect the IOP. A logical way for decreasing the effects of these factors was increasing the ICP in laboratory animals. Although general anesthesia and hypnotic drugs may decrease the IOP (17) and ketamine can increase it (19), the presence of a control group with identical condition except increased ICP could eliminate these effects.

Likely causes for the of the expected increment in the IOP in response to elevated ICP are as follows: (1). The compartmentalization of increased ICP (20) around the cannulation sitemay be responsible. The sudden increase in episcleral venous pressure such as that produced by valsalva maneuver is expected to result in an immediate rise in IOP (12, 2). Since small increments in the extraocular venous pressure tend to reverse the collapse of ocular veins as they pass through the sclera without affecting the intraocular venous pressure, the amount of increment of the ICP not sufficient to elevate extraocular venous pressure to the level that overcomes this collapse. (3) Duration of each experiment may be shorter than it actually should have been.

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