# Experimental Study on Ultrasonic Attenuation in the Brain

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An experimental study was made with rabbits to see what factors participate and how they participate in the change of ultrasonic attenuation in the brain. The experiment was carried out by means of bilateral ligation of the jugular veins, auto-rebreathing of exhaled gas, blocking carotid blood flow and injection of physiological salt solution upon puncture of the posterior cistern, and relation of the change particularly of water content of the brain, cerebral blood flow and CSF pressure with the change of ultrasonic attenuation in the brain was studied. The conclusions obtained as follows.

1) The increase of ultrasonic attenuation in the brain is participated by either of the decrease of water content of the brain, the decrease of cerebral blood flow, or the increase of CSF pressure.

2) The decrease of ultrasonic attenuation in the brain is participated by either of the increase of water content of the brain, the increase of cerebral blood flow, or the decrease of CSF pressure.

## INTRODUCTION

Medical application of ultrasound has made rapid progress by the studies of many investigators ever since DUSSIK<sup>7)</sup> reported on its possibility in 1942 after his experiment with transmission method. Recently, ultrasound is used for diagnosis of intracranial disease<sup>22)30)</sup> and of the diseases of the mammary gland<sup>10)41)</sup>, digestive organs<sup>14),35)</sup>, circulatory organs<sup>27)39)</sup>, thyroid glands<sup>17)18)</sup>, lungs, mediastinum<sup>43)</sup>, etc., and for stereotaxic neurosurgery<sup>30)</sup> and therapy of malignant tumors<sup>34),38)</sup>.

Along with the advancement of medical application, quantitative study of ultrasound has become prosperous in the field of neurosurgery.

This is an attempt to serve in the diagnosis of organic or func-

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tional change of the organ upon measuring attenuation of ultrasonic energy. Basic studies on ultrasonic attenuation in the biological tissue have been reported by  $H_{UETER^{15(16)}}$ ,  $D_{UNN^{6)}}$  and  $I_{SHIKAWA^{19)}}$ . Their method have been utilized for edema of the brain<sup>32)</sup> by means of measuring ultrasonic attenuation in the brain, for measurement of cerebral circulation<sup>24)</sup> and for diagnosis of epilepsy<sup>20)</sup>. However, much is unknown about the nature of ultrasonic attenuation. Even with attenuation in the brain, many problems remain unsolved as to what type of intracranial change (organic or functional) is indicated by the attenuation.

Many factors of ultrasonic attenuation may be anticipated such as scattering due to transmitting distance, reflection on the surface of the medium, and absorption due to viscosity. Various attempts were made to clarify the nature of ultrasonic attenuation from the standpoint of physics. In 1952, GIERKE et al.<sup>11)</sup> reported that ultrasonic attenuation is similar to that in vibration of elastic object. In 1953,  $E_{PSTEIN}^{9)}$  indicated that ultrasonic attenuation is affected by STOKES type attenuaton.

In 1953,  $C_{ARSTENSEN^{4)}}$  reported that ultrasonic attenuation is mostly affected by the volume of protein in the blood. Nevertheless, none of these reports is adequate to clarify the nature.

In Japan, report on ultrasonic attenuation in the brain was first made by  $T_{ANAKA^{37)}}$  in 1953, followed by many other reports with various results. Even with a single phenomenon of edema of the brain, there has been no determinate view. I<sub>SHIKAWA<sup>19)</sup></sub> (1964),  $K_{ANAYA^{21)}}$  et al. (1965) and  $M_{ARUTA^{25)}}$  et al. (1965) reported that edema increases attenuation.

 $N_{AITO^{280}}$  et al. (1964), YOSHIMURA<sup>44)</sup> (1965) and TORII<sup>40)</sup> et al.(1966) indicated that brain edema decreases attenuation.  $H_{ASEGAWA^{13)}}$  (1965) reported that edema does not affect attenuation at all.

Ultrasonic attenuation in the brain seems to be affected by various factors, either individually or in complex, such as cell density, cell distribution (tumor), water content of the brain (edema), blood flow and intracranial pressure<sup>23)</sup>. Hence, the author conducted various experiments in an attempt to clarify the relation of ultrasonic attenuation in the brain with three factors, namely, intracranial pressure, water content of the brain and cerebral blood flow, which seem to be most affected by the disease of the brain.

## I. EXPERIMENTAL METHOD

1. Experimental Animals

Seventy-eight adult rabbits each weighing about 3 kg were used (anesthetized by intravenous injection of Nembutal 1 mg/kg and fixed in prone position). A burr-hole was provided at the right

parietal bone.

- 2. Measuring Devices
  - 1) Medical analyser: USZ-10 (JRC)
  - 2) Step-attenuator: NJZ-109 (JRC)
  - 3) Echo selector: SSZ-41 (JRC)
  - 4) Automatic recorder: The one for Texas type electromagnetic flowmeter was used.
  - Fig. 1 shows the block diagram of the measuring devicces.

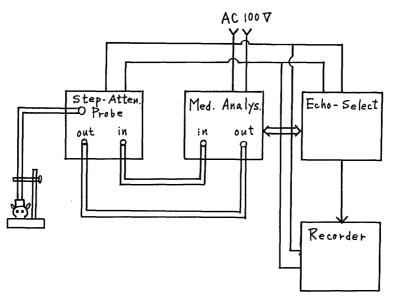


Fig. 1. Block diagram

- 3. Items and Methods of Measurement
  - 1) Blood pressure: Femoral artery canulation method
  - 2) Water content of the brain: Cobalt chloride method<sup>2)</sup>

HATSCHECK'S cobalt chloride method conducted by  $A_{MPO}$  was employed. In this method, the brain was removed after cutting the carotid artery and discharging the blood, and was fixed in 25% formalin for three days. Then free water and bound water were measured in the following procedure.

- a) Immerged in 10% cobalt chloride for 8 to 12 hours until the color turned red and then dehydrated by filter paper and weighed  $\cdots$   $P_0$
- b) Dried at  $30^{\circ}$ C for 12 hours, dehydrated (blue in color) and weighed .....  $P_1$
- c) Dried at 100°C for three days (grayish black in color, solid) and weighed ………  $P_{\rm 2}$

Free water = 
$$\frac{P_0 - P_1}{P_0} \times 100 \, (\%)$$

Bound water =  $\frac{P_1 - P_2}{P_0} \times 100$  (%) Total water = Free water + Bound water

3) Common carotid blood flow: bubble flowmeter

A by-pass of the common carotid artery with a U-shaped glass tube of known capacity was made and upon inserting about 0.2 ml of bubble from the central side, the passage speed of the bubble in the U-shaped tube was measured and the blood flow was calculated according to the following equation.

- Blood flow/min = Capacity of U-tube (ml)/Passage speed (sec) × 60
- 4) Cerebrospinal fluid (CSF) pressure: Posterior cistern puncture method
- 5) Exhaled gas partial pressure: Gaschromatography
- 6)  $PO_2$ ,  $PCO_2$  of arterial blood: Astrup micro-method
- Ultrasonic attenuation in the brain: Ultrasonic pulse reflection method<sup>5</sup>
- \*(1) Ultrasonic probe and its fixation

The probe used was a flat round probe of 5 Mc made of barium titanate measuring 10 mm in diameter (JRC, common use for transmitting and receiving). In order to eliminate measurement error due to the device, an apparatus as shown in Photo 1 was devised. A burrhole measuring 12 mm in diameter was provided at the right parietal bone and the probe was fixed in such manner that the ultrasonic beam axis may be rectangular to the base of the skull at the position where the end of probe contacts the dura. Liquid paraffin was used as

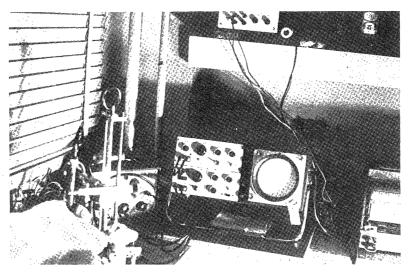


Photo 1. Measuring devices

coupling medium at the contacting surface between the probe and the dura.

- \*(2) Expression of ultrasonic attenuation in the brain (Fig. 2; Photo 2, 3, 4)
  - a) Ultrasonic output of the device was maintained constant throughout the course of experiment.
  - b) The height of reflection echo to be measured was previously adjusted by the step attenuator to be almost saturated with the incident echo. This height was used as the standard.
  - c) The recorder was arranged to record the peak of reflection echo and was calibrated at the intervals of 0.5 db through stepattenuator before experiment. Thus, the real value was to be obtained on the recorder even in the reflection echo on the oscilloscope was saturated.
  - d) The change of attenuation was evaluated by the change in height of reflection echo: the reflection echo higher than standard line was evaluated to indicate a decreased attenuation (loss)

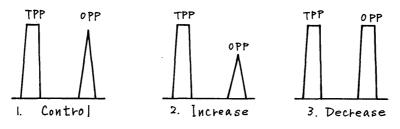


Fig. 2. Expression of ultrasonic attenuation

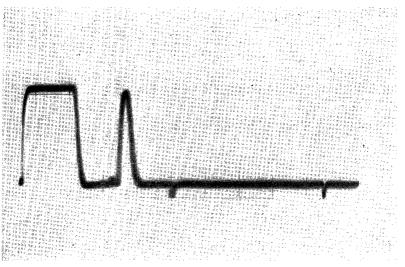


Photo 2. Control

of ultrasonic energy; on the contrary, the reflection echo lower than the standard line was evaluated to indicate an increased attenuation. Photo 2, 3, 4 illustrate the above procedure.

# \*(3) Iufluence of anesthesia

Ultrasonic attenuation in the brain showed little change in the control group of only Nembutal anesthesia.

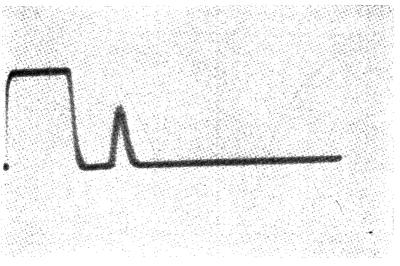


Photo 3. Increase

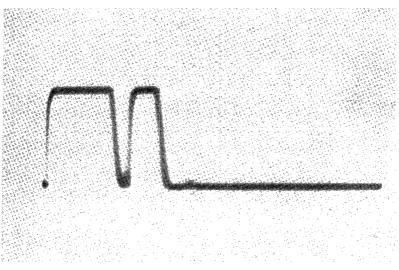


Photo 4. Decrease

## **II. EXPERIMENTS AND RESULTS**

Experiment 1) Change of ultrasonic attenuation in the brain due to bilateral ligation of the jugular veins<sup>1)</sup> (Fig. 3, 4; Tables 1, 2, 3; Photos 5, 6, 7)

a) Bilateral ligation of the jugular veins was performed and attenuation was measured at intervals of 15 minutes during the period

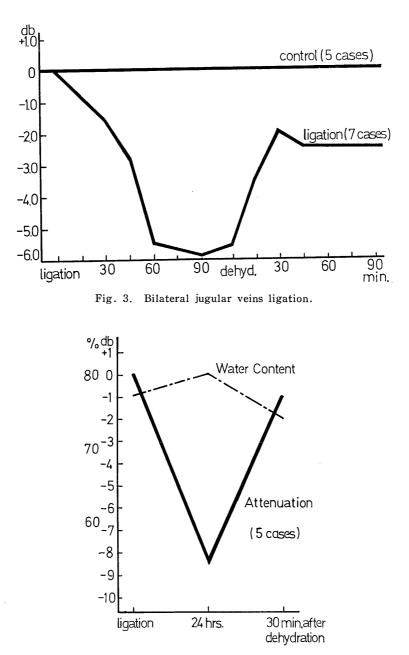


Fig. 4. Water content and attenuation after ligation and dehydration.

of 90 to 120 minutes. Then, upon dehydration by means of intravenous injection of Fructon-M (10 cc/kg), attenuation was measured again at intervals of 15 minutes during the period of another 90 to 120 minutes.

## **RESULTS**:

Attenuation value following bilateral ligation of the jugular veins gradually decreased in all cases reaching the lowest level of -5.8 db in 90 minutes. The injection of Fructon-M resulted in urination of 60

| Tab.1. |     | cal water content      | 24hrs  |    | bilat. | water content,<br>jugular veins | Fruct | on—   | M inj | ter content,<br>ection after |
|--------|-----|------------------------|--------|----|--------|---------------------------------|-------|-------|-------|------------------------------|
| no.    | 1.  | 78.18%                 | ligati | on | 1      |                                 | 24hrs | . lig | ation |                              |
| no.    | 2.  | 76.51                  | no.    |    | 1.     | 79.84%                          | no.   | 1.    |       | 74.89%                       |
| no.    | 3.  | 76.37                  | no.    | :  | 2.     | 80.30                           | no.   | 2.    |       | 75.41                        |
| no.    | 4.  | 74.57                  | no.    |    | 3.     | 78.84                           | no.   | 3.    |       | 75.22                        |
| no.    | 5.  | 76.38                  | no.    | 4  | 4.     | 79.94                           | no.   | 4.    |       | 74.10                        |
| no.    | 6.  | 76.24                  | no.    | ļ  | 5.     | 79.01                           | no.   | 5.    |       | 75.00                        |
| no.    | 7.  | 77.78                  | no.    | (  | 6.     | 78.85                           | no.   | 6.    |       | 75.23                        |
| no.    | 8.  | 78,06                  | no.    | 1  | 7.     | 78.73                           | no.   | 7.    |       | 74.81                        |
| no.    | 9.  | 77.13                  | no.    | ł  | 8.     | 79.81                           | no.   | 8.    |       | 76.43                        |
| no.    | 10. | 74.60                  | no.    | 9  | 9.     | 79.65                           | no.   | 9.    |       | 76.25                        |
| no.    | 11. | 77.73                  | no.    | 10 | 0.     | 79.46                           |       |       | M=7   | $75.32 \pm 0.24\%$           |
| no.    | 12. | 75.20                  |        |    |        |                                 |       |       |       | 5.52±0.24/0                  |
| no.    | 13. | 78.43                  |        |    | Μ      | $=79.44 \pm 0.17\%$             |       |       |       |                              |
| no.    | 14. | 74.33                  |        |    |        |                                 |       |       |       |                              |
| no.    | 15. | 73.81                  |        |    |        |                                 |       |       |       |                              |
|        |     | $M = 76.42 \pm 0.38\%$ |        |    |        |                                 |       |       |       |                              |

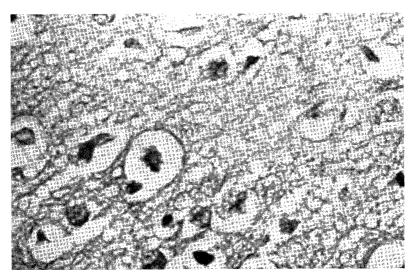


Photo 5. Control

to 100 cc in 30 minutes. The attenuation value began to increase reaching  $+2.5\sim3.0$  db in 60 to 90 minutes (Fig. 3).

b) The change of attenuation was observed for 24 hours after bilateral ligation of the jugular veins thereafter effect of dehydration due to Fructon-M was studied.

Measurement of water content after 24 hour ligation was made with other rabbits. Histological specimens were also prepared.

## **RESULTS** :

Histological findings: The cerebral tissue of rabbit after 24 hour

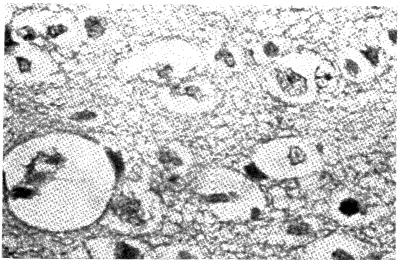


Photo 6. Edema

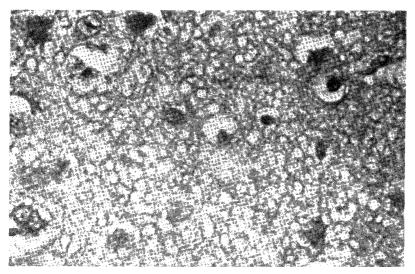


Photo 7. Dehydration

ligation apparently showed the picture of edema of the brain with swollen nerve cells, lost medullary sheath and neuroglia extending into the parenchyma in the shape of sieve. The cerebral tissue dehydrated by Fructon-M was rather contracted as compared with normal tissue. Water content of the brain : After 24 hour ligation, bound water, free water as well as total water showed a trend of increase; total water increased by approximately 3%. However, the water content decreased below the preligation level after dahydration by Fructon-M.

Ultrasonic attenuation in the brain: Attenuation value decreased to the mean of -8.2 db ranging from -5 db to -17 db after 24 hour ligation and returned to the preligation value after dehydration by Fructon-M. Attnuation value decreased as the water content of the brain increased and increased as the latter decreased.

Experiment 2) Change of attenuation during auto-rebreathing<sup>3),33)</sup> (Hypoxia and Hypercapnea) (Figs. 5, 6, 7; Tables 4-7)

In order to cause simultaneous change of blood flow, CSF pressure and water content eight adult rabbits were forced to rebreathe their own exhaled gas. A tube connected to a vinyl bag containing 3.5 liters of air was inserted into the trachea and the change of ultrasonic

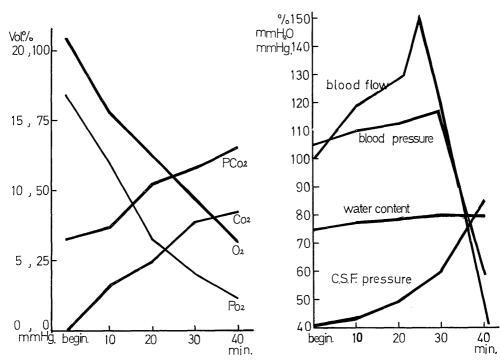
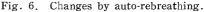


Fig. 5. Changes by auto-rebreathing



attenuation was observed for 40 minutes or more until they died.

On the other hand, water content of the brain, right common carotid blood blood flow, CSF pressure, blood pressure, oxygen and carbon dioxide of the exhaled gas in the bag and  $PO_2$  and  $PCO_2$  of the arterial blood were measured chronologically. The experiment was discontinued when the fixation of probe became unstable due to vehement body movement resulting from rough breathing. RESULTS :

Oxygen in the bag significantly decreased reaching 15.3 vol% in 10 minutes, 12.1 vol% in 20 minutes, 9.2 vol% in 30 minutes and 7.3 vol% in 40 minutes. On the contrary, carbon dioxide increased to 3.4 vol% in 10 minutes, 5.1 vol% in 20 minutes, 7.3 vol% in 30 minutes, and 8.1 vol% in 40 minutes. The content of  $PO_2$  in the

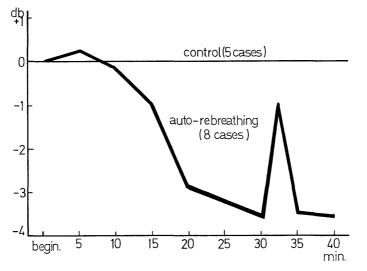


Fig. 7. Change of attenuation by auto-rebreathing

|     |     | al water content,<br>eathing, 10 min. |     |     | al water content,<br>eathing, 20 min. |             |     | al water content,<br>eathing, 30 min. |
|-----|-----|---------------------------------------|-----|-----|---------------------------------------|-------------|-----|---------------------------------------|
| no. | 1.  | 75.06%                                | no. | 1.  | 78.09%                                | no.         | 1.  | 77.57%                                |
| no. | 2.  | 78.08                                 | no. | 2.  | 79.00                                 | no.         | 2.  | 80.76                                 |
| no. | 3.  | 79.80                                 | no. | 3.  | 77.47                                 | no.         | 3.  | 79.98                                 |
| no  | 4.  | 75.32                                 | no. | 4.  | 79.03                                 | no.         | 4.  | 80.54                                 |
| no  | 5.  | 77,83                                 | no. | 5.  | 79.48                                 | no.         | 5.  | 78.49                                 |
| no  | 6.  | 76.25                                 | no. | 6.  | 77.51                                 | no.         | 6.  | 81.66                                 |
| no  | 7.  | 76.49                                 | no. | 7.  | 77.92                                 | no.         | 7.  | 81.36                                 |
| no. | 8.  | 79.22                                 | no. | 8.  | 78.10                                 | no.         | 8.  | 79.57                                 |
| no. | 9.  | 78.77                                 | no. | 9.  | 78.53                                 | no.         | 9.  | 80.62                                 |
| no. | 10. | 76.91                                 | no. | 10. | 76.28                                 | no.         | 10. | 79.36                                 |
|     |     | M=77.37±0.45%                         |     |     | M=78.14±0.29%                         | · _ · · · · |     | M=79.99±0.40%                         |

|      |      | tal water content, |
|------|------|--------------------|
| auto | rebr | eathing, 40 min.   |
| no.  | 1.   | 76.96%             |
| no.  | 2.   | 78.54              |
| no.  | 3.   | 78.37              |
| no.  | 4.   | 82.02              |
| no.  | 5.   | 79.74              |
| no.  | 6.   | 78.55              |
| no.  | 7.   | 81.38              |
| no.  | 8.   | 78.63              |
| no.  | 9.   | 78.01              |
| no.  | 10.  | 82.48              |
|      |      | M=79.47±0.59%      |

arterial blood which was 84.8 mmHg in the mean value before auto-rebreathing decreased to 13.0 mmHg in 40 minutes and  $PCO_2$  increased contrary to  $PO_2$ . Change of water content was measured for 10 cases each at the intervals of 10 minutes.

The value showed a trend of increase with the maximum in 30 minutes as shown in Fig. 6 and Tables 4-7. The right common carotid blood flow increased to 118.8% in 10 minutes, 127.6% in 20 minutes and the maximum value of 155.5% in 25 minutes, which was

followed by a rapid decrease thereafter. Change of blood pressure showed a similar trend.

In eight cases of auto-rebreathing, attenuation began to decrease to  $-2 \sim -6$  db after 10 minutes of rebreathing, increased transiently immediately before death, and again decreased to  $-4 \sim -5$  db after death, the value remaining unchanged thereafter. CSF pressure increased gradually. In this experiment, all of CSF pressure, water content and blood flow changed and ultrasonic attenuation in the brain also decreased.

However it is still unknown whether or not these three factors participate in ultrasonic attenuation and, if so, whether they increase or decrease the attenuation.

Experiment 3) Change of attenuation by blocking common carotid blood flow (Figs. 8-12; Table 8)

This experiment was carried out for the purpose of knowing how change of common carotid blood flow among other factors in experiment 2) participate in the change of ultrasonic attenuation in the brain.

Having the bilateral common carorid arteries exposed and blocked by clip for 1 to 5 minutes, the change of attenuation before and during blocking and after release was observed chronologically.

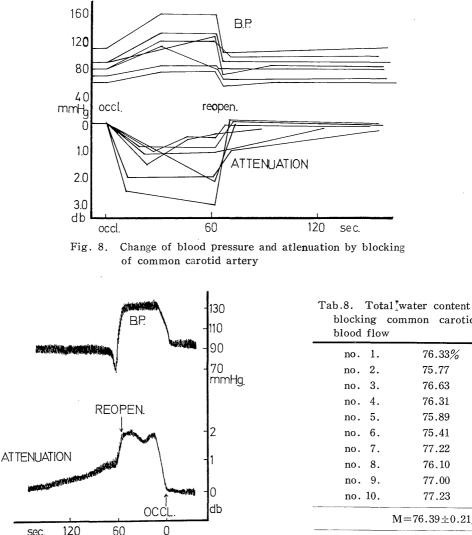
Blood pressure, CSF pressure and water content of the brain were also measured chronologically.

**RESULTS** :

When the bilateral common carotid arteries were blocked for one minutes, blood pressure increased by 15 to 20% in 30 seconds and then remained constant during blocking. The value decreased transiently at release and soon returned to the level before blocking. Attenuation began to increase immediately after blocking  $+1 \sim +3$  db

in 30 to 40 seconds and remained constant thereafter. It decreased rapidly after release and returned to the level before blocking. These findings indicate that the decrease of blood flow increases attenuation.

When the blocking was continued for five minutes, blood pressure increases in 30 to 60 seconds, then remained constant and decreased again to the original level upon release. Attenuation also increased to  $+1 \sim +3$  db in 30 to 60 seconds, then remained constant and upon release, decreased again to the level before blocking. It was thus confirmed that the decrease of blood flow and the increase of attenuation occur synchronously. There was observed no influence ρ blocking of blood flow on CSF pressure and water content.



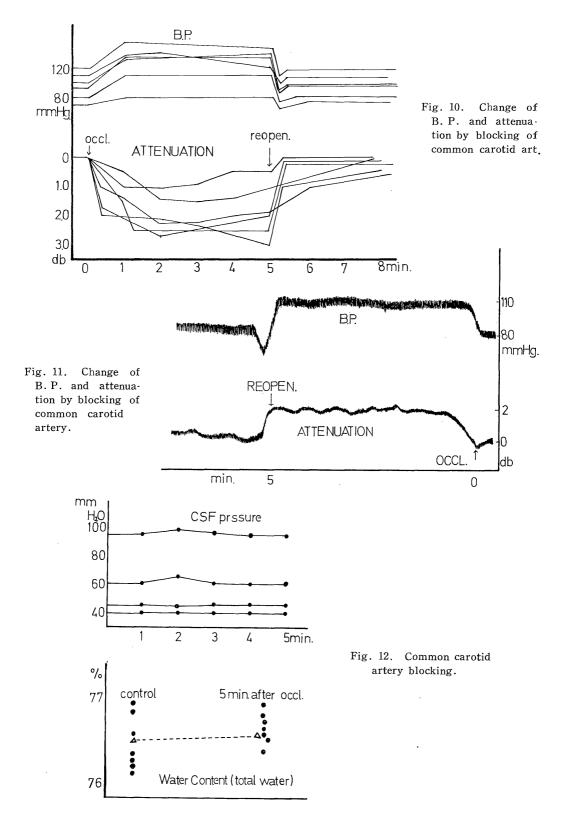
Eig. 9. Change of B.P. and attenuation by blocking of common carotid art.

| 50 OI | a  |     |  |  |  |
|-------|----|-----|--|--|--|
| ence  | of | the |  |  |  |
|       |    |     |  |  |  |

| rab.o. rotar, water content, |         |            |  |  |  |  |  |  |
|------------------------------|---------|------------|--|--|--|--|--|--|
| blocki                       | ng comm | on carotid |  |  |  |  |  |  |
| blood flow                   |         |            |  |  |  |  |  |  |
| no.                          | 1.      | 76.33%     |  |  |  |  |  |  |
| no.                          | 2.      | 75.77      |  |  |  |  |  |  |
| no.                          | 3.      | 76.63      |  |  |  |  |  |  |
| no.                          | 4.      | 76.31      |  |  |  |  |  |  |
| no.                          | 5.      | 75.89      |  |  |  |  |  |  |
| no.                          | 6.      | 75.41      |  |  |  |  |  |  |
| no.                          | 7.      | 77.22      |  |  |  |  |  |  |
| no.                          | 8.      | 76.10      |  |  |  |  |  |  |
| no.                          | 9.      | 77.00      |  |  |  |  |  |  |

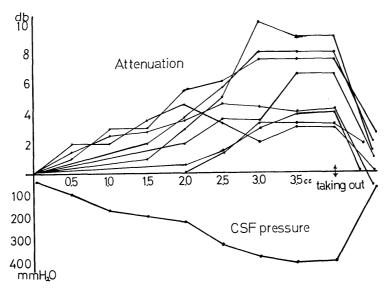
 $M = 76.39 \pm 0.21\%$ 

77.23



Experiment 4) Influence of the change of CSF pressure on ultrasonic attenuation in the brain (Figs. 13-16; Table 9)

After performing puncture of the posterior cistern, 0.5 cc of physiological salt solution was injected at intervals of one minute totalling 3.5 cc and then the cerebrospinal fluid was allowed to flow out naturally so as to decrease CSF pressure and change of attenuation during the course was observed. CSF pressure, water content of the brain and right common carotid blood flow were also measured chronologically.



Eig. 13. Cisternal puncture.

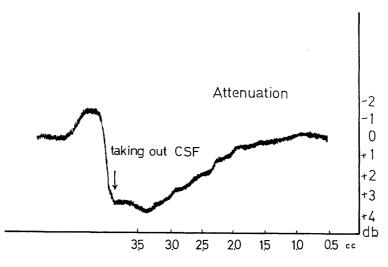


Fig. 14. Cisternal puncture.

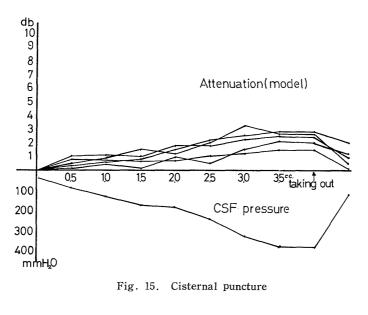
# **RESULTS**:

CSF pressure which was  $30 \sim 40 \text{ mmH}_2\text{O}$  before injection increased to  $205 \text{ mmH}_2\text{O}$  by the injection of a total of 2.0 cc and up to 400  $mmH_2O$  by the injection of a total of 3.5 cc. Then, by natural effluence, the value returned nearly to the original level.

Attenuation value began to increase immediately after injection of physiological salt solution reaching +2.7 db with the injection of 2.0 cc.

Then it showed a rapid increase and reached +6.0 db with the injection of 3.5 cc. The above findings indicate that the increase of CSF pressure and the increase of attenuation are in parallel relation.

With the decrease of CSF pressure, attenuation value began to



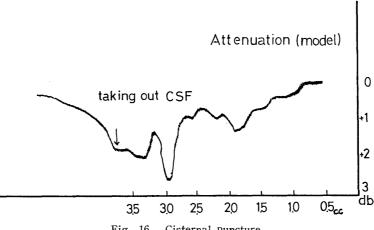


Fig. 16. Cisternal puncture

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decrease reaching the value before injection of the solution. Common carotid blood flow somewhat decreased along with the increase of CSF pressure. It decreased by 22.8% while the latter increased to 400 mmH<sub>2</sub>O.

It did not show any increase immediately after the effluence of cerebrospinal fluid. Water content of the brain increased by about 2% after injection of 3.5 cc. When the same procedure of injecting physiological salt solution was performed on died rabbits, attenuation value also increased but in less degree than that in alive subjects.

| Tab.9. Tot                | al water content,      |  |  |  |  |  |  |
|---------------------------|------------------------|--|--|--|--|--|--|
| change of CSF pressure by |                        |  |  |  |  |  |  |
| cisternal puncture        |                        |  |  |  |  |  |  |
| no. 1.                    | 78.48%                 |  |  |  |  |  |  |
| no. 2.                    | 80.18                  |  |  |  |  |  |  |
| no. 3.                    | 81.03                  |  |  |  |  |  |  |
| no. 4.                    | 78.20                  |  |  |  |  |  |  |
| no. 5.                    | 77.60                  |  |  |  |  |  |  |
| no. 6.                    | 78.33                  |  |  |  |  |  |  |
| no. 7.                    | 80.75                  |  |  |  |  |  |  |
| no. 8.                    | 76.84                  |  |  |  |  |  |  |
| no. 9.                    | 77.29                  |  |  |  |  |  |  |
| no. 10.                   | 76.80                  |  |  |  |  |  |  |
|                           | $M = 78.55 \pm 0.50\%$ |  |  |  |  |  |  |

In summary, the increase of CSF pressure resulted in an increase of attenuation, a slight decrease of common carotid blood flow and a remarkable increase of water content.

## DISCUSSION

1) Relation between ultrasonic attenuation in the brain and water content of the brain (edema of the brain)

The change of ultrasonic attenuation in the brain in the state of edema of the brain had long attracted the attention and may well be considered as the beginning of the study on ultrasonic attenuation in the brain. However, there have been various opinions concerning the effect of edema of the brain<sup>2)36)</sup> (increased water content) on ultrasonic attenuation: some investigators<sup>19),21),25)</sup> say that attenuation is increased and some others<sup>28),40),44)</sup> say that it is decreased. TORII<sup>40)</sup>, Y<sub>OSHIMURA</sub><sup>44)</sup> and I<sub>SHIKAWA</sub><sup>19)</sup> reported that the change of water content at the time of edema was approximately 6%. There was observed no case in the author's experiment that showed value exceeding 4%.

 $K_{AYO}^{23}$  reported that attenuation based on  $4\sim 6\%$  increase of water content is negligible as compared to that due to morphological change of the intracranial constituents at the time of edema of the brain.

 $M_{ARUTA}^{24}$  studied the change of ultrasonic attenuation in the brain following craniotomy and reported that edema of the brain causes an increase of attenuation. However, it is suspected that attenuation might have been affected by the dehydrating agent and the incoming air into the skull during craniotomy. His report on the relation between edema of the brain and increased attenuation has no experimental justification. OKA et al.<sup>31)</sup> observed the chenge of attenuation in the patients of cephalic trauma and, from the change of their clinical symptoms and from autopsy findings, reported that edema of the brain decreases attenuation value.

In his experiments of blocking the common jugular veins and of brain perfusion in 20% mannitol solution,  $N_{AITO^{29}}$  observed a decrease and an increase of attenuation, respectively.

 $K_{ANAYA}^{21}$  et al produced artificial edema of the brain upon exposing unilateral hemicerebrum of the dog to the air and compared the ultrasonic attenuation with that in the normal hemicerebrum. As the result, it was noted that attenuation was less in the exposed hemicerebrum

WAKISAKA<sup>42)</sup> et al. studied the relation between ultrasonic attenuation in the brain and water content of the brain, and observed that attenuation decreased as water content increased. Furthermore, NAITO <sup>29)</sup> studied the change of attenuation when the enucleated rabbit brain was swollen in distilled water and when it was immerged in hypertronic uric solution, and found that attenuation decreased and increased, respectively.

In the author's experiment by bilateral ligation of the jugular veins, attenuation definitely decreased along with the increase of water content and somewhat increased when water content decreased after intravenous injection of Fructon-M. Histological examination also disclosed the pattern of edema. The above findings may suggest that water content of the brain at the time of edema participate in the change of ultrasonic attenuation in the brain, increased water content resulting in decreased attenuation and decreased water content resulting in increased attenuation.

2) Relation between cerebral blood flow and ultrasonic attenuation in the brain

It is manifest that the internal carotid blood flow decreases by blocking of common carotid blood flow even if the blood flow in the vertebral artery system increases.

 $M_{ARUTA}^{26}$  et al. found that the common carotid blood flow remarkably decreased several seconds after the injection of acetyl choline in the external jugular vein of the dog and that ultrasonic attenuation in the brain increased along with the decrease of blood flow and returned to the original level as recovering of blood flow.

 $H_{ATA}^{12}$  et al reported that ultrasonic attenuation in the brain in man increased as cerebral blood flow decreased upon oppression of the carotid arteries and that attenuation decreased as cerebral blood flow increased upon inhalation of carbon dioxide.

In the author's experiment also, attenuation value increased after blocking of the common carotid blood flow and returned to the original value after release of the blood flow. In this experiment, there was noted no influence of blood flow blocking on CSF pressure and water

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content of the brain, and the change of attenuation occurred synchronously with the change of blood flow. Accordingly, it was suggested that decreased cerebral blood flow increased ultrasonic attenuaton in the brain and increased blood flow decreased attenuation. However, it is anticipated that the change of blood flow may cause change of vascular resistance as well as that of water content and cerebral pressure if the blood flow continues to change for a lengthy period of MARUTA<sup>26)</sup> et al. reported their attempt to estimate cerebral time. blood flow from the change of ultrasonic attenuation in the brain, but it is questionable whether the change of ultrasonic atteunation is caused only by the change of cerebral blood flow.  $E_{BARA^{8}}$  et al. reported as the result of model experiment that the change of blood flow velocity did not affect ultrasonic attenuation in the brain. KAYO<sup>23)</sup> reported that inhalation of carbon dioxide resulted in an increase of local blood flow in the brain, blood content in the brain and intracranial pressure, but ultrasonic attenuation in the brain did not indicate any specific trend of change.

In the author's experiment by accumulation of carbon dioxide, cerebral blood flow, CSF pressure and water centent of the brain increased whereas ultrasonic attenuation decreased. These results disagree to KAYO's report but the disagreement may be due to the difference in concentraion of  $CO_2$  in the blood and in degree of the change of water content in the brain<sup>30</sup>.

It may be concluded that ultrasonic attenuation in the brain increases when only cerebral blood flow decreases while there is no change of intracranial pressure and water content of the brain, and vice versa.

3) Relation between CSF pressure and ultrasonic attenuation in the brain

CSF pressure and attenuation indicated exactly synchronous changes. On the other hand, carotid blood flow decreased along with the increase of CSF pressure, and water content increased slightly.

 $K_{AYO}^{23}$  acknowledged by the same experimental procedure as the author's that intracranial pressure and attenuation changed exactly snchronously.

However, he reported that the change of attenuation might not be due to the change of intracranial pressure itself but by morphological or structural change of intracranial constituents possibly caused by the increase of intracranial pressure. In the author's model experiment with rabbite killed by blood discharge, the change of CSF pressure and the change of attenuation synchronized.  $E_{BARA^{8}}$  et al. reported that the change of intracranial pressure alone did not affect attenuation in their model experiment by the use of polyethylene container. ULTRASONIC ATTENUATION IN THE BRAIN

Clinically, the change of intracranial pressure is secondary phenomenon resulting from intracranial disease such as brain tumor, edema of the brain or hematoma, therefore it is not likely that the change of intracranial pressure has direct influence on the change of attenuation. However, this may be an index of the change of CSF pressure when ultrasonic attenuation in the brain is utilized in the same condition.

4) Relation of water content, blood flow and CSF pressure with attenuation

The results of experiment are as shown in Fig. 17. After intravenous injection of Fructon-M, water content, blood flow and CSF

|    |                               | attenuation | water<br>content | blood<br>flow | CSF<br>pressure |
|----|-------------------------------|-------------|------------------|---------------|-----------------|
| 1. | Fructon-M inj                 | 1           | l                | Ļ             | Ļ               |
| 2. | bilat.c.c.artery<br>occlusion | 1           |                  | ţ             |                 |
| Ζ. | reopening                     | ↓           |                  | 1             |                 |
| 3. | cisternal punct.<br>taking in | 1           | Ŷ                | t             | 1               |
| J. | taking out                    | ↓           | ſ                | ł             | ł               |

Summary of all Experiments

Fig. 17.

Relation between Attenuation and three Factors

| factor<br>attenuation | water content | blood flow | CSF pressure |
|-----------------------|---------------|------------|--------------|
| increasing            | Ļ             | Ļ          | Î            |
| decreasing            | Î             | Î          | Ļ            |

pressure decreased and attenuation increased. The blocking of the bilateral carotid arteries was followed by decreased blood flow, unchanged water content and CSF pressure and increased attenuation. After the injection of physiological salt solution upon puncture of the posterior cistern, CSF pressure increased, water content increased slightly, blood flow decreased slightly, and attenuation increased. With the natural discharge thereafter, CSF pressure decreased, water content increased slightly, and attenuation increased. With the natural discharge thereafter, CSF pressure decreased, water content increased slightly, blood flow decreased slightly, and attenuation increased.

Fig. 18 shows the relation of attenuation with these three factors.

(1) When attenuation increased, the decrease of water content and blood flow participated at most but the change of CSF pressure was not constant, showing an increase at times and a decrease some other times.

(2) When attenuation decreased, the increase of water content and blood flow participated mostly and CSF pressure decreased.

Thus, it was confirmed that the change of attenuation was participated mostly by the change of water content and blood flow. The change of CSF pressure was rather considered as a secondary phenomenon resulting from the change of water content and blood flow and having no direct relation with attenuation,

Clinically, however, in the state that water content of the brain increases such as in edema of the brain, cerebral blood flow is naturally considered to decrease. In author's experiment, in the state of the brain edema (experiment 1) attenuation value decreased.

Hence, it is not possible to clarify by these series of expirement whether the increase of water content or the increase of blood flow participates more in the decrease of attenuation.

From the above results, the following conclusions were obtained.

(1) The increase of ultrasonic attenuastion in the brain is participated by either of the decrease of water content of the brain, the decrease of cerebral blood flow, or increase of CSF pressure.

(2) The decrease of ultrasonic attenuation in the brain is participated by either of the increase of water content of the brain, the increase of cerebral blood flow, or decrease of CSF pressure.

## CONCLUSION

The author carried out an experimental study with rabbits to see what intracranial changes participate in the change of ultrasonic attenuation in the brain. The results were as follows.

1) After bilateral ligation of the jugular veins, water content of the brain increased and ultrasonic attenuation in the brain decreased.

2) After intravenous injection of Fructon-M, water content of the

brain decreased and ultrasonic attenuation in the brain increased.

3) Auto-rebreathing resulted in a decrease of ultrasonic attenuation in the brain as well as in a change of water content of the brain, CSF pressure and carotid blood flow.

4) When CSF pressure was increased by puncture of the posterior cistern, ultrasonic attenuation in the brain also increased; when the former was decreased, the latter also decreased.

5) Ultrasonic attenuation in the brain increased after the blocking of carotid blood flow and decreased after release.

From the above results, the following conclusions were obtained.

(1) The increase of ultrasonic attenuation in the brain is participated by either of the decrease of water content of the brain, the decrease of cerebral blood. flow, or the increase of CSF pressure.

(2) The decrease of ultrasonic attenuation in the brain is participated by either of the increase of water content of the brain, the increase of cerebral blood flow, or the decrease of CSF pressure.

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